UNIVERSIDAD DE LEÓN
FACULTAD DE VETERINARIA

DEPARTAMENTO DE MEDICINA, CIRUGÍA Y ANATOMÍA VETERINARIA

PROGRAMA DE DOCTORADO DECALIDAD DE "MEDICINA Y CIRUGIA ANIMAL"
(MCD2004-00342)

TESIS DOCTORAL

ESTUDIO DE NUEVAS TECNICAS EN CIRUGIA VETERINARIA: SHUNT PORTOSISTEMICO. DUCTUS ARTERIOSUS. QUILOTORAX

NEW TECHNIQUES IN VETERINARY SURGERY: PORTOSYSTEMIC SHUNT. DUCTUS ARTERIOSUS. CHYLOTHORAX

NUOVE TECNICHE IN CHIRURGIA VETERINARIA: SHUNT DI PORTOSISTEMICO. ARTERIOSUS DI DUCTUS. CHYLOTHORAX

Memoria presentada por el
Médico Veterinario ***Don Roberto Bussadori,*** para optar al grado de
Doctor Internacional.

UNIVERSIDAD DE LEON
2015.

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MEMORIA DE TESIS

PRESENTADA POR ROBERTO DARIO BUSSADORI

PARA OPTAR AL GRADO DE
DOCTOR EN VETERINARIA POR LA UNIVERSIDAD DE LEÓN

CON MENCIÓN DE

DOCTORADO INTERNACIONAL

DOCTORADO DE CALIDAD

DOCTORADO POR COMPENDIO DE PUBLICACIONES

2015

DIRIGIDO POR:

Dr. JOSE MANUEL GONZALO ORDEN
Dra. MARIA ASUNCION ORDEN RECIO

DEDICATORIA

A mi familia y amigos

AGRADECIMIENTOS

Son muchas las personas que me han animado y ayudado a seguir hasta aquí, así que no voy a intentar plasmarlas en estos agradecimientos pero saben que nunca me olvido de éllas.

De entre mi familia quiero destacar a Claudio que siempre me ha motivado a esforzarme y que quizás es el que más valora este PhD.

A mi mujer y mis hijos que ya me conocieron haciendo esta tesis y que no entienden como puede durar tanto.

A los miembros de equipo de Cirugía de la Facultad de Veterinaria de León, que me redescubrieron la vocación por la veterinaria y especialmente por la Cirugía que ha sido desde entonces mi trabajo principal.

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I.- INTRODUCCION GENERAL

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La relación que tiene la Cirugía Veterinaria con la Cirugía humana, llevó a este equipo a establecer muchas relaciones en cursos de postgrado, en los que participaban tanto veterinarios como médicos.

En mi estancia Erasmus durante la Licenciatura de Veterinaria estuve como alumno interno en la unidad de Cirugía y Radiología de la Facultad de Veterinaria de León, y bajo la dirección del Profesor JM. Gonzalo Orden es donde empecé a trabajar en cirugía endovascular y Radiología Intervencionista.

Posteriormente estuve matriculado en el Doctorado de Medicina y Cirugía Animal, obteniendo el DEA en el curso 2005-06 con el titulo "Embolización mediante coil intravenoso para el tratamiento del shunt portosistémico intrahepático" bajo la dirección del Profesor José Manuel Gonzalo Orden.

Como consecuencia de esta formación hemos desarrollado aptitudes para realizar intervenciones de cauterización selectiva, embolización ATP y Stent en localizaciones periféricas, viscerales y carotideas con protección cerebral. También de desplegamiento de endoprotesis aorticas abdominales y torácicas, para corrección de aneurismas; prácticas de catéter monorraíl y punciones ecoguiadas.

Nuestro deseo fue aplicar todas estas técnicas a casos clínicos que se presentan en la Clínica Veterinaria.

I.1.- Reseña histórica

La cirugía moderna y la del futuro, es y será cada vez menos invasiva y agresiva al igual que la especial habilidad de poder ver e interpretar las imágenes dentro del cuerpo.

El desarrollo de dispositivos como los catéteres y los catéteres-balón, dió lugar a la aparición de la Radiología Intervencionista en los años 70, siendo de hecho, los pioneros en la práctica de angiografía coronaria y otros tratamientos mínimamente invasivos en las arterias.

La progresión ha conseguido que ya desde el año 1992 se haya reconocido en los EEUU como especialidad médica. Actualmente existen dos grandes sociedades que recogen a estos especialistas: la SIR en los EEUU y la CIRSE en Europa

El desarrollo de una serie de técnicas y procedimientos quirúrgicos con el uso y la guía de los métodos de imágenes ha revolucionado la medicina actual permitiendo que instrumentos de alta tecnología sean introducidos al cuerpo humano, a través de orificios muy pequeños en la piel, comunicándose con arterias y venas.

I.2. Parte Primera

I.2.1.- Concepto de Radiología Intervencionista y cirugía endovascular.

La radiología intervencionista es un área de la medicina de rápido crecimiento que se caracteriza por la realización de tratamientos mínimamente invasivos guiados de forma precisa por técnicas de imagen, normalmente por RX o ecografía.

La radiología intervencionista presenta algunas ventajas y de hecho remplaza en muchos casos a la cirugía abierta.

Como características principales tenemos el hecho de no tener que realizar grandes incisiones, ya que se utiliza la introducción de pequeños dispositivos de poco tamaño para introducirse a través de los vasos sanguíneos u otros conductos del organismo para llegar a los órganos diana donde se realizará el tratamiento.

Generalmente comportan menor riesgo, menor dolor y una recuperación más rápida del paciente.

I.2.1.2.- ¿Cómo se realiza?

La mayoría de estos métodos se practican utilizando acceso percutáneo, pero existen situaciones en las que el abordaje quirúrgico del punto de entrada es obligatorio o preferible (cirugía combinada, necesidad de introducir dispositivos de gran calibre).

I.2.1.3.- ¿Qué técnicas incluye?

En los últimos años ha tenido lugar un desarrollo muy grande y exponencial de la tecnología endovascular con la invención, desarrollo y perfeccionamiento de muchos dispositivos de aplicación endoluminal:

Este gran desarrollo no sólo ha tenido lugar en el ámbito terapéutico, sino también en el diagnóstico con la aparición de técnicas como la angioscopia y la ecografía endoluminal.

Actualmente los procedimientos o técnicas que mas habitualmente se realizan para tratamientos endovasculares son básicamente

- Angiografía -Con los aparatos de rayos X convencionales se puede captar la imagen de las arterias y venas para detectar un bloqueo o estrechamiento de los vasos.
- Angioplastia-Es el uso de un pequeño globo en la punta de un catéter introducido en un vaso sanguíneo para abrir un área de bloqueo dentro del vaso. Angioplastia transluminal percutánea con balón (PTA). PTA asistida con Láser
- Embolización.- Es la introducción de una sustancia a través de un catéter en un vaso sanguíneo para detener una hemorragia o sangrado excesivo.
- Ecografía intravascular Es el uso de ultrasonidos dentro de un vaso sanguíneo para visualizar mejor el interior del vaso con el fin de detectar problemas dentro del vaso sanguíneo.
- Colocación de endoprótesis vascular (stent) - espiral expandible y diminuta, también llamada "stent", que se coloca en el interior de un vaso sanguíneo en la zona de un bloqueo, con el fin de abrir el vaso bloqueado.
- Extracción de cuerpo extraño - Es el el uso de un catéter introducido en un vaso sanguíneo para recuperar un cuerpo extraño.
- Biopsia por punción Es la introducción de una pequeña aguja en la zona anormal de casi cualquier parte del cuerpo, guiada por técnicas de imagen, para obtener una biopsia de tejido. Este tipo de biopsia puede proporcionar un diagnóstico sin intervención quirúrgica. Un ejemplo de este procedimiento se denomina biopsia por punción del seno.
- Filtros de coágulos de sangre - Se introduce un pequeño filtro en un coágulo de sangre para capturar y romper los coágulos.
- Inyección de agentes trombolíticos - Los agentes trombolíticos, como el activador del plasminógeno tisular (su sigla en inglés es TPA), son inyectados en el cuerpo para disolver los coágulos de sangre, incrementando de este modo el flujo de sangre al corazón o al cerebro.
- Inserción de catéteres - Se introduce un catéter en las venas grandes para administrar fármacos quimioterapéuticos, apoyo nutricional y hemodiálisis.

- Tratamiento del cáncer - Administración de medicamentos contra el cáncer directamente en la zona del tumor.
- Neuroradiologia Esta técnicas estan dedicadas al Diagnóstico y Terapéutica para el estudio de las imágenes del sistema nervioso central (cerebro y médula espinal), permitiendo diagnosticar enfermedades y posteriormente, realizar el tratamiento por dentro de las arterias (endovascular), o percutáneo para resolver lesiones dentro del cráneo como: Aneurismas, malformaciones, tumores o trombosis cerebral.

I.2.1.4.- Qué ventajas comporta respecto a las técnicas tradicionales?

La Cirugía Endovascular y Radiología intervencionista consigue la resolución de muchos casos de enfermedad vascular (estenosis, oclusiones, aneurismas, fístulas...) con un simple abordaje percutáneo.

Mediante la introducción de guías y catéteres a través de un vaso, habitualmente la arteria femoral, que sirven de transporte a balones de dilatación, endoprótesis. el paciente se beneficia de una técnica menos agresiva y un postoperatorio más corto y confortable.

1.2.2.- Prevalencia de defectos congénitos en el corazón en perros.

Consideramos muy importante conocer la epidemiologia de las malformaciones congénitas del sistema cardiovascular.

El doctorando ha participado en la realización de varios trabajos en los que se estudiaba la prevalencia de diversos defectos congénitos en el corazón, pudiendo destacar entre ellos:

Bussadori C, Pradelli D, Borgarelli M, et al. Congenital heart disease in Boxer dogs: Results of 6 years of breed screening. *The Veterinary Journal* **2009**; 181:187–192.

P. Oliveira, O. Domenech, J. Silva, S. Vannini, R. Bussadori, and C. Bussadori.- Retrospective Review of Congenital Heart Disease in 976 Dogs.- *J. Vet. Intern. Med*.- **2011** May;25(3):477-83.

Estos trabajos se hicieron en Italia en la Clinica Veterinaria Gran Sasso, entre los años 1997 y 2010 . Estos datos son de un centro de referencia cardiológica.

De un total de 4.480 perros sometidos a exploración cardiológica se encontraron un total den 976 perros, con enfermedad cardíaca congénita (CHD)

Su objetivo fué repasar la incidencia de los defectos congénitos del corazón en perros en Italia y determinar la predisposición a padecerlos según la raza y el sexo del animal.

Se realizó un análisis retrospectivo de los expedientes médicos atendiendo al diagnóstico, la anamnesis, el examen clínico, el examen radiográfico, el examen electrocardiográfico, el examen ecocardiográfico la angiografía, y la autopsia.

La predisposición de la raza y del sexo se determinaron con la prueba del cociente de las probabilidades.

La CHD fue observada en 21.7% de casos atendidos.

De un total de 1.132 defectos encontrados, se observo que con un solo defecto había 832 casos (el 85%), con 2 defectos concurrentes en 132 casos (el 14%), y con 3 defectos concurrentes en 12 casos (el 1%).

Los defectos más comunes eran

la estenosis pulmonar (PS; 32.1%),

la estenosis subaórtica (SAS; 21.3%),

el ductus arteriosus persistente (20.9%),

el defecto del tabique ventricular (VSD; 7.5%),

estenosis aórtica valvular (COMO; 5.7%),

displasia tricuspídea (3.1%).

El SAS, el PS, y VSD están asociados con frecuencia a otros defectos.

La prevalencia de la enfermedad congénita cardiaca en función de la raza y del sexo se ha determinado en varios defectos en la muestra analizada.

La prevalencia de los defectos congénitos del corazón en función de la raza y del sexo pueden ser de utilidad para el diagnóstico y la investigación de CHD en perros.

El porcentaje relativamente alto de los defectos concurrentes del corazón acentúa la importancia de los exámenes exactos y completos para su diagnóstico.

I.3.- Parte segunda

I.3.1.- Concepto de Shunt portosistémico

En un hígado anatómicamente normal la sangre de estómago, intestino, bazo y páncreas entra por la vena porta y riega el hígado a través de una red sinusoidal, para después ir a las venas hepáticas y después a la vena caudal.

Los shunts portosistémicos son anomalías vasculares que producen una desviación de la sangre desde la vena porta a la circulación sistémica, sin pasar en parte por los sinusoides hepáticos y el parénquima hepático. Esto produce al cabo del tiempo una atrofia y una insuficiencia hepática, debido al menor aporte de factores tróficos en la sangre portal. También la insuficiencia hepática y la presencia del shunts portosistémicos hace que aumente la presencia de sustancias tóxicas en la circulación sistémica y pueden presentarse alteraciones en el sistema nervioso central.

1.3.2.- Clasificación:

Los Shunts portosistémicos pueden ser congénitos o adquiridos, simples o múltiples**.**

Shunts portosistémicos congénitos: suelen ser vasos únicos, y raramente dos o más, que comunican el suministro venoso portal y la circulación sistémica salvando el hígado (Berent, A 2009). Se consideran congénitos cuando hay una vena única sin hypertension portal concurrente (Szatmari V., Rothuzen, J. 2006)

Shunts portosistémicos adquiridos, son secundarios a una hipertensión portal crónica, suelen presentarse como un vaso turtuoso largo entre la vena esplénica y la vena renal izquierda , o bien como múltiples y turtuosos vasos extrahepáticos , normalmente localizados en la region renal (Bertolini, G. 2010).

Las etiologías mas frecuentes son cirrosis hepática, hipertensión portal no cirrótica, malformaciones arteriovenosas hepáticas, hipoplasia venosa portal congénita, entre otras.

La edad es un Factor a tener en cuanto a su presentación.

Los shunts congénitos intrahepáticos presentan pronto cuadros clínicos y se detectan pronto en perros jóvenes, y en cambio los que tienen shunts congénitos extrahepáticos tardan más en presentarlos (Lamb CR 1996).

1.3.3.-Tratamiento:

Las cirugías clásicas de técnicas quirúrgicas abiertas eran de varios tipos y en todas ellas se pretendía atenuar u obstruir el flujo sanguíneo a través de las derivaciones portocava intrahepáticas.

Se han publicado varias técnicas con sus ventajas e inconvenientes, en las que hay que identificar y aislar la rama de la vena porta que nutre el lóbulo hepático que contiene derivación y la otras técnicas que consisten en el aislamiento de la derivación donde se comunica con la vena hepática antes de que la vena hepática comunique con la vena caudal prehepática. (Breznock. el al.1989)

Dentro de las técnicas extravasculares tenemos: (Pérez Rivero 2012)

Ligadura con seda total o parcial del vaso anómalo

Aplicación del ameroide constrictor

Aplicación de banda de celofán, etc

Actualmente se hace mediante técnicas de radiología intervencionista, técnica que fué por primera vez publicada en el mundo en el trabajo (Gonzalo-Orden 2000), y que supuso un avance muy importante y que marcó un antes y un después en esta cirugía.

1.4. Parte tercera

1.4.1.- **Concepto de Ductus arteriovenoso.**

El ductus arterioso persistente (PDA) o conducto arterioso persistente (CAP) o persistencia del conducto arterioso (PCA) es la persistencia, después de nacer, de la comunicación que normalmente existe entre el sistema arterial pulmonar y la aorta durante la vida fetal, denominado ductus arteriosus.

La causa se desconoce y en el 90 por ciento de los casos se presenta como un defecto único. (Bonagura y Lehmkuhl 1999; Buchanan 2001)

1.4.1.1.- Prevalencia

Afecta con más frecuencia a las hembras en todas las razas, si bien se han encontrado con más frecuencia en los pastores Alemanes (Bonagura y Lehmkuhl 1999; Fossum 2004).

Según un estudio realizado por P. Oliveira et al en 2011 en el que hacen un revisión de 979 perros con enfermedades congénitas del corazón, y dentro de éllas el estudio de 237 casos de perros con ductos arteriosos encontraron los resultados siguientes:

PDA 237 Casos

Raza	Numero	%	Odds Ratio	P
Pastor Alemán	58	24,5	5,2	0,0001
Perros mestizos	41	17,3	0,65	0,18
Terranova	13	5,5	4,65	0,001
Maltes o Bichon Maltes	12	5,1	4,14	0,001
Dóberman	12	5,1	2,8	0,007
Caniche	12	5,1	1,18	NS
Yorkshire terrier	9	3,8	0,71	NS
Cavalier King Charles Spaniel	9	3,8	3,7	0,006
Chihuahua	5	2,5	3,66	0,028
West Highland White Terrier	4	1,7	1,93	NS
Pomerania	5	2,1	2,5	NS
Setter Irlandes				
Pastor Belga	4	1,7	4,38	0,0059
Pastor Australiano	4	1,72		0,001
Otros	37	15,6		

El conducto arterial persistente es común en cachorros con problemas cardíacos congénitos tales como el síndrome del corazón izquierdo hipoplásico, transposición de los grandes vasos y estenosis pulmonar.

Los cachorros con conducto arterial persistente con frecuencia presentan un soplo cardíaco que se puede escuchar con un estetoscopio; sin embargo, en los cachorros prematuros, es posible que dicho soplo no se pueda oír.

El veterinario puede sospechar de la afección si el cachorro tiene problemas para respirar o alimentarse poco tiempo después del nacimiento.

Se pueden observar cambios en radiografías del tórax.

El diagnóstico se confirma con una ecocardiografía.

La ecocardiografía bidimensional ofrece imágenes cardiacas con aspecto anatómico y en movimiento (estudio en tiempo real), contribuyendo a realizar diagnósticos muy precisos de las diferentes CC, lo que relega a los métodos invasivos, como el cateterismo cardiaco diagnóstico, a un segundo plano.

Algunas veces, es posible que un conducto arterial persistente y pequeño no se diagnostique hasta más tarde, cuando el perro desarrolla una enfermedad obstructiva de los vasos pulmonares, lo que produce una inversión del flujo sanguíneo y cianosis.

El conducto arterioso persistente debe cerrarse a la edad más temprana posible.

1.4.1.2.- Tratamientos.

Cierre percutáneo

El tratamiento de elección es el cateterismo terapéutico que consiste en introducir a través de catéter un dispositivo que ocluya el ductus.

El cierre percutáneo del ductus arterioso es el de elección respecto a la cirugía cuando la arteria femoral del paciente tenga un tamaño que permita la introducción del dispositivo y cuando la morfología del conducto lo permite y el paciente pesa más de 6 kg.

Aunque con los nuevos dispositivos el tamaño y el peso ya no es tan importante.

Los resultados a corto y medio plazo están siendo muy buenos, y las complicaciones mínimas•

La oclusión percutánea del ductus arterioso persistente es la técnica de elección en la mayoría de los casos.

Los dispositivos más utilizados son los *coils* de liberación controlada (pequeño tamaño, (coil de Gianturco)), el Nitoccluder (tamaño mediano) (Gamboa R, 2007) y el Amplatzer duct occluder (grandes y medianos)(Wang 2007).

Los resultados a corto y medio plazo están siendo muy buenos, y las complicaciones mínimas.

Tratamiento quirúrgico:

En algunas circunstancias, como pacientes prematuros o de muy pequeño peso, puede ser necesaria una intervención quirúrgica convencional.

1.4.2. Troponina I

Dentro de los avances diagnósticos laboratoriales, puede encontrarse la determinación de la troponina.

La troponina es una proteína que colabora en el acoplamiento actina-miosina que se produce durante la contracción muscular.

Comprende tres subunidades denominadas troponina T, troponina I y troponina C.

A su vez existen tres subtipos principales de la troponina I, localizados en diferentes tejidos:

- La isoforma troponina I en el músculo liso de contracción lenta.
- La isoforma troponina I en el músculo liso de contracción rápida,
- La isoforma troponina I cardíaca.

Las troponinas I y T presentes en el músculo cardíaco presentan unas características peculiares, y se han desarrollado técnicas de inmunoanálisis específicas para su detección que no presentan reactividad cruzada alguna con las formas de troponina T y troponina I existentes en el músculo esquelético

La TnIc ha sido objeto de varios estudios para analizar su utilidad en el diagnóstico del infarto perioperatorio , en razón de su especificidad y dado el poco valor

que ofrecen la CPK total y CPK-MB debido a la importante lesión musculosquelética que conlleva la cirugía.

La particular cinética de las TnIc y TnTc hacen de ellas una herramienta muy útil para la valoración de cuadros indicativos de cardiopatía isquémica cuando ya han pasado varios días desde el supuesto evento isquémico.

La cardioespecificidad de la TnIc es casi absoluta, pero presenta las mismas limitaciones que el resto de las enzimas miocárdicas para la identificación de la etiología de la lesión miocárdica, por lo que una elevación de TnIc no es sinónimo de cardiopatía isquémica con obstrucción coronaria, sino de mínima necrosis.

1.5.- Parte Cuarta

1.5.1. **Concepto de Quilotóra**x

El quilotórax es una acumulación de linfa en la cavidad pleural. Fué descrito por primera vez por Pisek en 1917

1.5.2.Clasificación:

Se describen 2 grandes grupos etiológicos: el quilotórax congénito y el adquirido.

La mayor parte de los quilotórax congénitos son idiopáticos, mientras que los adquiridos suelen presentarse como complicación de cualquier tipo de cirugía torácica, especialmente la cardíaca.

También se relaciona con la formación de trombos en el territorio de la vena cava superior y al aumento de la presión venosa central por disfunción miocárdica

1.5.3..- Tratamiento:

El tratamiento puede ser conservador o agresivo dependiendo del panorama clínico.

Los objetivos del tratamiento, basado en medidas dietéticas, farmacológicas y quirúrgicas, tratan de aliviar la afectación respiratoria, mantener un estado nutricional adecuado, intentar disminuir la producción de quilo y disminuir la morbilidad asociada.

<u>El tratamiento quirúrgico</u>

Las recomendaciones sobre el tipo de cirugía de elección también son variables

a) El tratamiento convencional del Quilothorax idiopático (IC) implica la ligadura torácica del conducto (TD) (con/sin linfagiografia) combinada con pericardiectomia subfrénica.

b) El tratamiento quirúrgico en casos refractarios conlleva la ligadura del conducto torácico, pleurodesis y en algunos casos shunt pleuroperitoneal

c) La adición del omentalisation pleural a la ligadura del bloque del conducto torácico TD y a pericardiectomia subfrénica, no parece que produzca mejores resultados. (Bussadori 2011)

II:- OBJETIVOS:

1º.- Ver todos aquellos procesos patológicos que se presentan en los perros y gatos que son susceptibles de tratarse mediante estas técnicas.

2º.- En función de la casuística que nos llega a nuestras Clínicas seleccionar aquellos defectos congénitos o adquiridos más representativos como ductus arteriosus, fístulas portosistémicas congénitas, o quilotórax.

III.- METODOLOGÍA Y PLAN DE TRABAJO:

1º.- Cada caso clínico que se nos presente se documentará totalmente, y se estudiará por el doctorando supervisado por sus directores.

2º.- Se procurará operar a los animales en Italia o España, donde sea más próximo. En Italia los animales que lleguen a la Clinica Veterinaria Gran Sasso y al Departamento de Cirugia de la Universidad de Torino. En España los animales que sean referidos al Departamento de Medicina, Cirugia y Anatomia Veterinaria de León.

3º.- Una vez conseguidos casos de una misma patología, se elaborara el trabajo correspondiente a esa patología, procurando publicarlo en revistas de índice de impacto para una mayor difusión.

IV.- RESULTADOS

IV.- RESULTADOS

Los resultados obtenidos en esta tesis, corresponden a los objetivos que nos habíamos propuesto.

Primer objetivo:

Ver todos aquellos procesos patológicos que se presentan en los perros y gatos que son susceptibles de tratarse mediante estas técnicas.

El doctorado ha participado en varios trabajos y en ellos se recogen los resultados dentro del objetivo primero.

Bussadori C, Pradelli D, Borgarelli M, et al. Congenital heart disease in Boxer dogs: Results of 6 years of breed screening. *The Veterinary Journal* **2009**; 181:187–192.

P. Oliveira, O. Domenech, J. Silva, S. Vannini, R. Bussadori, and C. Bussadori.- Retrospective Review of Congenital Heart Disease in 976 Dogs.- *J. Vet. Intern. Med*.- **2011** May;25(3):477-83.

El segundo objetico

En función de la casuística que nos llega a nuestras Clínicas seleccionar aquellos defectos congénitos o adquiridos más representativos como ductus arteriosus, fistulas portosistémicas congénitas, o quilotórax.

Se recogen los resultados en los siguientes trabajos, que dieron lugar a publicaciones en revistas recogidas en JCR

Shunt Portosistemicos

Bussadori, R.; Bussadori C, Millan L, Costilla S, Rodríguez-Altonaga JA, Orden MA, Gonzalo-Orden JM.- Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs.- *The Veterinary Journa*l. 2007. Volume: 176.Issue: 2.Pages**:** 221-226. Índice de impacto: 1.802 según el JCR del 2008. Ocupa el lugar 35/135

Rossi F, Domenech O , Bussadori R , Bussadori C .- Role of multi-modality imaging to guide procedures of transvenous coil embolisation for the treatment of canine intrahepatic porto-systemic shunt.-: *Veterinary Radiology & Ultrasound* . 2010.Volume: 51 Issue: 2 Pages: 198-198.I

Ductus arteriosus.

Bussadori R.; Tamborini A, Locatelli C, Palermo V, Brambilla PG.- Troponin I perioperative trend in dogs undergoing the correction of patent ductus arteriosus: preliminary investigations.. *Vet Res Commun*. 2008. Sep;32 Suppl 1:S255-8. Indice de impacto 1,050. JCR año 2009. Lugar que ocupa 53/142

Quilotorax

Bussadori R, Provera A, Martano M, Morello E, Gonzalo-Orden JM, Rosa GL, Stefano N, Maria RS, Sara Z, Buracco P. Pleural omentalisation with en bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats. *The Veterinary Journal*. 2011 May;188(2):234-6. Epub 2010 Jun 18. Indice de impacto: 2,239. Lugar que ocupa 8/145.

V.-. ABSTRACT. SOMMARIO. RESUMEN.

V.-. ABSTRACT. SOMMARIO. RESUMEN.

V.1.. **Shunts Portosistemicos**

Bussadori, R.; Bussadori C, Millan L, Costilla S, Rodríguez-Altonaga JA, Orden MA, Gonzalo-Orden JM.- Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs.- *The Veterinary Journa*l. 2007. Volume: 176.Issue: 2.Pages**:** 221-226

ABSTRACT

This article describes the treatment of single congenital portosystemic shunts (CPSs) (intrahepatic and extrahepatic) using an interventional radiology technique involving embolisation of anomalous vessels with percutaneous coils.

Briefly, a multipurpose catheter was introduced into the caudal vena cava and then into the portosystemic shunt.

An autoexpandable stent was placed in the caudal vena cava, next to the shunt, in order to avoid coil migrations, and a cobra-like vascular catheter was used to pass through the stent and to place the coils in the shunt.

This technique was used for treatment of CPS in six dogs.

The results indicate that percutaneous embolisation of a CPS using coils, a less invasive technique than the traditional surgical technique, may result in complete closure of the anomalous vessel without development of portal hypertension.

SOMMARIO

Questo articolo descrive il trattamento di shunt portosistemici singoli (CPSs) (intraepatici o extraepatici) per mezzo di radiologia interventistica con tecnica di embolizzazione percutanea con coils dei vasi anomali.

Un catetere multipurpose fu introdotto nella vena cava caudale e quindi nello shunt portosistemico.

Uno stent autoespandibile fu posizionato nella vena cava caudale in prossimita dello shunt per evitare la migrazione dei coils, quindi un catetere cobra-like fu usato per passare attraverso lo stent e rilasciare I coils nel vaso anomalo.

Questa tecnica fu usata per il trattamento del CPSs in sei cani.

I risultati di questo studio indicano che la tecnica di embolizzazione percutanea di CPSs con coils è una tecnica meno invasive rispetto alle tecniche attuate in chirurgia tradizionale e puo portare a complete chiusura del vaso anomalo senza sviluppo di ipertensione portale.

RESUMEN

En este artículo se describe el tratamiento de los shunt portosistémicos (CPSs) (intrahepáticos y extrahepáticos), mediante una técnica de radiología intervencionista de embolizacion percutánea usando coils para el cierre del vaso anómalo.

Se introduce un catéter desde la vena cava caudal hasta la desviación portosistémica. Este catéter se utiliza para colocar el stent y los coils.

El stent autoexpandible se colocó al lado de la desviación del shunt, para evitar migraciones de los coils.

Esta técnica fué utilizada para el tratamiento del CPS en seis perros.

Los resultados indican que el embolizacion percutánea de un CPS usando las coils es una técnica menos invasiva que la técnica quirúrgica tradicional, y que da lugar al cierre completo del shunt sin desarrollar hipertensión portal.

V.2.- **Ductus arteriosus.**

Bussadori R, Tamborini A, Locatelli C, Palermo V, Brambilla PG.- Troponin I perioperative trend in dogs undergoing the correction of patent ductus arteriosus: preliminary investigations.. *Vet Res Commun*. 2008. Sep;32 Suppl 1:S255-8.

ABSTRACT

The PDA closure result in preload reduction and acute variation in aorta and left ventricle load pressure this could produce a reléase of cardiac troponin I (cTnI), an intracellular miofribbillar protein, that in courrently considered the gold standard for the diagnosis of miocardial injury.

This study evaluates a cTnI concentrations in two groups of ten dogs. Eight dogs aged less than 13 months were asynptomatics, two adults presented symptoms of left congestive heart failure.

We have found that the increase of cTnI concentration in dogs was concomitant with the acute variations in aorta and left ventricle pressure following PDA closure. This means that the damage within the myocardial cell could be considered transitory.

SOMMARIO

La chiusura del dotto arterioso porta ad una riduzione del preload e a variazioni acute di pressione nel ventricolo destro questi eventi possono produrre un rilascio di troponina caridaca I (cTnI), una proteína miofibrillire intracellulare che viene correntemente considerata il gold standard per la diagnosi del danno miocárdico.

Questo studio valuta le concentraioni di cTnI in due gruppi di 10 cani. Otto cani con età inferiore a 13 mesi erano asintomatici, due adulti presentavano sintomi di cardiopatía congestizia sinistra.

Abbiamo rilevato che l'aumento dei livelli di cTnI era concomitante alle variazioni acute di pressione in aorta e nel ventricolo sinistro dopo la chiusura del dotto arterioso. Questo suggerisce che il danno cellulare miocardico può essere considerato transitorio.

RESUMEN

El cierre del ductus arterioso conlleva una reducción de la carga y a variaciones agudas de la presión en el ventrículo derecho y estos acontecimientos puede producir un alteración en la tasa de la Troponina I cardiaca (cTnI), un proteína cuya determinación y valor se considera como el patrón mejor para valorar el daño del miocárdico.

Se estudiaron las concentraciones de cTnI en dos grupos de 10 perros. Ocho perros con edad inferior a 13 meses no tenían sintomatología clínica, dos perros adultos presentaban sintomatología cardiopatía congestiva izquierda.

Hemos encontrado que el aumento de los niveles del cTnI era concomitante a las variaciones agudas de la presión en aorta y del ventrículo izquierdo después de cerrar del ductus arteriovenoso.

Los valores obtenidos de cTnI sugieren que el daño celular del miocardio se pueda considerar transitorio.

V.3.- **Quilotorax**

Bussadori R, Provera A, Martano M., Morello E., Gonzalo-Orden,JM., La Rosa G, Niccoli S, Zabarino S, Buracco P.- Pleural omentalisation with en bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats. Veterinary J. 2011 May;188 (2):234-6. Epub 2010 Jun 18.

ABSTRACT

Conventional treatment of idiopathic chylothorax (IC) involves thoracic duct (TD) ligation (with/without lymphagiography) combined with subphrenic pericardiectomy.

Nine dogs and four cats with IC, which received intrathoracic omentalisation with TD en bloc ligation (not preceded by lymphangiography) and subphrenic pericardiectomy, were evaluated retrospectively.

Seven of nine dogs and 3/4 cats were still alive and disease-free at the time of reporting (range 10-53 and 19-31months, respectively).

Clinical signs of IC did not decrease after the first surgery in one cat and one dog; in another dog clinical signs recurred after 5months.

Overall efficacy rate of this one-stage combined procedure was 77% (6months), 73% (12months), and 57% (24months).

Where a second surgery was performed in case of failure, the success rate in dogs was 89% (6months) and 80% (24months).

Addition of pleural omentalisation to TD en bloc ligation and subphrenic pericardiectomy does not seem to improve results when compared with published data and at present does not seem advisable as a first choice.

SOMMARIO

Il trattamento convenzionale del chilotrace idiopático (IC) comprende la legatura del dotto toracico (TD) (con o senza linfangiofrafia) e la pericardectomia subfrenica

Nove cani e quattro Gatti con IC trattati con omentalizzazione intratoracica, legatura del dotto toracico (non preceduta da linfangiografia) e pericardectomia subfrenica sono stati valutati.

Sette di nove cani e tre di quattro gatti risultavano ancora vivi e clinicamente guariti al momento della stesura dell'articolo (range 10-53 e 19-31 mesi, rispettivsamente).

I Segni clinici di IC non diminuirono dopo il primo intervento in un gatto e in un cane; In un altro cane i Segni clinici si ripresentarono 5 mesi dopo l'intervento chirurgico.

L'efficacia di questa combinazione di tecniche con il primo intervento fu del 77% (6 mesi), 73% (12 mesi),e 57% (24 mesi).

Quando fu attuato un secondo intervento chirurgico per insuccesso del primo l'efficacia del trattamento nei cani fu dell' 89% (6 mesi). L'aggiunta dell'omentalizzazione pleurica alla legatura en bloc del dotto toracico e alla pericardectomia subfrenica non sembra migliorare il tasso di successo se compariamo questo studio con altri presenti in letteratura e non sembra consigliabile come técnica d'elezione.

RESUMEN

Tratamiento convencional de quilotórax idiopático (IC) incluye la ligadura del conducto torácico (TD) (con/sin lymphagiography) combinado con pericardiectomía subfrénica.

Se evaluaron en forma retrospectiva nueve perros y cuatro gatos con quilotórax idiopático, a los que se les opero con una técnica de omentalizacion intratorácica, ligadura del conducto torácico (no precedida de linfangiografia) y pericardiectomia subfrénica

Siete de los nueve perros y 3 de los cuatro gatos todavía estaban vivos y libres de enfermedad en el momento de la presentación de informes (rango 10-53 y 19 a 31 meses, respectivamente).

Los signos clínicos del quilotórax idiopático no disminuyeron después de la primera cirugía en un gato y en un perro. En otro perro los signos clínicos reaparecieron después de 5 meses.

La tasa de eficacia de esta combinación de técnicas quirúrgicas fué de 77% a los 6 meses, de 73%a los 12 meses), y el 57% a lo 24 meses

Se presentó un caso de fracaso, y cuando se reoperó la tasa de éxito de los perros subió al 89% (a los 6 meses).

En el tratamiento del quilotórax idiopático el realizar una omentalización intratoracica junto a la ligadura en bloque del conducto torácico y pericardiectomía subfrénica no parece mejorar los resultados en comparación con los datos publicados y en la actualidad no parece aconsejable como primera opción.

VI. CONCLUSIONES. CONCLUSIONS. CONCLUSIONI

CONCLUSIONES

1º.- El método combinado de embolización con coils y la implantación de stent en la vena cava caudal es un procedimiento valioso para el tratamiento del shunt portosistémico en los perros.

Por otra parte, esta técnica permite la oclusión del vaso anómalo en intervenciones sucesivas si es necesario: la radiopacidad de los coils permite la identificación inmediata del vaso anómalo en los procedimientos posteriores.

2º.- El análisis y determinación de los valores de la Troponina cardiaca I (cTnI) , nos permite analizar el daño miocárdico que se produce con diferentes técnicas quirúrgicas en la corrección del Ductus arteriovenoso persistente.

También permitirá determinar la concentración de los valores de la cTnI, en los que se pueda establecer el nivel de riesgo quirúrgico.

Sin embargo consideramos que esta conclusión deberá comprobarse con el estudio de mas animales con Ductus arteriovenoso persistente.

3º.- En el tratamiento del quilotórax idiopático, el realizar una omentalización intratorácica junto a la ligadura en bloque del conducto torácico y pericardiectomía subfrénica, no parece mejorar los resultados en comparación con los datos publicados y en la actualidad no es aconsejable como primera opción.

CONCLUSIONS

1º.- The combined method of embolization with coils and stent implantation in the caudal vena cava is a valuable procedure for the treatment of CPS in dogs.

Moreover, this technique permits occlusion of the anomalous vessel in several interventions if necessary: the radiopacity of the coils allows immediate identification of the anomalous vessel in later procedures.

2º.- This could allow us to analyze the consequences of different techniques in correcting PDA, on heart and vascular system as well as to investigate the perioperative trend of cTnI, as has already been performed. This will allow us to determine the predictive concentration and the cut-off values of cTnI, with the aim of establishing the level of surgical risk.

3º.- Treatment of idiopathic chylothorax (IC): Addition of pleural omentalisation to TD en bloc ligation and subphrenic pericardiectomy does not seem to improve results when compared with published data and at present does not seem advisable as a first choice.

CONCLUSIONI

1°.- Il metodo combinato di embolizzazione con coils e l'inserimento di stent nella vena cava caudaleè un valido procedimento per il trattamento dello shunt portosistemico nei cani.

Inoltre, questa técnica permette l'occlusione del vaso anómalo in diversi tempi se necessario: La radiopacità dei coils permette l'immediata identificazione del vaso anomalonei successivi procedimenti.

2°.- L'analisi e la determinazione dei valori di troponina cardiaca I (cTnI), ci permette di analizzare il danno miocardicoche si produce con differenti tecniche chirurgiche per la correzione del dotto arterioso persistente.

Inoltre permettetrà di determinare la concentrazione dei valori di cTnI, entro i quali si possa stailire il livello di rischio chirurgico.

Senza dubio consideriamo che questa conclusione dovra essere confermata con studi comprendenti un numero piu cospiquo di animali con dotto arterioso persistente.

3.- Nel trattamento del chilotorace idiopático la realizzazione di una omentalizzazione intratoracica assieme alla legatura en bloc del condotto torácico e pericardiectomia subfrenica non sembra migliorare i risultati in comparazione coi dati pubblicati e non sembra quindi consigliable come prima opzione.

VII.- BIBLIOGRAFÍA

VII.- BIBLIOGRAFÍA

Adrega da Silva, C., Monnet, E., Bardet, J.F., - Long-term clinical outcome of surgical treatment for idiopathic chylothorax in 10 dogs. In: *Proceedings of the 18th Annual Scientific Meeting of the European College of Veterinary Surgeons*, Nantes, France, 2009. pp. 289–291.

AM POP, Gheorghe, CRAMER, Etienne, TIMMERMANS, Janneke et al. Liberación de troponina I en reposo y con ejercicio en sujetos con miocardiopatía hipertrófica. Efecto de betabloqueadores. *Arch. Cardiol. Méx.* 2006, vol. 76, no. 4 pp. 415-418

Asano, K., Watari, T., Kuwabara, M., Sasaki, Y., Teshimo, K., Kato, Y., Tanaka, S.- Successful treatment by percutaneous transvenous coil embolisation in a small-breed dog with intrahepatic portosystemic shunt. *Journal of Veterinary Medicine Sciences* 2003. - 65, 1269–1272.

Baumgartner C, Glaus TM. Congenital cardiac diseases in dogs: A retrospective analysis. *Schweiz Arch Tierheilkd* 2003; 145: 527–533, 535–526.

Berent, A.; Cand Tobbias KM. - Portosytemic vascular anomalies- *Vet. Clin. North Am. Small Anim. Pract*. 2009. 39 (3) 513 -541

Bonagura J.D. and Lehmkuhl L.B., Congenital heart disease. In *Textbook of canine and feline cardiology*, P.R Fox, D.D. Sisson, N.S. Moise (Eds.), 2nd edition. WB Saunders: Philadelphia, 1999. 471–535.

Brotmacher L, Campbell M. Ventricular septal defect with pulmonary stenosis. *Br Heart J* 1958; 20: 379–388.

Buchanan J.W, Patent ductus arteriousus morphology, pathogenesis, types and treatment. *Journal of Veterinary Cardiology*. 2001. 1, 7–16.

Buchanan JW. Causes and prevalence of cardiovascular disease. In: Kirk RW, Bonagura JD, eds. *Current Veterinary Therapy XI: Small Animal Practice*. Philadelphia, PA: WB Saunders; 1992:647–655.

Buchanan JW. Pathogenesis of single right coronary artery and pulmonic stenosis in English Bulldogs. *J Vet Intern* Med 2001; 15:101–104.

Buchanan JW. Prevalence of cardiovascular disorders. In: Fox PR, Sisson DD, Moise NS, eds. *Textbook of Canine and Feline Cardiology*, 2nd ed. Philadelphia, PA: WB Saunders Company; 1999:458–463.

Buchanan JW. Pulmonic stenosis caused by single coronary artery in dogs: Four cases (1965–1984). *J Am Vet Med Assoc* 1990; 196:115–120.

Burton, C.A., White, R.N.-.Portovenogram findings in cases of elevated bile acid concentrations following correction of portosystemic shunts. *Journal of Small Animal Practice.* 2001. 42, 536–540.

Bussadori C, Amberger C, Le Bobinnec G, et al. Guidelines for the echocardiographic studies of suspected subaortic and pulmonic stenosis. *J Vet Cardiol* 2000; 2:15–22.

Bussadori C, DeMadron E, Santilli RA, et al. Balloon valvuloplasty in 30 dogs with pulmonic stenosis: Effect of valve morphology and annular size on initial and 1-year outcome. *J Vet Intern Med* 2001; 15:553–558.

Bussadori C, Domenech O, Longo A, Pradelli D, Bussadori R.- Percutaneous catheter-based treatment of pulmonic stenosis and patent ductus arteriosus in a dog.- *J Vet Cardiol.* 2002. Nov. 4(2):29-34

Bussadori C, Pradelli D, Borgarelli M, et al. Congenital heart disease in Boxer dogs: Results of 6 years of breed screening. *The Veterinary Journal* 2009; 181:187–192.

Bussadori C, Quintavalla C, Capelli A. Prevalence of congenital heart disease in Boxers in Italy. *J Vet Cardiol* 2001; 3: 7–11.

Bussadori R, Bussadori C, Millán L, Costilla S, Rodríguez-Altónaga JA, Orden MA, Gonzalo-Orden JM. - Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs. The *Veterinary Journal***.** 2008. May; 176 (2):221-6.

Bussadori R, Tamborini A, Locatelli C, Palermo V, Brambilla PG.- Troponin I perioperative trend in dogs undergoing the correction of patent ductus arteriosus: preliminary investigations.. *Vet Res Commun*. 2008. Sep;32 Suppl 1:S255-8.

Bussadori,R.; Provera,A.; Martano, M.; Morello,E. , Gonzalo-Orden, J.M. La Rosa, G.; Stefano,N.; , Repetto,S.M.; Zabarino,S.; Buracco,P.- Pleural omentalisation with en

bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats. T*he Veterinary Journal* 188 (2011) 234–236

Campbell FE, Thomas WP, Miller SJ, et al. Immediate and late outcomes of transarterial coil occlusion of patent ductus arteriosus in dogs. *J Vet Intern Med* 2006;20:83–96.

Carobbi, B., White, R.A.S., Romanelli, G., Treatment of idiopathic chylothorax in 14 dogs by ligation of the thoracic duct and partial pericardiectomy. *Veterinary Record* 2008. 163, 743–745.

Chetboul V, Charles V, Nicolle A, et al. Retrospective study of 156 atrial septal defects in dogs and cats (2001–2005*).- J Vet Med A Physiol Pathol Clin Med* 2006; 53:179–184.

Corno AE, Festa P. Ventricular septal defect. In: Corno AF, ed. *Congenital Heart Defects*. Germany: Steinkopff Verlag; 2009:49–55.

Costilla, S., Balboa, O., Gonzalo Orden, J.M., Magadán, V., Millán, L., Altónaga, J.A.R. Desarrollo de un sistema introductor de prótesis bifurcadas autoexpandibles para su implantación por técnicas de radiología intervencionista**.** *VII Congreso Nacional de la de la Sociedad Española de Radiología Vascular e Intervencionista (SERVEI***.)** Huelva, 9-11 de mayo 2001.

Costilla, S., Balboa, O., Gonzalo Orden, J.M., Magadán, V., Millán, L. Oclusión de fístula portocava congénita en perros con stent-coils. Posible aplicación en humanos. *VII Congreso Nacional de la de la Sociedad Española de Radiología Vascular e Intervencionista (SERVEI)***.** Huelva, 9-11 de mayo 2001.

Costilla, S., Balboa, O., Hernández, L., Gonzalo Orden, J.M., Magadán, V., Millán, L. Stents bifurcados autoexpandibles: diseño de una pieza y sistema de introducción. *Congreso Nacional de la Sociedad Española de Radiología Vascular e Intervencionista (SERVEI).* Alicante, 7-10 de mayo de 2003.

Costilla, S., Balboa, O., Magadán, V., Millán, L., Orden, M.A., Gonzalo Orden, J.M. Stents bifurcados: diseño en una pieza única y prototipo de un sistema transportador para su colocación por acceso percutáneo**.** *XII Congreso Internacional de Cirugía Veterinaria (SECIVE).* León, 20-22 de marzo de 2003.

Costilla, S., Hernández, L., Balboa, O., Magadán, V., Millán, L., Gonzalo Orden, J.M. Bifurcated stents: design in a single piece and its implantation procedure. *European Congress of Radiology***.** Viena, 2003.

Fonfara S, Martinez Pereira Y, Swift S, et al. Balloon valvuloplasty for treatment of pulmonic stenosis in English Bulldogs with an aberrant coronary artery. *J Vet Intern Med 24:354– 359*.

Fossum T.W., Chirurgia dei piccoli animali, 2th edition. Elsevier-Masson: Milano. Fellahi J.L., Gue X., Richomme X., Monier E., Guillon L., Riou B., 2003. Short-and long-term prognostic value of postoperative cardiac troponin I concentration in patients undergoing coronary artery bypass grafting. *Anesthesiology*, 2004. 99, 270–274.

Fossum, T.W., Birchard, S.J., Jacobs, R.M., Chylothorax in 34 dogs. *Journal of American Veterinary Medical Association.* 1986. 188, 1315–1318.

Fossum, T.W., Mertens, M.M., Miller, M.W., Peacock, J.T., Saunders, A., Gordon, S., Pahl, G., Makarski, L.A., Bahr, A., Hobson, P.H., Thoracic duct ligation and pericardectomy for treatment of idiopathic chylothorax. *Journal of Veterinary Internal Medicine* 2004. 18, 307–310.

Gamboa R, Mollón FP, Ríos-Méndez RE, Arroyo GM, Fogel A, Villa DM. Cierre del ductus arterioso permeable con un nuevo tipo de dispositivo: Nit-Occlud. *Rev Esp Cardiol. 2007;60:445-8.*

Golli, M., Kriaa, S., Said, M., Belguith, M., Zbidi, M., Saad, J., Nouri, A., Ganouni, A., Intrahepatic spontaneous portosystemic venous shunt: value of color and power Doppler sonography. *Journal of Clinical Ultrasound* .2000. 28, 47–50.

Gonzalo Orden, J.M., Altónaga, J.A.R., Costilla, S., Millán, L., Gonzalo Cordero, J.M., Orden, M.A., Transvenous coil embolization of intrahepatic portosystemic shunt in a dog. *Veterinary Radiology & Ultrasound*, 2000. 41 (6): 516-518.

Gonzalo Orden, J.M., Costilla, S., Millán, L., Iglesias, I., Altónaga, J.A.R., Orden, M.A. Tratamiento quirúrgico mediante radiología intervensionista de un caso de comunicación portosistémica intrahepática en un perro. *X Congreso Internacional de Cirugía Veterinaria (SECIVE)***.** Madrid, 31 de marzo a 1 de abril de 2001. Proceeding

Gonzalo Orden, J.M., Millán, L., Fueyo, F., Altónaga, J.A.R., Borge, A., Orden, M.A. Resolución quirúrgica de una hernia peritoneopericárdica en perro. Caso clínico**.** *XI Congreso Internacional de Cirugía Veterinaria (SECIVE).* Murcia, 8-10 de marzo de 2002**.** Proceeding

Gonzalo Orden, JM.- Ponencia: Radiología intervencionista en veterinaria**.-** *XVI Congreso Internacional de la Sociedad Española de Cirugía Veterinaria (SECIVE)***.** Zaragoza (España) 11,12 y 13 de abril de 2008. Proceeding.

Gonzalo-Orden, J.M., Altonaga, J.R., Costilla, S., Gonzalo Cordero, J.M., Millan, L., Orden Recio, M.A.,. A transvenous coil embolisation of an intrahepatic portosystemic shunt in a dog. *Veterinary Radiology and Ultrasound* 2000.41, 516–518.

Gutgesell HP, Huhta JC, Latson LA, et al. Accuracy of twodimensional echocardiography in the diagnosis of congenital heart disease. *Am J Cardiol* 1985;55:514–518

Harada K., Toyono M., Tamura M., 2004. Effects of coil closure of patent ductus arteriosus on left anterior descending coronary artery blood flow using transthoracic Doppler echocardiography. *Journal of the American Society of Echocardiograph*, 17, 659–663.

Hayashi, K., Sicard, G., Gellasch, K., Frank, J.D., Hardie, R.J., McAnulty, J.F., 2005. Cisterna chili ablation with thoracic duct ligation for chylothorax: results in eight dogs. *Veterinary Surgery* 34, 519–523.

Hoffman JI, Kaplan S. The incidence of congenital heart disease. *J Am Coll Cardiol* 2002;39:1890–1900.

Holt, D.E., Saunders, H.M., Schelling, C., Orsher, R.J., Diagnosis of portosystemic shunts: correlation of ultrasound with surgical, portographic and necropsy findings: 63 cases (1987–1993). *Veterinary Surgery.* 1994. 23, 403.

Hottinger, H.A., Walshaw, R., Hauptman, J.G., Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. *Veterinary Surgery.* 1995. 24, 331–336.

Hunt GB, Church DB, Malik R, et al. A retrospective analysis of congenital cardiac anomalies (1977–1989). *Australian Veterinary Journal* 1990:70–75.

Hunt, G.B., Bellenger, C.R., Pearson, M.R.B., Transportal approach for attenuating intrahepatic portosystemic shunts in dogs. *Veterinary Surgery.* 1996. 25, 300–308.

Hunt, G.B., Hughes, J.,. Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. *Australian Veterinary Journal* 1999. 77, 303–307.

Immer F.F., Stocker F., Seiler A.M., Pfammater J.P., Bachmann D., Printzen G., Carrel T., Troponin-I for prediction of early postoperative course after pediatric cardiac surgery. *Journal of the American College of Cardiology*, 1999. 33, 1719–1723.

Kim, I.O., Cheon, J.E., Kim, W.S., Chung, J.W., Yeon, K.M., Yoo, S.J., Seo, J.K., Choi, J.H., Congenital intrahepatic portohepatic venous shunt: treatment with coil embolisation. *Pediatric Radiology* 2000. 30, 336–338.

Kittleson MD. Patent ductus arteriosus. In: Kittleson MD, Kienle RD, eds. *Small Animal Cardiovascular Medicine*. St Louis, MO: Mosby; 1998:218–230.

Kudo, M., Tomita, S., Tochio, H., Minowa, K., Todo, A., Intrahepatic portosystemic venous shunt: diagnosis by color Doppler imaging. *The American Journal of Gastroenterology*. 1993. 88, 723–729.

Lafond, E., Weirich, W.E., Salisbury, S.K., 2002. Omentalization of the thorax for treatment of idiopathic chylothorax with constrictive pleuritis in a cat. *Journal of the American Animal Hospital Association* 38, 74–78.

Lamb, C., Ultrasonography of portosystemic shunts in dogs and cats. *Vet. Clin. North Am. Small Anim. Prac*. 1998. 28, 725–753.

Lamb, C.,. Ultrasonographic diagnosis of congenital portosystemic shunts in dogs: results of a prospective study. *Veterinary Radiology and Ultrasound.* 1996. 37, 281–288.

Lamb, C; Morphology of congenital intrahepatic portocaval shunts in dogs and cats. *Veterinary Record*, 1988 142: 55-60.

Lawrence, D., Bellah, J.R., Diaz, R.,. Results of surgical management of portosystemic shunts in dogs: 20 cases (1985–1990*). Journal of the American Veterinary Medical Association.* 1992. 201, 1750–1753.

Leveille, R., Pibarot, P., Soulez, G., Wisner, E.R. Transvenous coil embolisation of an extrahepatic portosystemic shunt in a dog: a naturally occurring model of portosystemic malformations in humans. *Pediatric Radiology* ., 2000. 30, 607–609.

MacDonald KA. Congenital heart diseases of puppies and kittens. *Vet Clin North Am Small Anim Prac*t 2006;36:503–531,

MacDonald, N.J., Noble, P.J.M., Burrow, R.B., 2008. Efficacy of en bloc ligation of the thoracic duct: descriptive study in 14 dogs. *Veterinary Surgery* 37, 696–701.

Monnet, E., 2003. Pleura and pleural space, third ed.. In: Slatter, D. (Ed.), *Textbook of Small Animal Surgery*, vol. 1 Saunders, Philadelphia, PA, USA, pp. 387–405.

Mulvihill JJ, Priester WA. Congenital heart disease in dogs: Epidemiologic similarities to man. *Teratology* 1973;7:73–77.

Partington, B.P., Partington, C.R., Biller, D., Toshach, K.,. Transvenous coil embolisation for treatment of patent ductus venosus in a dog. *Journal of the American Veterinary Medical Association*. 1993. 202, 281–284.

Patterson DF, Haskins ME, Schnarr WR. Hereditary dysplasia of the pulmonary valve in Beagle dogs. Pathologic and genetic studies. *Am J Cardiol* 1981;47:631–641.

Patterson PD. Two hereditary forms of ventricular obstruction in the dog: Pulmonary valve dysplasia and discrete subaortic stenosis. In: Nora J, Takao A, eds. *Congenital Heart Disease. Causes and Processes*. Mount Kisco: Futura Publishing Co; 1984:43–63.

Perez de Prado, A.; Pérez Martínez,C.; Cuellas Ramón,C.; Gonzalo Orden, JM.; Rodríguez Altónaga, J.A.; Garcia Iglesias, MJ.; Regueiro Purriños,M.; Orden, MA. Garcia Marin,JF.;Fernández-Vázquez,F. Endothelialization of Coronary Stents: What Happens when the Strut is Located Over the Ostium of a Side Branch?. Congreso: Transcatheter Cardiovascular Therapies (TCT**)** Washington October 2007. *Am.J. Cardiol.* 2007; 100(8) 177L.

Perez de Prado, A.; Pérez Martínez,C.; Cuellas Ramón,C.; Gonzalo Orden, JM.; Rodríguez Altónaga,J.A.; García Iglesias,M.J.; Regueiro, M.; A. Orden,MA.; Fernández-Vázquez,F.“Time pattern of stent endothelization in a swine coronary model: characterization and quantification**.** Congreso Transcatheter Cardiovascular Therapies (TCT), Washington, Octubre 2007. *Am J Cardiol*. 2007; 100(8): 177L.

Pérez de Prado, A.; Pérez Martínez,C.; Cuellas Ramón,C.; Gonzalo Orden, JM.; Rodríguez Altónaga, J.A.; Regueiro Purriños, M.; García Iglesias, M.J.; Fernández Vázquez,F.; . Endotelización de los stents coronarios: ¿Qué sucede con la malla colocada sobre la salida de las ramas laterales**?** Congreso Nacional de las Enfermedades Cardiovasculares, Madrid, Octubre 2007. *Rev Esp Cardiol* 2007;60 Supl 2 / pág. 118.

Pérez de Prado, A.; Pérez Martínez,C.; Cuellas Ramón,C.; Gonzalo Orden, JM.; Rodríguez Altónaga,J.A Regueiro Purriños, M.; García Iglesias, M.J.; Fernández Vázquez,F.- Patrón temporal de endotelización del stent en modelo coronario porcino: caracterización y cuantificación. Congreso Nacional de las Enfermedades Cardiovasculares, Madrid, Octubre 2007. *Rev Esp Cardiol* 2007;60 Supl 2 / pág. 63.

Perez Rivero,A.- *Hepatología y Cirugía Hepática en pequeños animales y exóticos.* Edita SErvet.ISSN: 97884Edita SErvet.ISSN: 978 84-92569.86-1 (2012).

Pisek G. Report of a chylothorax. *Arch Pediatr.* 1917;34:929-37.

Popelova´ J, Oechslin E, Karemmerer H, et al. Ventricular septal defect. In: Popelova J, Oechslin E, Karemmemer H, Sutton M, eds. *Congenital Heart Disease in Adults*. London: Informa UK Ltd; 2008:32–39.

Pyle RL, Patterson DF, Chacko S. The genetics and pathology of discrete subaortic stenosis in the Newfoundland dog. *Am Heart J* 1976; 92:324–334.

Schneider M., Schneider I., Hildebrandt N., Wehner M., Percutaneous angiography of Patent Ductus Arteriosus in dogs: techniques: results and implications for intravascular occlusion. *Journal of Veterinary Cardiolgy*, 2003. 2, 21–27.

Serrano Soto, AM**.-.** El cerdo como modelo animal experimental en toracoscopia: aplicaciones en cirugía veterinaria. *Tesis doctoral* **.** Universidad de Leon. 2005**.**

Slatter, DH (ed): *Textbook of Small Animal Surgery.* WB Saunders Philadelphia. 1989

Sleeper M.M., Clifford C.A., Laster L.L., Cardiac troponin I in the normal dog and cat. *Journal of Veterinary Internal Medicine*, 2001. 15, 501–503.

Spratt D.P., Mellanby R.J., Drury N., Archer J., 2005. Cardiac troponin I: evaluation of a biomarker for the diagnosis of heart disease in the dog. *Journal of Small Animal Practice*, 46, 139-145

Swalec KM. Portsystemic shunts. *In: Bojrab MJ, Ed. Disease mechanisms in small animal surgery*. 2nd Ed. Philadelphia: Lea and Febiger, 1993; 298-305

Szatmari V., Rothuzen, J.- Ultrasonographic identification and characterización of congenital portosystemic shunts and portal hypertensive disorders in dogs and cat- In *.Rothuzen J.; Bunch SE-:Charles, JA.- WSAVA Standards for clinical and histological diagnosis of canine and feline disease,* WSAVA liver stanndization group. Saunders Elsevier 2006 pp 15-40.

Thomas WP, Gaber CE, Jacobs GJ, et al. Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. Echocardiography Committee of the Specialty of Cardiology, American College of Veterinary Internal Medicine. *J Vet Intern Med* 1993;7:247–252.

Tidholm A. Retrospective study of congenital heart defects in 151 dogs. *J Small Anim Pract* 1997;38:94–98.

Tobias, K.M., 2002. In: Slatter, D. (Ed.), *Textbook of Small Animal Surgery, third ed.* W.B. Saunders, Philadelphia, PA, USA, pp. 727– 752.

Tobias, K.M., Rawlings, C.A., 1996. Surgical techniques for extravascular occlusion of intrahepatic shunts. *The Compendium on Continuing Education for the Practicing Veterinarian* 18, 745–755.

Van Israel N, Dukes-McEwan J, French AT. Long-term follow- up of dogs with patent ductus arteriosus. *J Small Anim Pract* 2003;44:480–490.

Vara Manso,J.; Rodríguez García, MA.; Iglesias Gárriz, I.; Fernández Vázquez, F.; Cuellas Ramón, JC.; Pérez de Prado, A.; J. Gonzalo Orden, JM.; Rodríguez Altónaga, JA.- Strain 2D en la valoración de la isquemia miocárdica aguda. Congreso Nacional de Enfermedades cardiovasculares. Madrid Octubre 2007.*Rev Esp Cardiol* 2007;60 Supl 2 / pág. 176

Viehoff, F.W., Stokhof, A.A., 2003. En bloc ligatie van de ductus thoracicus bij twaalf honden met idiopatische chylothorax. *Tijdschrift Diergeneeskunde* 128, 278– 283.

Vogt, J.C., Krahwinkel Jr., D.J., Bright, R.M., Daniel, G.B., Toal, R.L., Rohrbach, B., Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using the ameroid constrictor. *Veterinary Surgery.* 1996. 25, 495–502.

Wang JK, Wu MH, Hwang JJ, Chiang FT, Lin MT, Lue HC. Transcatheter closure of moderate to large patent ductus arteriosus with the Amplatzer duct occluder. *Catheter Cardiovasc Interv*. 2007;69:572-8. .

Williams, J.M., Niles, J.D., 1999. Use of omentum as a physiologic drain for treatment of chylothorax in a dog. *Veterinary Surgery* 28, 61–65.

Winkler, J.T., Bohling, M.W., Tillson, D.M., Wright, J.C., Ballagas, A.J., 2003. Portosystemic shunts: diagnosis, prognosis, and treatment of 64 cases (1993–2001). *Journal of the American Animal Hospital Association* 39, 169–185.

Wrigley, R.H., Konde, L.J., Park, R.D., Lebel, J.L., Ultrasonographic diagnosis of portocaval shunts in young dogs. *Journal of the American Veterinary Medical Association* 1987.191, 421–424.

Yoshimitsu, K., Andou, H., Kudo, S., Matsuo, Y., Matsumoto, S., Nakao, T., Shimado, Y., 1993. Multiple intrahepatic portosystemic venous shunts: treatment by portal vein embolisation. *Cardiovascular and Interventional Radiology*. 1993. 16, 49–51.

Youmans, K.R., Hunt, G.B., 1998. Cellophane banding for the gradual attenuation of single extrahepatic portosystemic shunts in eleven dogs. *Australian Veterinary Journal*. 1988. 76, 531–537.

ANEXOS :

ANEXO I.- Artículos presentados para la obtención de la tesis por compendio de publicaciones.

Bussadori, R.; Bussadori C, Millán L, Costilla S, Rodríguez-Altónaga JA, Orden MA, Gonzalo-Orden JM.- Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs.- *The Veterinary Journa*l. 2007. Volume: 176.Issue: 2.Pages**:** 221-226

Bussadori R., Tamborini A, Locatelli C, Palermo V, Brambilla PG.- Troponin I perioperative trend in dogs undergoing the correction of patent ductus arteriosus: preliminary investigations.. *Vet Res Commun*. 2008. Sep;32 Suppl 1:S255-8.

Bussadori R, Provera A, Martano M, Morello E, Gonzalo-Orden JM, Rosa GL, Stefano N, Maria RS, Sara Z, Buracco P. Pleural omentalisation with en bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats. *The Veterinary Journal*. 2011 May;188(2):234-6. Epub 2010 Jun 18.

ANEXO II.- Otros artículos y comunicaciones del doctorando, que están relacionados con los temas de la tesis, pero que no se han seleccionado para la presentación de las misma por publicaciones.

P. Oliveira, O. Domenech, J. Silva, S. Vannini, **R. Bussadori**, and C. Bussadori.- Retrospective Review of Congenital Heart Disease in 976 Dogs.- *J. Vet. Intern. Med*.- 2011 May;25(3):477-83.

Rossi F, Domenech O , **Bussadori R** , Bussadori C .- Role of multi-modality imaging to guide procedures of transvenous coil embolisation for the treatment of canine intrahepatic porto-systemic shunt.-: *Veterinary Radiology & Ultrasound* . 2010.Volume: 51 Issue: 2 Pages: 198-198.

Available online at www.sciencedirect.com
ScienceDirect
The Veterinary Journal 176 (2008) 221–226
The Veterinary Journal
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Transvenous coil embolisation for the treatment of single congenital portosystemic shunts in six dogs

Roberto Bussadori [a], Claudio Bussadori [b], Lorena Millán [a], Serafín Costilla [c], José Antonio Rodríguez-Altónaga [a], María Asunción Orden [a], José Manuel Gonzalo-Orden [a,*]

[a] *Department of Surgery and Radiology, Faculty of Veterinary Medicine, University of León, 24071 León, Spain*
[b] *Cardiovascular Medicine Institute Fondazione Ospedale Maggiore, University of Milan, Milan, Italy*
[c] *Diagnostic Imaging Service, Complejo Asistencial de León, 24071 León, Spain*

Accepted 24 February 2007

Abstract

This article describes the treatment of single congenital portosystemic shunts (CPSs) (intrahepatic and extrahepatic) using an interventional radiology technique involving embolisation of anomalous vessels with percutaneous coils. Briefly, a multipurpose catheter was introduced into the caudal vena cava and then into the portosystemic shunt. An autoexpandable stent was placed in the caudal vena cava, next to the shunt, in order to avoid coil migrations, and a cobra-like vascular catheter was used to pass through the stent and to place the coils in the shunt. This technique was used for treatment of CPS in six dogs. The results indicate that percutaneous embolisation of a CPS using coils, a less invasive technique than the traditional surgical technique, may result in complete closure of the anomalous vessel without development of portal hypertension.

Keywords: Portosystemic shunt; Interventional radiology; Canine; Liver; Minimal access surgery

1. Introduction

Congenital portosystemic shunt (CPS) is one of the most common vascular anomalies in dogs and is much more common in this species than in humans (Yoshimitsu et al., 1993; Golli et al., 2000). It results from persistence of a fetal vessel that normally closes after birth or from abnormal communication, during growth, between the cardinal and vitelline venous systems. There are two kinds of CPSs: intrahepatic and extrahepatic. Intrahepatic shunts are divided into the following categories: (1) left divisional shunt, a relatively consistent, bent tubular shape that drains into the left hepatic vein; (2) central divisional shunt, a foramen between dilated portions of the intrahepatic portal vein and the caudal vena cava and (3) right divisional shunt, a large and tortuous vessel (Lamb, 1988). Dogs with CPS have signs related to hepatic encephalopathy or may remain clinically normal. Hepatic encephalopathy depends on numerous factors, including local accumulation of toxins in the brain (Swalec, 1993).

Medical treatment is only palliative. The only definitive treatment is surgical or interventional occlusion of the anomalous vessel. Complete or partial ligation of the anomalous vessel, the traditional surgical technique, is relatively easy to perform with extrahepatic shunts but may be technically more difficult with intrahepatic shunts (Tobias and Rawlings, 1996; Hunt and Hughes, 1999). However, complete ligation of the shunt in a single procedure is not possible in some shunts, where haemodynamic parameters indicate a risk of acute portal

* Corresponding author. Tel.: +34 987291219; fax: +34 987291269.
E-mail address: dmajgo@unileon.es (J.M. Gonzalo-Orden).

1090-0233/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.tvjl.2007.02.027

hypertension that can cause death. Partial ligation however, which leaves a more or less elevated residual flow, does not allow complete remission of clinical signs and requires careful intra-operative assessment of haemodynamic parameters to determine the point of optimal occlusion. A second surgery, two or three months later, is recommended by some surgeons in order to attempt full ligation (Burton and White, 2001). In recent years, use of the ameroid constrictor (Vogt et al., 1996) and cellophane banding (Youmans and Hunt, 1998) have produced excellent results with regard to surgical correction of shunts. The only disadvantage of these techniques is that more extensive dissection around the anomalous vessel is required.

Partington et al. (1993) described the use of a transvenous coil embolisation procedure for the treatment of a congenital intrahepatic portosystemic shunt for the first time in a 3-month-old dog. Leveille et al. (2000) were the first to report the use of a similar technique in a dog with an extrahepatic CPS. In the same year, our group described a modified procedure combining coils and stents for the treatment of an intrahepatic CPS (Gonzalo-Orden et al., 2000). In this article, we describe the treatment of single congenital intrahepatic or extrahepatic portosystemic shunts in six dogs using an interventional radiology technique involving embolisation of anomalous vessels with percutaneous coils.

2. Materials and methods

All dogs were referred to the Faculty of Veterinary Medicine at the University of León with a presumptive or definitive diagnosis of CPS. Diagnoses were based on clinical signs, blood tests, and urinalysis and were confirmed by ultrasound identification of the anomalous vessels. Clinical signs were typical of this disease: intermittent anorexia, diarrhoea, ascites, lethargy, and hepatic encephalopathy signs. Laboratory studies showed elevated aspartate aminotransferase, alanine aminotransferase, and alkaline phosphatase levels; decreased blood urea nitrogen concentration; and high preprandial blood ammonia levels. Ammonium urate crystals and an elevated protein content in urine were also present in two dogs (identified as Nos. 1 and 2). We also measured bile acid concentration during fasting and 2 h after eating.

Hepatic ultrasound can examine, non-invasively, the internal architecture of the hepatic parenchyma and the portal and hepatic vessels. The liver was scanned in the transverse and longitudinal planes by placing the transducer just distal to the xiphoid and angling the transducer beam cranially (Lamb, 1998). In all dogs, we used right intercostal windows, which turned out to be particularly useful in dogs with central or right divisional shunts. Two ultrasound machines were used: a General Electric Logic 500 MD equipped with 5.0- and 7.5-MHz phased array transducers and an Esaote Caris equipped with two multifrequency probes, one 5–7.5 MHz and one 7.5–10 MHz annular array. Colour Doppler was used to show blood flow direction and communication between vessels. Colour Doppler imaging combined with pulsed Doppler examination is a non-invasive procedure that is highly effective in diagnosing CPS (Kudo et al., 1993; Yoshimitsu et al., 1993).

In one dog (No. 1), magnetic resonance imaging (MRI) (Signa Profile, General Electric) was used to confirm the size and morphology of the shunt. The dog was placed in the magnetic resonance unit feet first and supine. The head coil was used during the examination, and spin-echo axial and coronal T1-weighted series, axial two-dimensional time-of-flight series, and three-dimensional reconstruction with maximum intensity projection were performed. Abdominal MRI was performed from the kidneys to the diaphragm.

All dogs were anaesthetized with midazolam (Roche, 0.25 mg/kg, IM) followed by propofol (Abbott, 2–4 mg/kg, IV) and maintained with isoflurane (Abbott) in oxygen in a semiclosed circle system. For the vascular venous approach, we used the Seldinger technique, and a vascular introducer (5–7 F, depending on the size of the dog) was placed into the femoral vein or the saphenous vein. A pigtail catheter with multiple holes was placed into the caudal vena cava using a 0.038-inch (0.97 mm) straight guide wire (Terumo). During cavography, the presence of flow turbulence of the portal vein makes it possible to determine the position of the shunt (Fig. 1).

After localisation of the shunt, a cobra-shaped angiographic catheter (Cordis Corporation) was introduced into the shunt through the vena cava and was pushed to the portal vein. At that time, selective angiography of the shunt was performed. Portography using digital subtraction showed the morphology and size of the shunt (Fig. 2). Using a radiopaque ruler, we were able to determine the real size of the shunt. We then chose the diameter and the approximate number of coils to be used. In addition, we measured the diameter of the vena cava in order to choose the correct stent diameter. Subsequently, an autoexpandable stent (expander; Bolton Medical) was placed in the vena cava with its centre at the point of the shunt to avoid migration of coils. The cobra-shaped catheter was used to go through the stent and to leave the coils (occluding spring embolus; William Cook Europe) in the shunt (Fig. 3). At regular intervals, almost after leaving each coil, a small amount of contrast was injected to evaluate flow reduction through the shunt.

The process was considered finished when the coils took up >75% of the shunt's diameter. At this time, the flow to the liver was converted into evident in the radioscopic image. Only when flow reduction was too effective and contrast was present for more than 3 s in the portal system, <75% of the shunt's diameter was closed. Finally, the catheter and the introducer system were removed, and the dogs recovered from the anaesthesia. Weekly ultrasounds were performed until complete closure of the shunt was confirmed. Haematological analyses were performed 6 months and 1 year after the intervention.

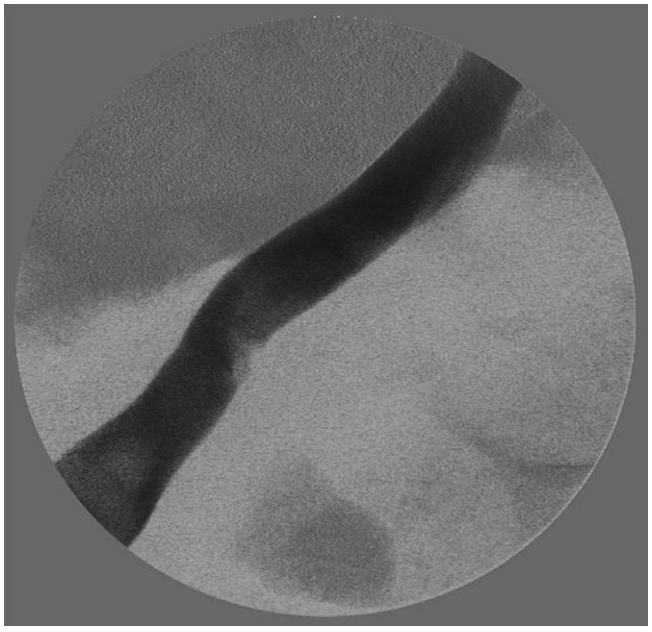

Fig. 1. Angiography of the cava. Note the lateral defect in the contrast column caused by a high-flush portosystemic shunt.

Fig. 2. Estimation of the morphology and size of the portosystemic shunt using retrograde portography.

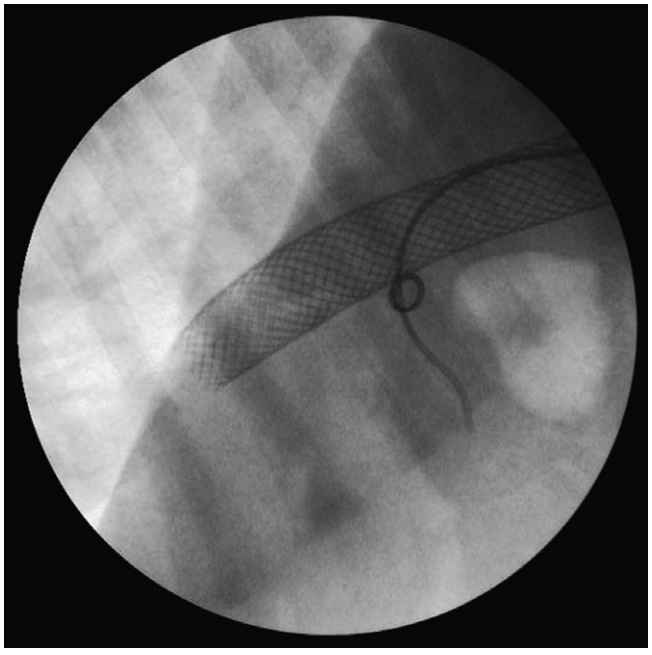

Fig. 3. Release of the coils into the shunt by means of a cobra-shaped catheter passing through the stent in the vena cava.

3. Results

Congenital portosystemic shunt was diagnosed by ultrasonography in all dogs. Ultrasonographic findings included small liver, decreased size and number of portal vascular structures within the liver, increased diameter of the caudal vena cava, and dilatation of the portal vein. Further examination using MRI was performed in one dog. The intrahepatic portocaval shunt was identified, confirming the presence of the anomalous vessel (Fig. 4). Magnetic resonance imaging yielded information about the morphology and position of the shunt that was useful in preparing the surgery, and cavography was no longer necessary.

The transvenous coil embolisation technique was quick and safe: the median intervention time was 58 min (minimum, 30 min; maximum, 120 min), and all dogs recovered from the anaesthesia without complications and were returned to their owners the day after the procedure. The technique resulted in complete occlusion of the CPS in 5/6 treated dogs in a median time of 6 weeks (minimum, 1 month; maximum, 2 months) (Fig. 5).

Clinical signs reported, abdominal ultrasonographic findings, estimated diameters of the shunt and the vena cava, number of embolisation procedures and number of coils used and the follow-up of the six dogs are presented in Table 1.

Fig. 4. Magnetic resonance imaging of a congenital portosystemic shunt in a dog. The imaging allowed identification of intrahepatic portocaval communication and provided important information about the morphology and position of the shunt.

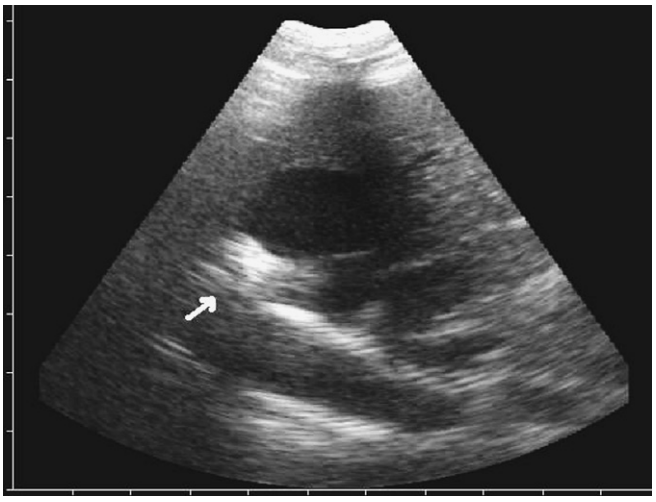

Fig. 5. Ultrasound showing complete occlusion of a congenital portosystemic shunt in a dog 1 month after transvenous coil embolisation.

In dog No. 1 embolisation of the shunt was done in two phases. In the first phase, embolisation was performed without placing the cava stent. The coil did not remain in the anomalous vessel but migrated to a peripheral pulmonary artery, with no effect on pulmonary circulation. A stent was then introduced into the caudal vena cava through the jugular vein, next to the anomalous vessel, to avoid further migration, and another coil was placed in the shunt. In the second phase, one month after the first attempt, two coils were introduced into the shunt with a catheter that was passed through the saphenous vein. Weekly ultrasounds were performed, and 2 weeks after the second intervention, the portal vein had increased in size and the ammonia concentration was in the normal range but the shunt was not completely closed. One month after the second intervention, the dog was clinically normal and the anomalous vessel was still minimally pervious; two months after the second intervention, the vessel was completely closed.

Dog No. 4 had a significant extrahepatic shunt and hepatic degeneration cirrhosis. In this case, because the hepatic condition predisposed the dog to portal hypertension, we decided to be more conservative and we only reduced the shunt's diameter by 50%, instead of 75%, during the interventional procedure. After initial improvement during the first 3 months following the intervention, the dog suffered again a hepatic encephalopathy crisis and was managed medically with lactulose (Duphalac, Solvay, 20 mL four times daily) and a commercial reduced protein diet (Hill's l/d diet). However, 6 months after partial occlusion of the shunt, the dog was manifesting serious hepatic insufficiency. At this point, the owner of the animal decided to stop the treatment and the dog was euthanased. At necropsy, liver examination revealed severe hepatic cirrhosis (Fig. 6).

Table 1
Treatment of canine portosystemic shunts by transvenous coil embolisation in six dogs: clinical signs, abdominal ultrasonographic findings, estimated diameters of the shunt and the vena cava, number of procedures and coils used and follow-up

Case no.	Signalment	Previous history	Abdominal ultrasonographic findings	Estimated diameters (mm)		Number of procedures	Number of coils	Follow-up/outcome
				Cava	Shunt			
1	Male 5 months Alaskan malamute	Two episodes of neurological disturbances compatible with hepatic encephalopathy	Right intrahepatic shunt	16	11.7	2	3	Anomalous vessel completely closed 2 months after surgery. Normal life for 7 months. Died of intestinal occlusion after ingesting a sock
2	Male 5 months Cocker spaniel	Frequent episodes of vomiting and diarrhoea. Polyuria and haematuria	Left intrahepatic shunt; 2 cm diameter urolith (ammonium urate)	14	9	1	3	Urolithiasis did not recur. The shunt was completely closed 6 weeks after the surgery. No clinical signs and normal haematological values at 1 and 2 years
3	Female 5 months Doberman	Severe hepatic encephalopathy	Right intrahepatic shunt	11	8.7	1	5	The shunt was completely closed 2 months after the surgery. No clinical signs and normal haematological values at 1 and 2 years
4	Female 1 year Collie	Severe hepatic encephalopathy, recurrent vomiting, diarrhoea and insufficient nutritional status	Extrahepatic shunt and hepatic degeneration cirrhosis	20	15	1	4	Six months after partial occlusion of the shunt, the dog was manifesting serious hepatic insufficiency and was euthanased
5	Male 8 months Brittany spaniel	Hepatic encephalopathy	Aneurysmal shunt	10	13	1	9	The shunt was completely closed 1 month after surgery. No clinical signs but abnormal haematological findings at 2 years
6	Male 5 months Giant schnauzer	Apathy and slightly insufficient nutritional status	Left intrahepatic shunt	15	8	1	6	The shunt was completely closed 1 month after the surgery. No clinical signs and normal haematological values at 1 and 2 years

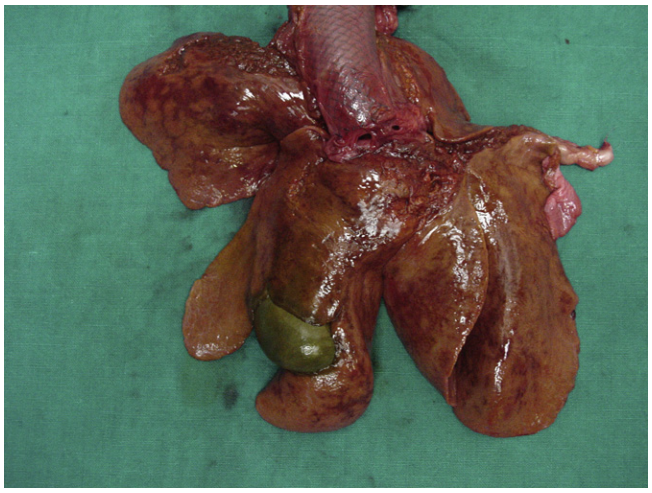

Fig. 6. Severe hepatic cirrhosis at necropsy of a dog euthanased 6 months after partial ligation of an extrahepatic shunt by transvenous coil embolisation.

4. Discussion

Ultrasonography is currently the best non-invasive method for assessing the internal structure of the liver, including the portal and hepatic vascular supply, without anaesthetising the dog. Congenital portosystemic shunt was diagnosed by ultrasonography in all dogs. As previously described (Wrigley et al., 1987), ultrasound allows determination of the position, diameter, and length of the shunt, and thus we could minimise risk during diagnosis and during monitoring after surgery. Moreover, as shown by one of the dogs (No. 4), ultrasonography of the liver can be useful for identifying lesions related to progression of parenchymal sclerosis and cirrhosis. In addition, MRI produced a three-dimensional image of the shunt, which helped us perform aneurysmal dilation in the correct place in the portal vein and vena cava and the anomalous vessel between them. We believe that this technique is less invasive than portography but is useful only in cases in which sonography is not satisfactory, because the intervention is expensive and must be performed under general anaesthesia.

The surgical attenuation of the shunting vessel is the treatment of choice for CPS (Tobias, 2002). One-stage suture complete ligation or progressive complete ligation is recommended for patients that can tolerate it, whereas progressive attenuation or partial suture ligation followed by complete ligation at a second surgery is recommended otherwise. Either of these approaches enables the liver to adapt to the increase in portal flow before complete closure, avoiding portal hypertension and splanchnic congestion (Hottinger et al., 1995; Hunt et al., 1996).

There is no ideal established technique to treat this pathology, but we believe that transvenous embolisation could become the method of choice. It is a minimally invasive procedure that allows progressive closure of the anomalous vessel over 1–2 months. This technique is likely to be useful in dogs with intrahepatic shunts where conventional surgical techniques depend greatly on the surgeon's skill and experience and are time consuming particularly in small-breed dogs (Asano et al., 2003). Moreover, this technique could be indicated in any dog where the owner wishes to avoid one or more invasive surgical procedures. Although follow-up in this study was good with a median of two years, further data are required to truly assess the long-term implications of this technique. The possibility of re-canalisation after a long period of time or the increased risk of multiple acquired shunts due to chronic portal hypertension should be evaluated. However, since 1975, coil embolisation has been successfully used in numerous applications in human medicine, including treatment of this condition (Kim et al., 2000).

One of the most important disadvantages of this approach is the possibility of coil migration to the heart and lungs, as previously described (Partington et al., 1993; Gonzalo-Orden et al., 2000). In our first case, when we tried to treat the shunt using only an appropriately sized coil, the coil, migrated through the systemic circulation to the pulmonary artery because of the high flow through the shunt. Although this complication did not lead to clinical signs, we tried to avoid the problem by placing an autoexpandable stent at the vena cava as a barrier. This modified technique was adopted in all further procedures. We have found that placement of a stent in the vena cava is useful for preventing coil migration and permits greater flexibility with regard to utilisation of coils of different diameters. A higher number of smaller coils can be used; coils do not have to be adapted to the size of the shunt because there is no danger of migration. So in our cases, the number of coils delivered to the shunt varied from three to nine according to the diameter of the anomalous vessel. We had to determine the stent diameter and length to be used in each case carefully because the vena cava is usually dilated because of the additional flow that it receives from the shunt. Diameters of both the vena cava and the shunt were estimated in each dog by two methods: ultrasonography before surgery, and fluoroscopy during the intervention procedure. Although the results obtained by the two methods are usually comparable, we highly recommend the use of both measurement techniques because they yield complementary information before and during the surgery.

In our study, transvenous coil embolisation for the treatment of CPS was quick and safe, and all the dogs were returned to their owners the day after the procedure. Afterwards, a restricted protein diet was recommended for all dogs because of the liver condition before the intervention (not because of the presence of the shunt). There have been no reports of clinical signs of portosystemic communication since the surgery, and at the time of writing, four dogs are reported to have no clinical signs and their blood test results remain within normal ranges, with the exception of dog No. 5, whose pre- and post-surgical bile acid levels have always been variable. The failure of post-surgical values of bile acids to return to within the normal range could be a result of continued shunting or irreversible liver

pathology. Complete closure of the shunt in this dog was confirmed by ultrasonographic examination one month after surgery. The sensitivity of the ultrasonographic examination for the detection of portosystemic shunting has been reported in the literature to range from 67–98% (Holt et al., 1994; Lamb, 1996; Winkler et al., 2003) and several factors such as movement of the patient, a full gastrointestinal tract or operator experience may affect the result of this imaging technique. Therefore, we cannot rule out the presence of residual flow through the shunting vessel or the development of multiple acquired shunts as a result of chronic portal hypertension since further ultrasonographic examination was not performed. On the other hand, several studies have described that paired bile acid values did not return to the reference range in all operated dogs (Lawrence et al., 1992; Hunt and Hughes, 1999; Winkler et al., 2003). Winkler et al. (2003) have recently proposed that bile acids alone may not be adequate for postoperative monitoring unless normal postoperative values are established.

One of the dogs (No. 1) died from an unrelated cause 6 months after surgery. The dog had had a normal life, and the shunt was completely closed. Another dog was euthanased 6 months following the surgical procedure, after manifesting serious hepatic insufficiency. Severe hepatic cirrhosis developed in this dog, probably because treatment began when hepatic failure was too advanced.

5. Conclusions

The combined method of embolisation with coils and stent implantation in the caudal vena cava is a valuable procedure for the treatment of CPS in dogs. Moreover, this technique permits occlusion of the anomalous vessel in several interventions if necessary: the radiopacity of the coils allows immediate identification of the anomalous vessel in later procedures.

Acknowledgements

The authors express their appreciation to Mireya Santoyo for translating the manuscript into English.

References

Asano, K., Watari, T., Kuwabara, M., Sasaki, Y., Teshimo, K., Kato, Y., Tanaka, S., 2003. Successful treatment by percutaneous transvenous coil embolisation in a small-breed dog with intrahepatic portosystemic shunt. Journal of Veterinary Medicine Sciences 65, 1269–1272.

Burton, C.A., White, R.N., 2001. Portovenogram findings in cases of elevated bile acid concentrations following correction of portosystemic shunts. Journal of Small Animal Practice 42, 536–540.

Golli, M., Kriaa, S., Said, M., Belguith, M., Zbidi, M., Saad, J., Nouri, A., Ganouni, A., 2000. Intrahepatic spontaneous portosystemic venous shunt: value of color and power Doppler sonography. Journal of Clinical Ultrasound 28, 47–50.

Gonzalo-Orden, J.M., Altónaga, J.R., Costilla, S., Gonzalo Cordero, J.M., Millán, L., Orden Recio, M.A., 2000. A transvenous coil embolisation of an intrahepatic portosystemic shunt in a dog. Veterinary Radiology and Ultrasound 41, 516–518.

Holt, D.E., Saunders, H.M., Schelling, C., Orsher, R.J., 1994. Diagnosis of portosystemic shunts: correlation of ultrasound with surgical, portographic and necropsy findings: 63 cases (1987–1993). Veterinary Surgery 23, 403.

Hottinger, H.A., Walshaw, R., Hauptman, J.G., 1995. Long-term results of complete and partial ligation of congenital portosystemic shunts in dogs. Veterinary Surgery 24, 331–336.

Hunt, G.B., Hughes, J., 1999. Outcomes after extrahepatic portosystemic shunt ligation in 49 dogs. Australian Veterinary Journal 77, 303–307.

Hunt, G.B., Bellenger, C.R., Pearson, M.R.B., 1996. Transportal approach for attenuating intrahepatic portosystemic shunts in dogs. Veterinary Surgery 25, 300–308.

Kim, I.O., Cheon, J.E., Kim, W.S., Chung, J.W., Yeon, K.M., Yoo, S.J., Seo, J.K., Choi, J.H., 2000. Congenital intrahepatic portohepatic venous shunt: treatment with coil embolisation. Pediatric Radiology 30, 336–338.

Kudo, M., Tomita, S., Tochio, H., Minowa, K., Todo, A., 1993. Intrahepatic portosystemic venous shunt: diagnosis by color Doppler imaging. The American Journal of Gastroenterology 88, 723–729.

Lamb, C., 1988. Morphology of congenital intrahepatic portocaval shunts in dogs and cats. The Veterinary Record 142, 55–60.

Lamb, C., 1996. Ultrasonographic diagnosis of congenital portosystemic shunts in dogs: results of a prospective study. Veterinary Radiology and Ultrasound 37, 281–288.

Lamb, C., 1998. Ultrasonography of portosystemic shunts in dogs and cats. The veterinary clinics of North America. Small Animal Practice 28, 725–753.

Lawrence, D., Bellah, J.R., Diaz, R., 1992. Results of surgical management of portosystemic shunts in dogs: 20 cases (1985–1990). Journal of the American Veterinary Medical Association 201, 1750–1753.

Leveille, R., Pibarot, P., Soulez, G., Wisner, E.R., 2000. Transvenous coil embolisation of an extrahepatic portosystemic shunt in a dog: a naturally occurring model of portosystemic malformations in humans. Pediatric Radiology 30, 607–609.

Partington, B.P., Partington, C.R., Biller, D., Toshach, K., 1993. Transvenous coil embolisation for treatment of patent ductus venosus in a dog. Journal of the American Veterinary Medical Association 202, 281–284.

Swalec, K.M., 1993. Portosystemic shunts. In: Bojrab, M.J. (Ed.), Disease Mechanisms in Small Animal Surgery, second ed. Lea and Febiger, Philadelphia, PA, USA, pp. 298–305.

Tobias, K.M., 2002. In: Slatter, D. (Ed.), Textbook of Small Animal Surgery, third ed. W.B. Saunders, Philadelphia, PA, USA, pp. 727–752.

Tobias, K.M., Rawlings, C.A., 1996. Surgical techniques for extravascular occlusion of intrahepatic shunts. The Compendium on Continuing Education for the Practicing Veterinarian 18, 745–755.

Vogt, J.C., Krahwinkel Jr., D.J., Bright, R.M., Daniel, G.B., Toal, R.L., Rohrbach, B., 1996. Gradual occlusion of extrahepatic portosystemic shunts in dogs and cats using the ameroid constrictor. Veterinary Surgery 25, 495–502.

Winkler, J.T., Bohling, M.W., Tillson, D.M., Wright, J.C., Ballagas, A.J., 2003. Portosystemic shunts: diagnosis, prognosis, and treatment of 64 cases (1993–2001). Journal of the American Animal Hospital Association 39, 169–185.

Wrigley, R.H., Konde, L.J., Park, R.D., Lebel, J.L., 1987. Ultrasonographic diagnosis of portocaval shunts in young dogs. Journal of the American Veterinary Medical Association 191, 421–424.

Yoshimitsu, K., Andou, H., Kudo, S., Matsuo, Y., Matsumoto, S., Nakao, T., Shimado, Y., 1993. Multiple intrahepatic portosystemic venous shunts: treatment by portal vein embolisation. Cardiovascular and Interventional Radiology 16, 49–51.

Youmans, K.R., Hunt, G.B., 1998. Cellophane banding for the gradual attenuation of single extrahepatic portosystemic shunts in eleven dogs. Australian Veterinary Journal 76, 531–537.

Vet Res Commun
DOI 10.1007/s11259-008-9122-2

ABSTRACT

Troponin I perioperative trend in dogs undergoing the correction of patent ductus arteriosus: preliminary investigations

R. Bussadori · A. Tamborini · C. Locatelli · V. Palermo · P. G. Brambilla

Keywords Dog · Patent ductus arteriosus · Troponin I

Abbreviations

2D	B-dimensional
CHF	congestive heart failure
cTnI	cardiac troponin I
EDVI	end-diastolic volume index
ESVI	end-systolic volume index
PDA	patent ductus arteriosus

Introduction

PDA derives from anatomical and functional non-obliteration of the fetal ductus arteriosus (Bonagura and Lehmkuhl 1999; Buchanan 2001). Females are more frequently affected; it can be found in all breeds, German Shepherd is the most represented (Bonagura and Lehmkuhl 1999; Fossum 2004). The hallmark of the left to right shunting PDA is a continuous machinery-type heart murmur, best heard over the left cranial thorax in the left axillary region (Bonagura and Lehmkuhl 1999; Fossum 2004). In adult patients volume overloading of the left atrium and ventricle can result in congestive left CHF, whereas young subjects are often asymptomatic (Fossum 2004). 2D echocardiography evidences the PDA, its size and morphology, and the cardiac alterations due to volume overload:

R. Bussadori
Dipartimento di Chirurgia e Radiologia Veterinaria, Università di Leon, Leon, Spain

A. Tamborini · C. Locatelli · V. Palermo · P. G. Brambilla
Dipartimento di Scienze Cliniche Veterinarie, Facoltà di Medicina Veterinaria, Università di Milano, Milano, Italy

P. G. Brambilla (✉)
Dipartimento di Scienze Cliniche Veterinarie, Sezione di Clinica Medica e Diagnostica di Laboratorio, via Celoria 10, 20133 Milano, Italy
e-mail: paola.brambilla@unimi.it

increased EDVI and ESVI, increased left atrium to aortic root ratio and reduction of the shortening fraction (Bonagura and Lehmkuhl 1999; Fossum 2004). Spectral and color flow Doppler show continuous and turbulent retrograde flow in the main pulmonary artery, with a peak velocity of between 4 and 6 m/sec. Angiography enables classification of the PDA type, measurement of the duct and identification of the degree of shunt (Schneider et al. 2003). Unlike medical treatment, a surgical approach is effective; surgical legation of the duct or minimally invasive transcatheter techniques can be performed (haemostatic clips, coils, Amplatzer Ductal Occluder, Amplatzer Canine Ductal Occluder and Amplatzer Vascular Plug) (Bonagura and Lehmkuhl 1999; Buchanan 2001; Schneider et al. 2003; Fossum 2004). The closure of PDA results in preload reduction and acute variation in aorta and left ventricle load pressures (Harada et al. 2004). Consequently, cTnI, an intracellular myofibrillar protein, could be released by damaged myocardium cells.cTnI is currently considered the gold standard for the diagnosis of myocardial injury (Sleeper et al. 2001). Human literature reports that variations in cTnI serum level evidence a high prognostic value of the outcome in short and long term follow up, before and after open heart surgery in children (Immer et al. 1999; Fellahi et al. 2003). The cutoff point for the definition of the postoperative mortality risk was set in human patients who underwent cardiac surgery ((Immer et al. 1999; Fellahi et al. 2003).

In human patients cTnI increases in serum within 5–7 hours after the ischemic damage, reaches a peak within 24 hours and decreases to baseline level in 5–20 days (Immer et al. 1999; Sleeper et al. 2001; Spratt et al. 2005). A similar trend was also identified in dogs following experimental induced myocardial infarction (Sleeper et al. 2001). A cTnI serum level less than 0.07 ng/ml is considered normal in dogs (Stratus (r) CS STAT Fluorometric Analyzer-Dade Behring Incorporated) (Sleeper et al. 2001).

The aims of the present study were to evaluate whether the perioperative trend of cTnI in dogs is similar to that previously described in humans; whether the enzyme trend could be considered an indicator of myocardium cell distress induced by changes in haemodynamic conditions after PDA closure and finally, whether this occurrence could be considered transitory.

Materials and methods

Ten dogs of different breeds, sex, age and weight underwent PDA correction by surgical ligation (7 dogs) or minimally invasive transcatheter techniques (3 dogs). The size of the duct determined the choice of closure method.

All dogs were submitted to clinical examination, electrocardiogram, 2D echocardiography, Doppler studies, angiography and cTnI dosage before and 24 hours after correction of the PDA. At 48 hours and 6 months after closure only serum values of cTnI were evaluated (Sleeper et al. 2001).

Results

The study group consisted of dogs of various breeds, German shepherd dogs and cross breeds were overrepresented as well as females (90%). Eight dogs aged less than 13 months were asymptomatic, 2 adults (60 months old) presented symptoms of left CHF. Two groups were identified according to the cTnI trend.

GROUP 1 (8 dogs)-7 dogs showed a cTnI peak concentration within 24 hours (avg: 1.88 ng/ml) and 1 dog within 48 hours (0.1 ng/ml) after closure of the PDA. The highest values were found in adult dogs (2.23 ng/ml and 8.24 ng/ml)

In 6 dogs the cTnI concentration was normal before closure of the PDA, while it was out of the normal range in a 3 month old puppy (0.29 ng/ml) and one adult dog (0.39 ng/ml), of which preoperative ECG presented paroxysmal ventricular tachycardia and atrial and ventricular multiform premature complexes. In 6 subjects ESVI and EDVI values were outside the normal range (ESVI Min: 72, Max: 191 ml/m2; EDVI Min: 161, Max: 283 ml/m2). In 2 dogs, ESVI and EDVI values were slightly over normal limits.

GROUP 2 (2 dogs)-both patients presented perioperative cTnI level, ESVI and EDVI close to normal limits.

Considering the asyntomatic subject belonging to both groups, we detected the most elevated values of ESVI and EDVI (118 and 232 ml/m2) in the puppy before closure of the PDA and also the most significant difference between pre and post-intervention of the EDVI value (94 ml/m2); this puppy also showed the highest cTnI basal concentration. The end-point of the study, set at 6 months after closure, has already been reached by 4 dogs (group 1: 3 dogs, group 2: 1 dog); all of them, including the puppy that showed a cTnI basal level beyond normal limits, presented a cTnI concentration within the normal range.

Discussion

We have found that the cTnI concentration trend in dogs is analogous to that reported in human patients: in 7 dogs the plasma level of the marker increased after the closure of PDA and the peak was reached within 24 hours (Immer et al. 1999; Sleeper et al. 2001; Spratt et al. 2005). This suggests that acute variations in aorta and left ventricle pressure following PDA closure could cause myocardium cell damage and consequently an increase in cTnI plasma concentration (Harada et al. 2004).

This occurrence is more evident in subjects with a marked difference in ESVI and EDVI before and after PDA closure, and in patients with the basal ESVI close to normal limits.

The hypothesis is that myocardium cells might have undergone a post-occlusion stress pressure, and this could be assessed by following the perioperative trend of serum cTnI.

A cTnI concentration within normal limits was evident in subjects that had already achieved the end point of the follow up, even in the puppy with a cTnI basal level greater than 0.07 ng/ml.

This means that the damage within the myocardial cells could be considered transitory. More detailed information about the trend of cTnI due to an“iatrogenic post occlusion damage” will be obtained at the end of the follow-up of the two adult dogs. The high level of the enzyme concentration evident in one of the patients older than 60 months is a consequence of severe ventricular arrhythmias.

The main limitation of this study is the small number of subjects involved; consequently, our work should be considered as a preliminary study. However, the encouraging results obtained would suggest continuation, and planning of a prospective study on a larger clinical group. This could allow us to analyze the consequences of different techniques in correcting PDA, on heart and vascular system as well as to investigate the perioperative trend of cTnI, as has already been performed in human medicine. This will allow us to determine the predictive concentration and the cut-off values of cTnI, with the aim of establishing the level of surgical risk.

References

Bonagura J.D. and Lehmkuhl L.B., 1999. Congenital heart disease. In *Textbook of canine and feline cardiology*, P.R Fox, D.D. Sisson, N.S. Moise (Eds.), 2th edition. WB Saunders: Philadelphia, 471–535.

Buchanan J.W, 2001. Patent ductus arteriousus morphology, pathogenesis, types and treatment. *Journal of Veterinary Cardiolgy*, **1**, 7–16.

Fossum T.W., 2004. *Chirurgia dei piccoli animali*, 2th edition. Elsevier-Masson: Milano.

Fellahi J.L., Gue X., Richomme X., Monier E., Guillon L., Riou B., 2003. Short-and long-term prognostic value of postoperative cardiac troponin I concentration in patients undergoing coronary artery bypass grafting. *Anesthesiology*, **99**, 270–274.

Harada K., Toyono M., Tamura M., 2004. Effects of coil closure of patent ductus arteriosus on left anterior descending coronary artery blood flow using transthoracic Doppler echocardiography. *Journal of the American Society of Echocardiograph*, **17**, 659–663.

Immer F.F., Stocker F., Seiler A.M., Pfammater J.P., Bachmann D., Printzen G., Carrel T., 1999. Troponin-I for prediction of early postoperative course after pediatric cardiac surgery. *Journal of the American College of Cardiology*, **33**, 1719–1723.

Schneider M., Schneider I., Hildebrandt N., Wehner M., 2003. Percutaneous angiography of Patent Ductus Arteriosus in dogs: techniques: results and implications for intravascular occlusion. *Journal of Veterinary Cardiolgy*, **2**, 21–27.

Sleeper M.M., Clifford C.A., Laster L.L., 2001. Cardiac troponin I in the normal dog and cat. *Journal of Veterinary Internal Medicine*, **15**, 501–503.

Spratt D.P., Mellanby R.J., Drury N., Archer J., 2005. Cardiac troponin I: evaluation of a biomarker for the diagnosis of heart disease in the dog. *Journal of Small Animal Practice*, **46**, 139–145.

The Veterinary Journal 188 (2011) 234–236

Contents lists available at ScienceDirect

The Veterinary Journal

journal homepage: www.elsevier.com/locate/tvjl

Short Communication

Pleural omentalisation with en bloc ligation of the thoracic duct and pericardiectomy for idiopathic chylothorax in nine dogs and four cats

Roberto Bussadori [a], Alessandra Provera [a], Marina Martano [b], Emanuela Morello [b], Josè Manuel Gonzalo-Orden [c], Giuseppe La Rosa [b], Nicoli Stefano [b], Repetto Silvia Maria [a], Zabarino Sara [b], Paolo Buracco [b,*]

[a] Clinica Veterinaria Gran Sasso, Via Donatello 26, 20124 Milano, Italy
[b] Ospedale DidatticoVeterinario (ODV), Dipartimento di Patologia Animale, School of Veterinary Medicine, Grugliasco, Turin, Italy
[c] Departamento de Medicina y Cirugía Veterinaria, School of Veterinary Medicine, León, Spain

ARTICLE INFO

Article history:
Accepted 7 May 2010

Keywords:
Idiopathic chylothorax
Dog
Cat
Ligation of thoracic duct
Pericardiectomy
Omentalisation

ABSTRACT

Conventional treatment of idiopathic chylothorax (IC) involves thoracic duct (TD) ligation (with/without lymphagiography) combined with subphrenic pericardiectomy. Nine dogs and four cats with IC, which received intrathoracic omentalisation with TD en bloc ligation (not preceded by lymphangiography) and subphrenic pericardiectomy, were evaluated retrospectively. Seven of nine dogs and 3/4 cats were still alive and disease-free at the time of reporting (range 10–53 and 19–31 months, respectively). Clinical signs of IC did not decrease after the first surgery in one cat and one dog; in another dog clinical signs recurred after 5 months. Overall efficacy rate of this one-stage combined procedure was 77% (6 months), 73% (12 months), and 57% (24 months). Where a second surgery was performed in case of failure, the success rate in dogs was 89% (6 months) and 80% (24 months). Addition of pleural omentalisation to TD en bloc ligation and subphrenic pericardiectomy does not seem to improve results when compared with published data and at present does not seem advisable as a first choice.

Chylothorax is a debilitating disease in both dogs and cats. Several possible causes have been reported (Fossum et al., 1986, 2004), but a cause is frequently not recognized and chylothorax is considered as idiopathic (IC). Currently, the conventional treatment of IC involves prediaphragmatic thoracic duct (TD) ligation with or without pre-ligation lymphangiography combined with subphrenic pericardiectomy (Fossum et al., 1986, 2004). Intrathoracic omentalisation has been reported (Williams and Niles, 1999; Lafond et al., 2002; Adrega da Silva et al., 2009), but its association with conventional treatment has not been reported. Our goal was to evaluate retrospectively the outcome of intrathoracic omentalisation combined with prediaphragmatic en bloc ligation (not preceded by lymphangiography) of all caudal mediastinal tissue between the aorta and the thoracic vertebrae (including the TD and azygous vein), and subphrenic pericardiectomy.

Between January 2004 and June 2009 nine dogs and four cats with IC (diagnosis reached after exclusion of possible causes) received these three procedures at one stage (Table 1). A minimum follow-up of 6 months was conducted. All dogs were males and 2.5–8 year-old (median 4), and 7/9 were >28 kg. Cats were 4–7 year-old (median 4.75). Clinical sign duration was 1 week–13 months (median 4 months). Preoperative medical therapy was limited to a low fat diet. Work-up included chest radiographs, thoracocentesis and fluid analysis, ultrasonographic examination of thorax, heart and abdomen, and blood tests. A single or more thoracocenteses were performed to relieve respiratory signs, even just before anaesthesia.

Surgery consisted of (1) right (dogs) or left (cats) 9–10th intercostal thoracotomy, (2) undermining and en bloc ligation (with 1–3 3–0/2–0 polypropylene or silk sutures depending on animal size) of all structures dorsal to aorta and ventral to vertebral column (including TD and azygous vein but sparing the sympathetic trunk), without mesenteric lymphangiography, (3) subphrenic pericardiectomy (through a 4–5th intercostal thoracotomy), and (4) pleural space omentalisation. The omentum was moved intrathoracically without tension by dorsal transdiaphragmatic (10 animals) or costal-diaphragmatic (3 animals) aspiration by a Poole cannula inserted through a small tear that was then partially sutured. The omental sac was opened to extend the omentum cranially up to the cranial mediastinum. Omentopexy to the cranial and caudal mediastinum was performed with 3–0/4–0 synthetic absorbable monofilament sutures. The ventral caudal mediastinum was opened allowing communication between the two pleural cavities. Thickening of the lung serosa and pericardium was seen in 11 animals; in nine cases a biopsy of pericardium was subjected to histological

* Corresponding author. Tel.: +39 11 6709063; fax: +39 11 6709165.
E-mail address: paolo.buracco@unito.it (P. Buracco).

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doi:10.1016/j.tvjl.2010.05.010

Table 1
Summary of the 13 animals with idiopathic chylothorax and treated simultaneously with en bloc ligation of all caudal mediastinal tissue including the thoracic duct and azygos vein, pericardiectomy, and intrathoracic omentalisation.

Case number	Signalment	History (duration)	Postsurgical outcome	Follow-up
1	Male Dogue de Bordeaux, 2.5 years	Dyspnoea, coughing, progressive weight loss (5 months)	Resolution within 1 week	Alive and disease free after 45 months
2	Male Rottweiler, 3 years	Tachypnoea (2 months)	Resolution within 1 week	Alive and disease free after 53 months
3	Male Rottweiler, 3 years	Dyspnoea, progressive weight loss (13 months)	Resolution within 1 week. Recurrence after 5 months → no response to 2nd en bloc TD ligation	Euthanasia after 5 months due to failure of second surgery
4	Male Dogue de Bordeaux, 4 years	Casual diagnosis of chylothorax during a programmed clinical control (1 week – slight increase in breathing noted by owners)	Resolution within 1 week	Death after 6 months of gastric torsion
5	Male mongrel dog, 4 years	Dyspnoea, coughing, progressive weight loss (4 months)	Resolution within 1 week	Alive and disease free at 53 months
6	Male West Highland White Terrier, 5 years	Dyspnoea, progressive weight loss (4 months)	Persistent IC. Second surgery at day 18: en bloc ligation and cisterna chyli ablation	Alive and disease-free 35 months after the second surgery
7	Male Borzoi, 4 years	Dyspnoea, progressive weight loss (2 months)	Resolution within 1 week	Alive and disease free after 22 months
8	Male Bull Mastiff, 5 years	Dyspnoea (2 months)	Resolution within 1 week	Alive and disease free after 20 months
9	Male Doberman, 8 years	Dyspnoea (4 months)	Resolution within 1 month	Alive and disease free after 10 months
10	Male castrated domestic short hair cat, 5 years	Dyspnoea (1 month)	Resolution within 1 week	Alive and disease free at 31 months
11	Male castrated domestic short hair cat, 7 years	Dyspnoea, progressive weight loss (3 months)	Resolution within 1 week	Alive and disease free after 22 months
12	Female spayed domestic short hair cat, 4.5 years	Dyspnoea, coughing (5 months)	Resolution within 1 month	Alive and disease free after 19 months
13	Female spayed domestic short hair cat, 4 years	Dyspnoea, progressive weight loss (6 months)	Persistent IC	Owner declined further treatment. Euthanasia after 1 month

examination. A chest tube was installed. Postoperative management included administration of' intravenous (IV) crystalloids, buprenorphine (10 μg/kg three times a day) and carprofen (2 mg/kg twice a day) for 1–4 days. Animals were discharged from the clinic after removal of the thoracostomy tube (when less than 2 mL/kg/day of non-chylous fluid were withdrawn). This was possible in 11 animals within a mean of 5 days. Follow-up information was obtained through periodical clinical and radiographic examination or through the referring veterinarian (Table 1).

In all cases histology of pericardium was consistent with fibrosing serositis of varying severity. Resolution of IC occurred within 1 week in seven dogs and two cats and within 1 month in one dog and one cat. Chylothorax persisted after surgery in one cat and one dog. The thoracostomy tube was never removed from the cat, which was euthanised after 1 month; the dog had its tube removed at day 10 on owner's decision but was re-operated for en bloc ligation of TD and cisterna chyli ablation (Hayashi et al., 2005) 18 days after the first surgery; complete resolution of clinical signs occurred within 5 days (disease-free 35 months). Another dog showed recurrence of clinical signs 5 months after the first surgery and was re-operated for en bloc ligation of TD; the effusion decreased subsequently but did not resolve and the animal was euthanised at the request of the owner. Another dog died 6 months after surgery due to gastric torsion of unknown association with the previous treatment.

The survival range was 1–53 months. Seven of nine dogs (range 10–53 months) and 3/4 cats (19, 22 and 31 months, respectively) are alive and disease-free at the time of reporting. The overall success rate of the combined procedure was 77% at 6 months, 73% at 12 months, and 57% at 24 months. The overall success rate of surgery in dogs (i.e. considering also the two dogs that underwent two surgeries) was 89% at 6 months, 86% at 12 months, and 80% at 24 months.

IC is rare and most studies involve few animals. In addition, multiple procedures are typically performed at different times due to failure of the initial treatment. This makes an accurate estimation of the success rate of any single procedure challenging. Selective TD ligation (after lymphagiography) has a reported success rate of 20–80% (Fossum et al., 1986, 2004). If subphrenic pericardiectomy was also performed, the rate increased to 88.7–100% in dogs and 80% in cats (Fossum et al., 2004; Carobbi et al., 2008). En bloc ligation is an alternative to selective TD ligation with a reduced surgical time since lymphangiography is not performed; its reported success rate when used alone is 50% (Monnet, 2003; Viehoff and Stokhof, 2003). MacDonald et al. (2008) reported a 93% success rate of TD occlusion in normal dogs after TD en bloc ligation. Therefore, TD en bloc ligation combined with pericardiectomy may yield results overlapping those using selective TD ligation after lymphagiography and pericardiectomy.

In this retrospective study, en bloc ligation of TD and subphrenic pericardiectomy were combined with intrathoracic omentalisation. There was no difference in the amount of omentum transposed into the thorax between the two techniques (dorsal transdiaphragmatic and costal-diaphragmatic aspiration). Intrathoracic omentalisation has been proposed as a 'salvage' procedure when other techniques have failed (Williams and Niles, 1999; Lafond et al., 2002) or as an ancillary procedure in addition to TD ligation and pericardiectomy (Adrega da Silva et al., 2009). Results of this combined procedure when used as first-line treatment have not been reported. The role of the omentum in IC is controversial, as omental lymphatics eventually drain in the lymphatic-venous system involved in IC, even after TD closure. The other potential

roles of the omentum are angiogenesis and adhesion formation (Lafond et al., 2002). The success rate reported here overlaps with results already reported by others (cited previously in the text) that performed TD ligation and pericardiectomy with or without lymphangiography.

The main limitations of this study are the small number of animals included and the lack of a control group. Many animals responded well within the first postoperative week. However, the study failed to prove that pleural omentalisation can improve the long-term efficacy of the conventional treatment of IC (TD ligation plus pericardiectomy), when the results were compared with earlier published data. A comparative study with a larger number of animals is needed for a definitive answer. In practice, in case of IC, it seems reasonable to avoid lymphangiography and to perform en bloc ligation of all caudal mediastinal tissue including TD and pericardiectomy as a first choice for treatment. In case of failure, other procedures (e.g. cisterna chyli ablation and/or omentalisation) should be considered.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References

Adrega da Silva, C., Monnet, E., Bardet, J.F., 2009. Long-term clinical outcome of surgical treatment for idiopathic chylothorax in 10 dogs. In: Proceedings of the 18th Annual Scientific Meeting of the European College of Veterinary Surgeons, Nantes, France, pp. 289–291.

Carobbi, B., White, R.A.S., Romanelli, G., 2008. Treatment of idiopathic chylothorax in 14 dogs by ligation of the thoracic duct and partial pericardiectomy. Veterinary Record 163, 743–745.

Fossum, T.W., Birchard, S.J., Jacobs, R.M., 1986. Chylothorax in 34 dogs. Journal of American Veterinary Medical Association 188, 1315–1318.

Fossum, T.W., Mertens, M.M., Miller, M.W., Peacock, J.T., Saunders, A., Gordon, S., Pahl, G., Makarski, L.A., Bahr, A., Hobson, P.H., 2004. Thoracic duct ligation and pericardectomy for treatment of idiopathic chylothorax. Journal of Veterinary Internal Medicine 18, 307–310.

Hayashi, K., Sicard, G., Gellasch, K., Frank, J.D., Hardie, R.J., McAnulty, J.F., 2005. Cisterna chili ablation with thoracic duct ligation for chylothorax: results in eight dogs. Veterinary Surgery 34, 519–523.

Lafond, E., Weirich, W.E., Salisbury, S.K., 2002. Omentalization of the thorax for treatment of idiopathic chylothorax with constrictive pleuritis in a cat. Journal of the American Animal Hospital Association 38, 74–78.

MacDonald, N.J., Noble, P.J.M., Burrow, R.B., 2008. Efficacy of en bloc ligation of the thoracic duct: descriptive study in 14 dogs. Veterinary Surgery 37, 696–701.

Monnet, E., 2003. Pleura and pleural space, third ed.. In: Slatter, D. (Ed.), Textbook of Small Animal Surgery, vol. 1 Saunders, Philadelphia, PA, USA, pp. 387–405.

Viehoff, F.W., Stokhof, A.A., 2003. *En bloc* ligatie van de ductus thoracicus bij twaalf honden met idiopatische chylothorax. Tijdschrift Diergeneeskunde 128, 278–283.

Williams, J.M., Niles, J.D., 1999. Use of omentum as a physiologic drain for treatment of chylothorax in a dog. Veterinary Surgery 28, 61–65.

J Vet Intern Med 2011;■■:1–7

Retrospective Review of Congenital Heart Disease in 976 Dogs

P. Oliveira, O. Domenech, J. Silva, S. Vannini, R. Bussadori, and C. Bussadori

Background: Knowledge of epidemiology is important for recognition of cardiovascular malformations.

Objective: Review the incidence of congenital heart defects in dogs in Italy and assess breed and sex predispositions.

Animals: Nine hundred and seventy-six dogs diagnosed with congenital heart disease (CHD) of 4,480 dogs presented to Clinica Veterinaria Gran Sasso for cardiovascular examination from 1997 to 2010.

Methods: A retrospective analysis of medical records regarding signalment, history, clinical examination, radiography, electrocardiography, echocardiography, angiography, and postmortem examination was performed. Breed and sex predisposition were assessed with the odds ratio test.

Results: CHD was observed in 21.7% of cases. A total of 1,132 defects were observed with single defects in 832 cases (85%), 2 concurrent defects in 132 cases (14%), and 3 concurrent defects in 12 cases (1%). The most common defects were pulmonic stenosis (PS; 32.1%), subaortic stenosis (SAS; 21.3%), and patent ductus arteriosus (20.9%), followed by ventricular septal defect (VSD; 7.5%), valvular aortic stenosis (AS; 5.7%), and tricuspid dysplasia (3.1%). SAS, PS, and VSD frequently were associated with other defects. Several breed and sex predispositions were identified.

Conclusions and Clinical Relevance: The results of this study are in accordance with previous studies, with slight differences. The breed and sex predilections identified may be of value for the diagnosis and screening of CHD in dogs. Additionally, the relatively high percentage of concurrent heart defects emphasizes the importance of accurate and complete examinations for identification. Because these data are from a cardiology referral center, a bias may exist.

Key words: Breed predisposition; Canine; Congenital heart defects; Multiple heart defects; Sex predisposition.

Cardiovascular malformations represent a substantial cause of morbidity and mortality in dogs <1 year of age.[1] The exact prevalence of these malformations is difficult to determine because some do not cause audible cardiac murmurs, some lead to perinatal death, and regional differences in breeds affect their frequency. In humans, cardiovascular malformations represent the most common congenital anomalies.[2] Early recognition is of great importance to achieve appropriate medical or surgical management, improve outcome and provide an accurate prognosis. For this purpose, the epidemiology of cardiovascular defects plays an important role. Several reports exist in veterinary literature regarding the prevalence of congenital heart disease (CHD) in dogs, mostly from the United States, Australia, United Kingdom, Sweden, and Switzerland.[1,3–6] The most commonly reported defects in this species are patent ductus arteriosus (PDA), pulmonic (PS) and subaortic stenosis (SAS), ventricular septal defects (VSD), tricuspid dysplasia, and tetralogy of Fallot (TOF).[1,4,5] The aim of this study was to review the incidence of congenital heart defects in a large population of dogs in Italy.

Abbreviations:

AS	valvular aortic stenosis
ASD	atrial septal defect
CHD	congenital heart disease
PDA	patent ductus arteriosus
PS	pulmonic stenosis
SAS	subaortic stenosis
TOF	tetralogy of Fallot
TTE	transthoracic echocardiography
VSD	ventricular septal defect

Materials and Methods

The medical records of 4,480 dogs presented for cardiovascular examination at Clinica Veterinaria Gran Sasso between 1997 and 2010 were reviewed retrospectively. Cases that presented with congenital heart defects were identified, and information regarding signalment, history, clinical examination, radiography, electrocardiography, echocardiography, angiography, and postmortem examination was obtained.

A complete transthoracic echocardiographic examination (TTE) was performed in all patients. Echocardiographic examinations were performed with an Esaote Caris ultrasound machine with mechanical transducers ranging from 2 to 10 MHz,[a] or Esaote Megas, Esaote Mylab30Vet, Esaote MyLab60 ultrasound machines with electronic transducers also ranging from 2 to 10 MHz.[a] The exams were performed and interpreted by the authors and reviewed by an ECVIM board-certified cardiologist (C.B., O.D., or both). The patients were placed in right and left lateral recumbency and the examinations were performed according to the American Society of Echocardiography standards and guidelines and other published recommendations.[7] Angiographic procedures also were performed by the authors with a fluoroscopy system[b] in cases undergoing interventional percutaneous procedures or when necessary for diagnostic purposes. Postmortem examinations were performed under the supervision of C.B., O.D., or both.

SAS lesions were classified according to the Pyle & Patterson studies as type 1, when presenting with a thickened and raised septal endocardium to form small nodules; type 2, when presenting with a fibrous thickening and raising of the subaortic endocardium forming a complete, or incomplete, fibrous ring below the aortic valve; and type 3, when presenting with a concentrical ''tunnel-like'' lesion at the left ventricular outflow tract.[8]

From the Department of Cardiology of Clinica Veterinaria Gran Sasso, Milano, Italia. *The results from this study have been partially presented as an abstract at the 20th ECVIM Meeting 2010 in Toulouse, France.*

Corresponding author: Pedro Oliveira, DVM, Clinica Veterinaria Gran Sasso, Via Donatello 26, 20131 Milano, Italia; e-mail: pedro mpdo@gmail.com.

Submitted November 26, 2010; Revised January 15, 2011; Accepted February 2, 2011.

10.1111/j.1939-1676.2011.0711.x

PS lesions were classified as type A when presenting with pulmonic leaflet thickening and fusion, and type B when presenting with pulmonic ostium hypoplasia with varying degrees of leaflet thickening and fusion.[8]

Statistical Analysis

Descriptive statistics were performed. The Kolmogorov-Smirnov test was used to assess normality. The Mann-Whitney test was used to identify differences in breed distribution between the CHD group and a reference population composed of 7,780 dogs of the same age range presented to Clinica Veterinaria Gran Sasso during the same time period. Breed and sex predilections were assessed by means of calculation of the relative risk with the odds ratio test. Breed predilection was considered to be present if a statistically significant ($P < .05$) odds ratio > 1.5 was observed with the following classification: mild increased risk if $1.5 < OR < 2.9$, moderate risk if $3.0 < OR < 4.9$, and marked risk if $OR > 5.0$. Only breeds represented by a minimum of 4 dogs were tested, except in the case of tricuspid dysplasia, in which this limit was set to 3 due the lower number of cases. Data processing and statistics were performed by commercially available statistics software[c] and Microsoft Excel 2003.[d]

Results

CHD was diagnosed in 976 (21.7%) of the 4,480 dogs. Single defects were present in 832 cases (85%), 2 concurrent defects in 132 cases (14%), and 3 concurrent defects in 12 cases (1%), corresponding to a total of 1,132 heart defects. Observed congenital heart defects are presented in Table 1, including information regarding sex and age at presentation. The most common were PS (32.1%), SAS (21.3%), and PDA (20.9%), followed by VSD (7.5%), valvular aortic stenosis (AS) (5.7%), and tricuspid dysplasia (3.1%). Angiographic procedures were performed in 158 cases: 116 PS, 1 AS, 28 PDA, 1 VSD, 3 PS with anomalous right coronary artery, 1 PS with anomalous right coronary artery and SAS, 3 PS with persistent left cranial vena cava, 2 atrial septal defects (ASD), 1 reverse PDA, 1 anomalous subclavian artery, 1 anomalous pulmonary venous return. Postmortem examinations were performed in 43 dogs: 4 PDA, 7 type 1 SAS, 4 type 2 SAS, 3 type 3 SAS, 6 type A PS, 8 type B PS, 3 muscular VSD, 4 perimembranous VSD, 2 TOF, 1 case with 2 stenotic pulmonic arteries, 1 aortic-pulmonary window.

Seventy-seven breeds were represented, with Boxer (26%) and German Shepherd (10%) being the most common, followed by mixed breed dogs (9.9%), English Bulldog (3.7%), and Newfoundland (3.7%). A statistically significant difference between breed distribution in the CHD group and the overall population was observed ($P < .0001$). Purebred dogs showed a significantly higher probability of presenting with CHD when compared with Mongrel dogs (OR, 3.3). Additionally, a mild increased risk for CHD was observed for 7 breeds: Chihuahua (OR, 2.5), English Bulldog (OR, 2.4), Labrador Retriever (OR, 2.3), Italian Mastiff (OR, 2.1), and German Shepherd, Golden Retriever, and West Highland White Terrier, all with OR of 1.8. A moderate risk was observed for 3 breeds: Boxer (OR, 4.6), American Staffordshire Terrier (OR, 4.2), and Newfoundland (OR, 3.6). Finally, a marked risk was observed for 4 breeds: Weimaraner (OR, 9.4), French Bulldog (OR, 8.2), Standard Schnauzer (OR, 7.1), and Australian Shepherd (OR, 5.6). Breed distribution and predispositions for the most commonly observed defects are presented in Tables 2–7. Sex distribution was similar to that of the overall population with males being slightly more frequent (54%) than females (46%). Specific male predisposition was observed for PS (OR, 1.5), SAS (OR, 1.7), and AS (OR, 2.6) and female predisposition for PDA (OR, 2.7). Mean age at presentation was approximately 42 months, ranging from 1 to 187 months.

Lesion classification information was available in 333 cases of PS. Type A was most common (n = 235, 70.6%), followed by type B (n = 84, 25.2%), mixed (n = 13, 3.9%), and supravalvular stenosis (n = 1, 0.3%).[9–11] In the case of SAS, lesion classification according to Pyle & Patterson[8,12,13] was available in 134 cases, with type 2 being the most common (n = 69, 51%), followed by type 3 (n = 41, 31%), and type 1 (n = 24, 18%).

Reverse PDA was observed in 6 of the 237 dogs with this defect. Diagnosis was made with the aid of agitated saline echocardiographic contrast in 5 cases and angiography in 1 case.

Multiple Heart Defects

The various combinations of congenital heart defects observed are presented in Table 8. The most commonly observed were PS and SAS (n = 38, 26.4%), followed by PS and VSD (n = 24, 16.7%). PDA was associated with a concurrent defect in 9.3% (n = 22) of the cases, mostly PS (n = 10, 45%) and SAS (n = 7, 32%). VSD was associated to another defect in 48% of the cases, mostly PS (65%).

Discussion

PS, SAS, PDA, and VSD consistently have been reported as being the most commonly encountered cardiac defects in previous studies.[1,4,5,14] In the present study, PS was the most common cardiac defect, accounting for 32.1% in contrast to 18–23% reported in other studies.[1,4,5,14] This difference may be because of the fact that our center receives many referrals for pulmonary balloon valvuloplasty. The high percentage of Boxers in our study (26%), and corresponding predisposition for PS (OR, 5.7), also may account for this fact. Boxer predilection for PS has already been reported in the literature,[13,15] as well as a male predisposition in this breed.[15] In the present study, male predisposition was observed in the overall breed population (OR, 1.5). Additionally, other breed predilections were identified, some of which had already been reported previously, as is the case of the English Bulldog, West Highland White Terrier, and Chihuahua.[1] Lack of predisposition of Golden Retriever, Labrador Retriever, and Yorkshire Terrier also is in accordance with previously published results.[1] According to our results, type A is the most common form observed, representing 70.6% of the cases in this study, as compared with 25.2% with type B. Mixed lesions (type A and B) seem to be uncommon

Table 1. Congenital heart defects.

Heart Defect	N	Isolated	Associated	Male	Female	Age (Months)[a]
Pulmonic stenosis	363 (32.1%)	271 (75%)	92 (25%)	216 (60%)[b]	145 (40%)[b]	40 (1–161)
Subaortic stenosis	241 (21.3%)	195 (81%)	46 (19%)	150 (62%)	91 (38%)	52 (1–187)
Patent ductus arteriosus	237 (20.9%)	215 (90.7%)	22 (9.3%)	83 (35%)	153 (65%)	38 (2–155)
Ventricular septal defect	85 (7.5%)	45 (52%)	40 (48%)	39 (46%)	46 (54%)	40 (3–157)
Aortic stenosis	64 (5.7%)	52 (81%)	12 (19%)	45 (70%)	19 (30%)	78 (4–158)
Tricuspid dysplasia	35 (3.1%)	26 (74%)	9 (26%)	17 (48.5%)	18 (51.5%)	41 (2–124)
Mitral dysplasia	21 (1.9%)	13 (62%)	9 (38%)	10 (48%)	11 (52%)	21 (4–94)
Double chamber right ventricle	14 (1.2%)	9 (64%)	5 (36%)	6 (43%)	8 (57%)	24 (4–69)
Atrial septal defect	12 (1.1%)	8 (67%)	4 (33%)	3 (27%)	8 (73%)	47 (10–128)
Tetralogy of Fallot	11 (1.0%)	—	—	6 (55%)	5 (45%)	14 (1–33)
Aortic hypoplasia	9 (0.8%)	0 (0%)	9 (100%)	5 (56%)	4 (44%)	51 (29–103)
Anomalous right coronary artery	9 (0.8%)	2 (22%)	7 (78%)	7 (78%)	2 (22%)	38 (17–66)
Persistent left cranial vena cava	9 (0.8%)	0 (0%)	9 (100%)	6 (67%)	3 (33%)	18 (44–110)
Cor triatriatum dexter	3 (0.3%)	3 (100%)	0 (0%)	1 (33%)	2 (67%)	37 (10–60)
4th right aortic arch	3 (0.3%)	2 (67%)	1 (33%)	2 (67%)	1 (33%)	10 (2–19)
Peritoneal-pericardial diaphragmatic hernia	3 (0.3%)	2 (67%)	1 (33%)	0 (0%)	3 (100%)	49 (7–84)
Patent foramen ovale	2 (0.2%)	0 (0%)	2 (100%)	0 (0%)	2 (100%)	[c]
Situs inversus	2 (0.2%)	0 (0%)	2 (100%)	0 (0%)	2 (100%)	62 (28–95)
Supravalvular aortic stenosis	1 (0.1%)	1 (100%)	0 (0%)	0 (0%)	1 (100%)	3,6
Anomalous pulmonary venous return	1 (0.1%)	1 (100%)	0 (0%)	1 (100%)	0 (0%)	42
Truncus arteriosus	1 (0.1%)	1 (100%)	0 (0%)	0 (0%)	1 (100%)	[c]
Aortic-pulmonary window	1 (0.1%)	1 (100%)	0 (0%)	1 (100%)	0 (0%)	45
Bicuspid aorta	1 (0.1%)	0 (0%)	1 (100%)	1 (100%)	0 (0%)	65
Quadricuspid aorta	1 (0.1%)	0 (0%)	1 (100%)	0 (0%)	1 (100%)	49
Anomalous subclavian artery	1 (0.1%)	0 (0%)	1 (100%)	0 (0%)	1 (100%)	113
Two stenotic pulmonary arteries	1 (0.1%)	0 (0%)	1 (100%)	0 (0%)	1 (100%)	24
Pericardial cyst	1 (0.1%)	0 (0%)	1 (100%)	1 (100%)	0 (0%)	31
Total	1132 (100%)					42 (1–187)

[a]Age at presentation.
[b]Information relative to sex was unavailable in 2 cases.
[c]Unavailable.

Table 2. Breed distribution and predisposition—pulmonic stenosis.

	Pulmonic Stenosis (363 cases)			
	N	%	Odds Ratio	*P*
Boxer	116	31.9	5.27	< .0001
Mongrel	35	9.6	0.32	< .0001
English Bulldog	27	7.4	3.16	< .0001
French Bulldog	21	5.8	19.1	< .0001
Pinscher	14	3.8	3.1	.0001
German Shepherd	11	3.0	0.44	.0085
Beagle	10	2.7	2.66	.003
West Highland White Terrier	9	2.5	2.91	.003
American Staffordshire Terrier	8	2.2	16.9	< .0001
Chihuahua	8	2.2	3.11	.003
Cavalier King Charles Spaniel	6	1.6	1.62	NS
Cocker Spaniel	6	1.6	1.1	NS
Pitbull Terrier	6	1.6	4.48	.0009
Rottweiler	6	1.6	0.91	NS
Newfoundland	5	1.4	0.81	NS
Golden Retriever	5	1.4	0.97	NS
Shih-Tzu	5	1.4	0.78	NS
Yorkshire Terrier	5	1.4	0.23	.0013
Italian Mastiff	4	1.1	2.1	NS
Poodle	4	1.1	0.23	.0062
Standard Schnautzer	4	1.1	16.7	< .0001
Others	48	13.2		

(3.9%) and supravalvular stenosis is a rare occurrence (0.3%). The presence of an associated anomalous right coronary artery was suspected in 8 cases by TTE, and confirmed by angiography in 4 cases and by TEE in 1 case. Angiographic classification was available in only 3 cases, all presenting with type R2A anomaly.[16] Half of these dogs were English Bulldogs, a fact that is in agreement with previous reports of predisposition for this breed.[17,18]

The 2nd most commonly observed CHD in this study was SAS (21.3%), similar to what has been reported

Table 3. Breed distribution and predisposition—subaortic stenosis.

	Subaortic Stenosis (241 Cases)			
Breed	N	%	Odds Ratio	*P*
Boxer	121	49.8	9.4	< .0001
German Shepherd	24	9.9	1.8	.0166
Dogue de Bordeaux	18	7.4	11.2	< .0001
Newfoundland	23	9.5	7.0	< .0001
Rottweiler	16	6.6	4.5	< .0001
Golden Retriever	13	5.3	3.6	.0001
Mongrel	5	2.1	0.1	< .0001
Labrador Retriever	4	1.6	0.6	NS
Others	18	7.4		

Table 4. Breed distribution and predisposition—valvular aortic stenosis.

	Aortic Stenosis (64 Cases)			
Breed	N	%	Odds Ratio	*P*
Boxer	40	62.5	17.9	< .0001
Bull Terrier	5	7.8	41	< .0001
German Shepherd	5	7.8	1.14	NS
Others	14	21.9		

previously in the United States,[1,14] but in contrast to other European studies in which it was the most common cardiac defect, accounting for 31.5[5] and 35%[4] of all cardiac defects. In this study, subvalvular and valvular lesions were considered separately, with aortic valvular stenosis accounting for an additional 5.7% of the cases, and comparison between study results should take this fact into account. Nevertheless, combined SAS and AS still account for the 2nd most common congenital pathology with an incidence of 27%. Males seem to be predisposed (OR, 1.7) in contrast to previous reports in which sex predilection had not been confirmed,[1] except in the Boxer breed.[1,15] This breed represented, however, almost half (49.8%) of the cases of SAS in the present study and influenced the results. Data were reanalyzed excluding this breed and, although males were still more prevalent than females (63 versus 37%), a statistically significant higher relative risk for males was not confirmed (OR, 1.1; $P = .7$). Breed predilections observed for SAS, except for Dogue de Bordeaux (OR, 11.2), had already been described and are in agreement with a previous study.[1]

Regarding PDA, its prevalence in the United States seems to be higher[1,14] than in Europe.[4,5] In the present study, it was the 3rd most common defect (20.9%), slightly more prevalent than in previous European reports (4th most common, 11–13.7%)[4,5] and less common than in USA reports (most common, 27.7–32%).[1,14] Similar to PS, many PDA cases received by our center are referred for surgical ligation or transcatheter embolization, therefore increasing its apparent incidence in comparison with what would be expected in a first opinion practice center. For the same reason, the observed incidence of reverse PDA probably is an underestimate of the real incidence, because this condition is not suitable for occlusion. Females are consistently reported as being more often affected than males,[1,4,19–21] and the results of our study support this predilection, indicating a female predisposition for PDA (OR, 2.7). Considering breed incidence and predilections, the results of this study are slightly different from those of other reports. The German Shepherd was the most commonly affected breed and a markedly higher risk was observed (OR, 5.2), in contrast to previously published results in which a higher risk was not identified (nonsignificant odds ratio of 1.2), although this breed was among the most common breeds with PDA.[1] Furthermore, Poodle, Pomeranian, and Yorkshire Terrier predisposition reported by the same study[1] was not confirmed in the present study. This might be because of differences in breed popularity and incidence between the 2 geographic areas. The same applies to Chihuahua and Australian Shepherd for which a statistically significant higher risk was observed in this study in contrast to the other study.[1] In both studies, the Maltese breed was found to be at a markedly higher risk for PDA.

Table 5. Breed distribution and predisposition—patent ductus arteriosus (PDA).

	PDA (237 Cases)			
Breed	N	%	Odds Ratio	*P*
German Shepherd	58	24.5	5.2	< .0001
Mongrel	41	17.3	0.65	.018
Newfoundland	13	5.5	4.65	< .0001
Maltese	12	5.1	4.14	< .0001
Dobermann	12	5.1	2.8	.0007
Poodle	12	5.1	1.18	NS
Yorkshire Terrier	9	3.8	0.71	NS
Cavalier King Charles Spaniel	9	3.8	3.7	.0006
Dachshund	6	2.5	0.42	.0372
Chihuahua	6	2.5	3.66	.0028
West Highland White Terrier	4	1.7	1.93	NS
Pomeranian	5	2.1	2.5	NS
Irish Setter	5	2.1	0.94	NS
Belgian Shepherd	4	1.7	4.38	.0059
Australian Shepherd	4	1.7	27.3	< .0001
Others	37	15.6		

Table 6. Breed distribution and predisposition—ventricular septal defect (VSD).

	VSD (85 Cases)			
Breed	N	%	Odds Ratio	*P*
Mongrel	13	15.3	0.6	NS
Pinscher	5	5.9	39	< .0001
French Bulldog	4	4.7	7.2	.0003
German Shepherd	4	4.7	3.7	.001
Labrador Retriever	4	4.7	0.8	NS
Others	55	64.7		

VSD was the 4th most common cardiac defect in our study, with a lower incidence (7.5%) than reported previously (9.8–14.4%).[1,4,5] Interestingly, VSD was observed in conjunction with another cardiac defect in almost half of the cases (48%) and usually with PS (65%). This association was observed in 23 dogs, of which 19 were presented with a type A PS and a perimembranous VSD (n = 9) or a muscular VSD (n = 8).

Table 7. Breed distribution and predisposition—tricuspid dysplasia.

	Tricuspid Dysplasia (35 Cases)			
Breed	N	%	Odds Ratio	*P*
Labrador Retriever	9	25.7	11.13	< .0001
Boxer	5	14.3	1.96	NS
German Shepherd	5	14.3	2.24	NS
English Bulldog	3	8.6	6	.0035
Golden Retriever	3	8.6	6.6	.0022
Others	10	28.5		

Table 8. Multiple heart defects.

Associated Pathologies	N
PS + SAS	34
PS + VSD	19
PS + PDA	8
PDA + SAS	6
SAS + Mitral dysplasia	5
SAS + Perimembranous VSD	4
PS + Anomalous right coronary artery	4
SAS + Aortic stenosis	3
SAS + Aortic root hypoplasia	3
PS + 2 Muscular VSD	3
PS + AS	3
PS + Persistent left cranial vena cava	3
PS + Tricuspid dysplasia	3
AS + Aortic root hypoplasia	2
SAS + Situs inversus	2
VSD + Double chambered right ventricle	2
VSD + Mitral dysplasia	2
PS + AS + Aortic hypoplasia	2
PS + Persistent left cranial vena cava + Anomalous right coronary artery	2
AS + Bicuspid aorta	1
PDA + 4th right aortic arch	1
PDA + Anomalous Subclavian	1
PDA + AS	1
PDA + Mitral dysplasia	1
PDA + Tricuspid dysplasia	1
PDA + VSD	1
SAS + Muscular VSD	1
SAS + Persistent left cranial vena cava	1
SAS + Persistent left cranial vena cava + VSD	1
SAS + Persistent left cranial vena cava + mitral stenosis	1
SAS + Bicuspid aorta	1
SAS + Quadricuspid aorta	1
SAS + Tricuspid dysplasia	1
SAS + Double chambered right ventricle + Mitral stenosis	1
SAS + Aortic root hypoplasia + Supravalvular stenosis	1
Tricuspid dysplasia + Patent foramen ovale	1
Tricuspid dysplasia + ASD	1
Tricuspid dysplasia + Cor triatriatum dexter	1
Tricuspida dysplasia + Double chambered right ventricle	1
VSD + ASD	1
VSD + ASD like Sinus Venosus Coronaricus	1
VSD + Persistent left cranial vena cava	1
PS with 2 stenotic pulmonary arteries	1
PS + ASD	1
PS + Double chambered right ventricle	1
PS + PDA + Peritoneal-pericardial-diaphragmatic hernia	1
PS + Pericardial cyst	1
PS + Patent foramen ovale	1
PS + SAS + PDA	1
PS + SAS + Anomalous right coronary artery	1
PS + SAS + Aortic hypoplasia	1
PS + SAS + VSD	1
PS + VSD + Anomalous coronary trunk	1

AS, valvular aortic stenosis; ASD, atrial septal defect; PDA, patent ductus arteriosus; PS, pulmonic stenosis; SAS, subaortic stenosis; VSD, ventricular septal defect.

Only 4 cases were presented with a type B PS, 3 with a perimembranous VSD and 1 with a muscular VSD. In humans, VSD may coexist with nearly all varieties of CHD[22] and often is associated with ASD, PDA, PS, and right aortic arch.[23] Additionally, a possible association between PS and VSD has been suggested because their incidence was found to be more common than would be expected by chance.[24] A possible relationship between these defects and TOF also has been proposed but not proven.[24] If the pulmonary valve gradient is high enough, right ventricular hypertrophy, another characteristic of TOF, may occur. This was observed in most of the cases in the present study, however, without aortic overriding, and therefore not true TOF. Evidence of conotruncal development abnormality must be present in the case of TOF. No differences were identified in breed or sex incidence between these subpopulations and the overall population of dogs with PS. Pinscher, French Bulldog, and German Shepherd seem to be at higher risk. Other breed predilections have been described elsewhere.[1]

The incidence of tricuspid dysplasia (3.1%) also was slightly lower than reported previously (5.1–7.5%).[1,4,5] With the exception of English Bulldog, the predilections observed in the case of Labrador Retriever and Golden Retriever had already been previously reported and are in agreement.[1] A previously observed higher female incidence was not confirmed in the present study.[4]

ASD represented 1.1% of cardiac defects, a result in agreement with most previously published reports[1,4,25] but much lower than in 1 other study.[26] Additionally, no breed predilection was observed in contrast to other previous reports in which Boxers were at higher risk.[1,26] The diagnosis of ASD was achieved by echocardiographic examination, as in the case of the studies mentioned above.[4,26] In the past, it has been shown that the risk of overlooking cardiac defects with echocardiography was greater than the risk of overdiagnosing them.[27] This report, however, relied on the use of 2-dimensional echocardiographic imaging, and since it has been published, substantial advances have been made in echocardiographic imaging that in combination with Doppler technology have rendered this diagnostic method much more sensible and reliable.[26] Additionally, all echocardiographic examinations in this study were performed by experienced operators with a combination of 2D and Doppler techniques, and therefore, in our opinion, ASD were not underdiagnosed. TOF and persistent right aortic arch were less frequent in the present study than reported previously.[1]

In previous reports, the incidence of multiple heart defects was 7–8%,[4,5] approximately half that of the present study (15%). From the analysis of the results of this and other studies, the association between PS and SAS seems to be one of the most common associations between heart defects in dogs.[4,15] The frequent association between VSD and other defects, and the presence of concurrent PS and PDA in some dogs, emphasizes the importance of performing a complete echocardiographic examination in all dogs, even when an anomaly possibly explaining the clinical findings has already been identified.

Considering the breed predispositions observed in this study, we believe that it is prudent to advise systematic screening for congenital cardiac defects before breeding,

in order to decrease their prevalence, especially in breeds demonstrating a moderate or increased risk for these defects. Such is the case in the Boxer, Newfoundland, French Bulldog, English Bulldog, German Shepherd, Golden Retriever, and Labrador Retriever, all of which demonstrated an increased risk for cardiac defects in general, and for some defects in particular. Although other breeds appear to be at increased risk according to the results of this study, we believe that because of their lower incidence in the general population ($<1\%$), caution must be taken when interpreting the results and further studies are necessary. Nevertheless, owners and breeders of these breeds should be appropriately informed of this fact, and clinicians should have a high index of suspicion when examining individuals of these breeds, and promptly inform breeders of the existence of cardiac defects.

From an analysis of Table 1, it can be seen that the average age at the time of diagnosis was >2 years for most defects. These results are influenced by late diagnosis in some mild cases. Nevertheless, ideally we would expect these defects to be identified at a much earlier age in order to be able to optimize therapeutic management and maximize life expectancy and quality of life. A greater awareness of clinicians, owners, and breeders to the existence of these defects is necessary in order to improve medical care for these patients.

In conclusion, this study allowed us to characterize the incidence of CHD in a large population of dogs in Italy, and only minor differences were observed in comparison with previous studies from other geographical areas.[1,4,5,14] The retrospective nature, as well as the fact that the data derives from a cardiology referral center, constitute limitations to the design of our study. Particularly, defects amenable to surgical or percutaneous repair, such as PS, SAS, and PDA, may be overrepresented in this study. Breed and sex predilections were identified for some defects, confirming or adding to previously observed tendencies, and may be of value for the diagnosis and screening of CHD in dogs. Finally, concurrent heart defects represented a substantial percentage of cases, and their identification depends upon accurate and complete examinations.

Footnotes

[a] ESAOTE S.p.A., Firenze, Italy
[b] Villa Sistemi Medicali S.p.A., Buccinasco (MI), Italy
[c] MedCalc 10.2.0.0, Mariakerke, Belgium
[d] Microsoft Office Excel 2007, Microsoft Corp, Redmond, WA

References

1. Buchanan JW. Prevalence of cardiovascular disorders. In: Fox PR, Sisson DD, Moise NS, eds. Textbook of Canine and Feline Cardiology, 2nd ed. Philadelphia, PA: WB Saunders Company; 1999:458–463.

2. Hoffman JI, Kaplan S. The incidence of congenital heart disease. J Am Coll Cardiol 2002;39:1890–1900.

3. Hunt GB, Church DB, Malik R, et al. A retrospective analysis of congenital cardiac anomalies (1977–1989). Aust Vet Pract 1990:70–75.

4. Tidholm A. Retrospective study of congenital heart defects in 151 dogs. J Small Anim Pract 1997;38:94–98.

5. Baumgartner C, Glaus TM. Congenital cardiac diseases in dogs: A retrospective analysis. Schweiz Arch Tierheilkd 2003;145: 527–533, 535–526.

6. MacDonald KA. Congenital heart diseases of puppies and kittens. Vet Clin North Am Small Anim Pract 2006;36:503–531, vi.

7. Thomas WP, Gaber CE, Jacobs GJ, et al. Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. Echocardiography Committee of the Specialty of Cardiology, American College of Veterinary Internal Medicine. J Vet Intern Med 1993;7:247–252.

8. Bussadori C, Amberger C, Le Bobinnec G, et al. Guidelines for the echocardiographic studies of suspected subaortic and pulmonic stenosis. J Vet Cardiol 2000;2:15–22.

9. Bussadori C, DeMadron E, Santilli RA, et al. Balloon valvuloplasty in 30 dogs with pulmonic stenosis: Effect of valve morphology and annular size on initial and 1-year outcome. J Vet Intern Med 2001;15:553–558.

10. Patterson PD. Two hereditary forms of ventricular obstruction in the dog: Pulmonary valve dysplasia and discrete subaortic stenosis. In: Nora J, Takao A, eds. Congenital Heart Disease—Causes and Processes. Mount Kisco: Futura Publishing Co; 1984:43–63.

11. Patterson DF, Haskins ME, Schnarr WR. Hereditary dysplasia of the pulmonary valve in Beagle dogs. Pathologic and genetic studies. Am J Cardiol 1981;47:631–641.

12. Pyle RL, Patterson DF, Chacko S. The genetics and pathology of discrete subaortic stenosis in the Newfoundland dog. Am Heart J 1976;92:324–334.

13. Bussadori C, Quintavalla C, Capelli A. Prevalence of congenital heart disease in Boxers in Italy. J Vet Cardiol 2001;3: 7–11.

14. Buchanan JW. Causes and prevalence of cardiovascular disease. In: Kirk RW, Bonagura JD, eds. Current Veterinary Therapy XI: Small Animal Practice. Philadelphia, PA: WB Saunders; 1992:647–655.

15. Bussadori C, Pradelli D, Borgarelli M, et al. Congenital heart disease in Boxer dogs: Results of 6 years of breed screening. Vet J 2009;181:187–192.

16. Buchanan JW. Pathogenesis of single right coronary artery and pulmonic stenosis in English Bulldogs. J Vet Intern Med 2001;15:101–104.

17. Buchanan JW. Pulmonic stenosis caused by single coronary artery in dogs: Four cases (1965–1984). J Am Vet Med Assoc 1990;196:115–120.

18. Fonfara S, Martinez Pereira Y, Swift S, et al. Balloon valvuloplasty for treatment of pulmonic stenosis in English Bulldogs with an aberrant coronary artery. J Vet Intern Med 24:354–359.

19. Van Israel N, Dukes-McEwan J, French AT. Long-term follow-up of dogs with patent ductus arteriosus. J Small Anim Pract 2003;44:480–490.

20. Campbell FE, Thomas WP, Miller SJ, et al. Immediate and late outcomes of transarterial coil occlusion of patent ductus arteriosus in dogs. J Vet Intern Med 2006;20:83–96.

21. Kittleson MD. Patent ductus arteriosus. In: Kittleson MD, Kienle RD, eds. Small Animal Cardiovascular Medicine. St Louis, MO: Mosby; 1998:218–230.

22. Corno AE, Festa P. Ventricular septal defect. In: Corno AF, ed. Congenital Heart Defects. Germany: Steinkopff Verlag; 2009:49–55.

23. Popelová J, Oechslin E, Karemmerer H, et al. Ventricular septal defect. In: Popelova J, Oechslin E, Karemmemer H, Sutton M, eds. Congenital Heart Disease in Adults. London: Informa UK Ltd; 2008:32–39.

24. Brotmacher L, Campbell M. Ventricular septal defect with pulmonary stenosis. Br Heart J 1958;20:379–388.

25. Mulvihill JJ, Priester WA. Congenital heart disease in dogs: Epidemiologic similarities to man. Teratology 1973;7:73–77.

26. Chetboul V, Charles V, Nicolle A, et al. Retrospective study of 156 atrial septal defects in dogs and cats (2001–2005). J Vet Med A Physiol Pathol Clin Med 2006;53:179–184.

27. Gutgesell HP, Huhta JC, Latson LA, et al. Accuracy of two-dimensional echocardiography in the diagnosis of congenital heart disease. Am J Cardiol 1985;55:514–518.

RADIATION HAZARDS TO PATIENTS IN DIAGNOSTIC RADIOLOGY: CONSIDERATIONS ON COMPUTED TOMOGRAPHY IN DOGS AND CATS

Juergen Kiefer[3], Ingmar Kiefer[1], Eberhard Ludewig[1], Dieter Gosch[2], Gerhard Oechtering[1]. [1]Department of Small Animal Medicine, Faculty of Veterinary Medicine, University of Leipzig, Germany, An den Tierkliniken 23, 04103 Leipzig, [2]Klinik und Poliklinik für Diagnostische und Interventionelle Radiologie, Universitätsklinikum Leipzig, Germany and [3]Justus-Liebig-University Giessen, Am Dornacker 4, Wettenberg, Germany.

Introduction
Radiation protection of patients has recently gained considerable attention in human medicine. In veterinary medicine it is often tacitly assumed that the shorter lifespan of small animals is not sufficient for the development of clinical symptoms. One has to take into account, however, that the increasing use of computed tomography may lead to comparatively high exposures of critical organs. We show here by phantom computations that with protocols which are now typical standard in dogs doses to the eye may reach values which are so high that the risk of cataract formation cannot be excluded.
Methods
Three different helical CT-scans of the head, the nose and the whole respiratory tract of dogs were taken for the assessment of head malformations and for the planning of LATE-operations. The investigations were performed under anaesthesia (inhalation anaesthesia with apnoe-phase, if necessary) using a Philips Mx8000 IDT/Brilliance 6. The parameters were : head: 120 KV, 150 mAs/slice, Pitch 0,6, Collimation 6*0,75 mm, Slice thickness 1,00 mm; nose: 140 KV, 214 mAs/slice, Pitch 0,6, Collimation 2*0,6 mm, Slice Thickness 0,6 mm; respiratory tract: 120 KV, 250 mAs/slice, Pitch 0,9, Collimation 6*1,5 mm, Slice Thickness 2.00 mm. Organ doses were computed with the aid of the programme CT-Expo V1.6 which is based on conversions factors for different human phantoms (“Adam”, “Eva”, “child” and “baby”) with no special adjustments for dogs.
Results
The skeleton, the eye lens, the brain and the thyroid were found to be the most exposed organs. The computed doses depended on the phantoms used with “baby” giving the highest values. Considering the size of typical patients in small animal practice the use of this programme appears to be the most realistic approach. The total doses after the complete investigation with three scans were: skeleton 305 mGy, lens 218 mGy, brain 188 mGy and thyroid 80 mGy. Although these values cannot be generalised they may be considered as typical for a clinical situation. They are not low as far as a possible radiation hazard is taken into account.
Discussion
It is clear from these model computations that radiation hazards to patients in small animal veterinary cannot be neglected. The formation of eye cataracts cannot be excluded. There are only few data with dogs but recent investigations in human indicate that threshold doses (if they exist at all) are well below 500 mGy. They may be exceeded in the case of multiple examinations. It should, therefore, be considered if the scan geometry should be modified in such a way (e. g. by tilting the gantry) that the eye lens is not directly exposed to the primary beam. Dose reduction may also be achieved by changing the scan parameters but this leads to a deterioration of the image quality and thus also to a loss of diagnostic precision.

ROLE OF MULTI-MODALITY IMAGING TO GUIDE PROCEDURES OF TRANSVENOUS COIL EMBOLISATION FOR THE TREATMENT OF CANINE INTRAHEPATIC PORTO-SYSTEMIC SHUNT

Rossi F[1], Domenech O* , Bussadori R*, Bussadori C*. [1]Clinica Veterinaria dell’Orologio, Via Gramsci 1/4 - 40037 Sasso Marconi (Bologna) – Italy and [2]Clinica Veterinaria Gran Sasso – Via Donatello 26 - 20124 Milano – Italy.

Introduction
Transvenous coil embolisation is a recently described interventional radiology technique for the treatment of intra- and extrahepatic porto-systemic shunts (PSS) in dogs. Under fluoroscopic guidance, a stent and a variable number of coils are placed in the caudal vena cava and in the PSS with the aim of progressively embolise the anomalous vessel without development of portal hypertension. The aim of this study was to combine information obtained from helical Computed Tomography (CT), trans-esophageal endoscopic ultrasound (EU) and fluoroscopy (F) imaging to better understand the exact anatomy of the vascular anomaly and to guide the operator during the procedure. Advantages and disadvantages of the different imaging modality were highlighted.
Methods
Three dogs (one 5 months old female Labrador, one 6 months old male mixed breed and one 1 year old male Dachshund) affected by left intra-hepatic PSS underwent trasnvenous coil embolisation. Diagnosis was made by trans-abdominal ultrasonography and confirmed by helical dual phase CT scan of the abdomen using a single-slice unit (GE Pro-Speed). Transverse and reconstructed CT images were used to measure vessels diameters and select the proper size of the stents and coils. During the interventional procedure, CT images were available and oriented to match with the fluoroscopic images. Moreover, trans-esophageal EU (Esaote, My Lab 30Vet) and F (Villa Genius) were used to visualize the vessels and to follow the position of the catheters and devices in real time. Measurements of the vessels were repeated by ultrasound and compared with the CT results. The exact position of the catheters was confirmed by EU and F, injecting shaked saline solution or iodinated contrast medium to fill the PSS, respectively.
Results
CT images gave the best global representation of the vascular anatomy of the abdomen and were extremely useful to help the operator to guide the catheters in the correct position and angulation to enter the PSS during F. Matching the CT transversal images with the CT scout images enabled to correlate CT and F during the procedure. Trans-esophageal EU was able to show the caudal vena cava and the stent in this vessel, the portal vein, the origin of the PSS and the coils in all three cases. The tortuous PSS could be followed cranially however it was difficult to clearly visualize its entering point in the caudal vena cava, because of artefacts due to the caudal lung during respiration. Injection of saline was useful to confirm the exact position of the catheters before placing the coils. Combining CT and EU information, the use of F and iodinated contrast medium was reduced, with an advantage for the patient and the operators. There was a total agreement between CT and EU measurements.
Discussion/consclusions
Multi-modality imaging combining CT, EU and F is useful during procedures of transvenous coils embolisation by adding anatomical information and helping the quick correct placement of the stent and the coils.

A COMPARISON OF CT AND MRI FOR THE IDENTIFICATION OF INTRACRANIAL LESIONS IN DOGS AND CATS

Kromhout K[1], Gavin P[2], Van Ham L[3], van Bree H[1], Gielen I[1]. [1]Department of Medical Imaging & Small Animal Orthopaedics, Veterinary Faculty, Ghent University, Salisburylaan 133, 9820 Merelbeke, Belgium, [2]Radiology, Washington State University, Pullman, Washington, USA and [3]Department of Small Animal Medicine, Veterinary Faculty, Ghent University, Salisburylaan 133, 9820 Merelbeke, Belgium.

Introduction
Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) are the techniques commonly used for evaluating intracranial diseases. The choice between these techniques is mostly based on availability, economics and the neurological anamnesis. Previous studies have proved that MRI has a more superior soft tissue contrast resolution as CT is more sensitive for bone abnormalities. Lesions in the brainstem and cerebellum are frequently missed on CT due to beam hardening. The purpose of this study is to compare characteristics associated with lesions on MR and CT images of the brain.
Methods
Over a period of 2 years, 61 patients underwent both MRI and CT studies. MR images were obtained on a 0, 2 Tesla unit (Airis Mate, Hitachi Medical Corporation, Japan). The animals were positioned in dorsal recumbency under general anaesthesia with their head placed into a human head or wrist coil and submitted to transverse and sagittal slices in T1 and T2 spin-echo sequences. Additional dorsal FLAIR and transverse post-gandolinium T1-weighted sequences were applied. CT transverse pre- and postcontrast images were obtained with a 3rd generation helical CT scanner (GE ProSpeed, General Electric Co., Milwaukee, WI). The CT and MR images were blinded and evaluated independently. Evaluated parameters included: presence of lesions, occurrence, lesion localisation, pre- and post contrast intensity, size of the lesions, regularity/pattern, shape, margins, mass effect, peri-tumoural oedema, presence of hydrocephalus, calcification, cystic lesions, haemorrhage, herniation, and eventual involvement of calvarium.
Results
By use of CT in 34 and by use of MRI in 37 out of the 61 patients, lesions were detected. On CT in 32 cases and on MRI in 24 cases a mass effect was seen. Concerning the localisation of the lesions, on CT 22 were localised in the cerebrum, 7 in the cerebellum and 5 in the brain stem. On MRI 19 were localised in the cerebrum, 7 in the cerebellum and 8 in the brain stem. On CT 2 lesions were localised within the ventricles whereas on MRI 7 were found within the ventricles. On CT 30 solitary and 4 multiple lesions were detected and on MRI 29 solitary and 8 multiple lesions were found. On CT 16 lesions and on MRI 25 lesions were localised intra-axial.
Conclusions
By the use of CT most lesions could be detected especially when producing a mass effect. With MRI more lesions within the brain stem and within the ventricles could be seen and more multiple lesions were detected. The difference in identification of intra-cranial lesions between CT and MRI was not very spectacular. The advantages of CT are the lower cost of the equipment and the shorter duration of the examination time.

DIAGNOSTIC VALUE OF CT AND MRI IN DIAGNOSIS OF CORONOID PATHOLOGIES IN THE DOG

Stephan Klumpp, Kramer Martin. Klinikum Veterniärmedizin der Justus-Liebig-Universität Giessen. Klinik für Kleintiere, Radiologie, Frankfurter Str. 108, 35392 Giessen, Germany.

Introduction and methods
In the study we compared the results of different diagnostic imaging modalities in dogs, suffering from medial coronoid process pathology. In clinically and radiographically suspicious dogs for cornoid process pathology computertomograpy (CT) and magnetic reconance imaging (MRI) was performed. The lack of superimpositioning with both techniques allows a good evaluation of the medial coronoid process (pcm) for the following criterias:a) fissure at the level of the pcm, b) fragments at the level of the pcm, c) deformation at the level of the pcm, d) increased opacity at the level of the base of the pcm., e) heterogenous opacity at the apex of the pcm.
Results
Altogether 44 elbow joints of 12 different breeds were examined. Labrador Retriever (38,6%), mixed breed (22,7%) and Golden Retrievers were the three most common breeds. 68% ($n = 30$) were male and 32% ($n = 14$) were female. The age of the dogs ranged from 6 to 117 month. The body weigth was between 19 and 57 kg.
Within the following points CT and MRI were compared: a) fissure at the level of the pcm, b) fragments at the level of the pcm, c) deformation at the level of the pcm. Within these three points no significant difference between CT and MRI was seen., d) increased opacity at the level of the base of the pcm, e) heterogenous opacity at the apex of the pcm.
Discussion
Therefore both diagnostic imaging modalities are appropriate for evaluating a coronoid process pathology in the dog. The two points: d) increased opacity at the level of the base of the pcm, e) heterogenous opacity at the apex of the pcm are only evaluable on CT images. From our point of view at that time these two points can be of crucially importance for diagnosing a coronoid pathology in the dog. Further studies are necessary to evaluate the anatomically normal CT picture to secure these findings and their importance for the diagnosis of coronoid pathology in the dog. The shorter examination time in CT compared to MRI and the ability to evaluate both elbow joints with one examination are great advantages of CT. Therefore at this time CT is prior to MRI in clinical patients with suspicion of coronoid process pathology.