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Ventricular septal defects in cats: A retrospective study

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Summary

Ventricular septal defect (VSD) is one of the most common congenital heart defects in cats. It usually occurs as a single defect, but in rare cases it may be part of a complex defect. Echocardiography is a method that affords a reliable and non-invasive diagnosis of VSD. The objective of this study was to determine the incidence and type of ventricular septal defects (VSD) in a feline population over six years of follow-up. The study analyzed VSD incidence in 31 cats from the Department of Epizootiology and Clinic of Infectious Diseases, University of Life Sciences, Lublin, between January 2018 and December 2023. VSDs were classified into four groups by Soto. All 31 cats (13 males, 18 females), aged 2 months to 4 years, had normal basic biochemical panels. Clinical examinations detected heart murmurs in all cats, with 14 showing signs. Echocardiography assessed cardiac function and VSD flow. Statistical analysis examined correlations between the type of VSD, clinical signs, breed and sex, without age correlation. The study found no significant breed predisposition for VSD, although mixed-breed cats were diagnosed most frequently. Nor was there any significant correlation between the type of VSD and the cats' sex. The severity of clinical signs correlated with the location and size of VSD. The findings demonstrate the importance of routine echocardiographic screening to detect VSDs early, potentially improving therapeutic outcomes and the quality of life of affected cats.

Keywords: cats, echocardiography, ventricular septal defect

The mammalian heart comprises four cavities: two atria separated by the atrial septum and two ventricles separated by the interventricular septum. Under physiological conditions, these two septa remain continuous along their entire course. However, developmental defects may sometimes emerge which compromise the integrity of the septa (4). One of these is the ventricular septal defect (VSD), which is found more frequently in cats than in dogs (5). There are several types of VSD (12). A VSD may be an isolated defect or may co-exist with other defects, such as a patent ductus arteriosus or atrial septal defect. It is also one of the causes of tetralogy and pentalogy of Fallot and Eisenmenger syndrome (3, 8). VSD is a congenital defect, a consequence of harmful environmental factors affecting a susceptible embryo (1, 7). In cats, some breed predisposition to VSD is highlighted (6). The primary method in the diagnosis of VSD is echocardiography. Nowadays, thanks to the development of echocardiography, an increasing number of cases of this abnormality are discovered. A VSD located directly below the aortic and pulmonary artery valve is called a subclavian defect, otherwise known as a double committed (subclavian and supraclavicular) defect. The atrioventricular canal (endocardial protrusion defects, atrioventricular septal defect, atrioventricular canal (AVC) defect) comprises a group of congenital defects that share various degrees of atrial and interventricular septal underdevelopment and abnormal atrioventricular valves (this concerns the mitral and tricuspid valves) (9).

Considering the haemodynamic abnormalities resulting from the disease in question, VSDs can be divided into:

- mild VSD, otherwise known as restrictive defects, where the pressure gradient between the left and right ventricles is constant, and where the VSD can sometimes close spontaneously;
- moderate VSD, which may produce clinical symptoms;

• severe VSD, where pressure equalisation in the right and left ventricles occurs;
• Eisenmenger syndrome, where the pressure in the right ventricle begins to exceed that in the left ventricle, and there is a reversal of flow, which is called 'right-to-left' flow.

The objective of this study was to determine the incidence and type of ventricular septal defects (VSD) in a feline population over six years of follow-up.

Material and methods

The study material consisted of data in the clinical records of the Department of Epizootiology and Clinic of Infectious Diseases at the University of Life Sciences in Lublin. The period analysed in detail for this paper spanned from January 2018 to December 2023. The authors conducted a detailed analysis of the incidence, locations and clinical significance of VSD.

During this period, 866 cats were examined cardiologically, including 506 males, (58.4%) and 360 females (41.6%). Among those animals, there were 389 purebred cats (44.9%) and 477 non-purebred cats (55.1%). They comprised 255 young cats of up to 2 years of age (29.4%), 434 cats aged 2 to 10 years (50.1%) and 177 old cats over 10 years of age (20.4%). A total of 31 cats were diagnosed with VSDs, which accounted for 3.5% of all cats examined.

This study included a group of 31 cats in which a VSD was confirmed by echocardiography. All cats had normal results of bloodwork and basic biochemical panels, including aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (AP), urea, creatinine (CREA) and total bilirubin (BIL T).

The VSDs were classifed according to Soto into the following groups (12):

• Group 1 – Perimembranous defects (most common), adjacent to the fibrous ring of the atrioventricular valves. Perimembranous defects are usually not severe and are located below the aortic valve, although they can sometimes involve other parts of the septum (Fig. 1);
• Group 2 – Muscle defects located in muscle segments apically or in the middle part of the interventricular septum, not bordered by fibrous rings. They can be singular and isolated (less frequent) or multiple (more frequent). Extensive defects in the muscular part can quickly lead to the development of pulmonary hypertension. A colour Doppler then shows a very characteristic short stream of blood flow or no flow at all, with the same echo colour on the left and right sides of the ventricle;
• Group 3 – Malalignment outflow tract defects, coexisting with aortic dextroposition, (Fallot syndrome, biostial right ventricle) are those in which the conus septum is displaced in relation to the muscular septum;
• Group 4 – Inflow defect of the atrioventricular canal type (in association with a common atrioventricular valve), or of the atrioventricular defect type (coexisting with a rightward shift of the aorta). Large inflow defects usually coexist with a common atrioventricular valve and are referred to as complete atrioventricular canal (CAVC) defects (Fig. 2).

The 31 cats tested were 13 males (41.9%) and 18 females (58.1%) aged between 2 months and 4 years. The animals

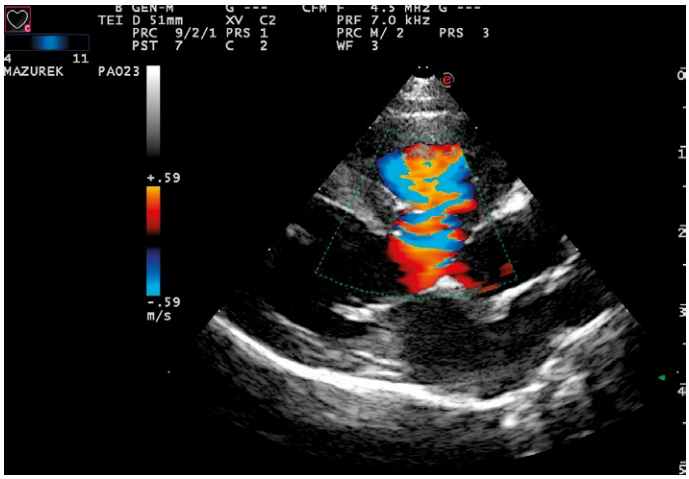

Fig. 1. Ventricular septal defect in the membranous septum with left-to-right flow diagnosed in a 3-year-old mixed breed female

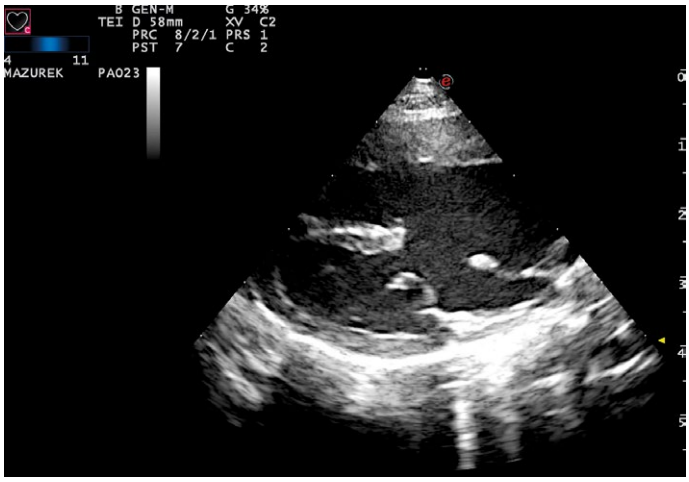

Fig. 2. Atrioventricular canal (AVC) defects diagnosed in a one-year-old Norwegian Forest male

were subjected to a routine clinical examination. All of them revealed varying degrees of heart murmur on auscultation. Impaired activity and dyspnoea were found in 14 cats, while no clinical signs were found in the remaining 17.

Cardiological examination. Echocardiographic examinations of the animals were performed in the supine position using an Esaote Mylab Class C ultrasound machine with a PA023 4-12 MHz phased-array multifrequency cardiac transducer. Each time, the myocardial structure and function were assessed.

The examinations were performed with two-dimensional (2D) imaging, M-mode measurements and the pulsed/colour-labelled Doppler technique. Two-dimensional imaging included long-axis and short-axis imaging planes from the right and left parasternal acoustic windows. The left atrial and aortic diameters were measured to determine the ratio of the aorta to the left atrium. The following measurements were taken on M-mode examination of the left ventricle:

• left ventricular lumen in diastole (LVDd),
• left ventricular lumen in systole (LVDs),
• interventricular septal thickness in diastole (IVSd),
• interventricular septal thickness in systole (IVSs),
• left ventricular posterior wall thickness in diastole (PWd),

• left ventricular posterior wall thickness in systole (PWs),
• fractional shortening (FS).

Each time, the flow through the VSD was assessed using CFM (Color Doppler Flow Mapping) and CW (Continuous Wave Doppler).

The following were analysed in detail: type of the ventricular defect – NUMBERING (1-4), age of the animal, breed, sex, clinical signs, such as reduced physical activity observed by the owner, dyspnoea. The cats included in the study are summarised in Table 1.

Statistical analysis. As VSDs are a congenital defect, the correlation between their incidence and the age of the animals was not investigated. However, the relationship between the VSD type and the incidence of clinical signs, as well as the breed and sex of the cats, was statistically analysed. This was done using Pearson's chi-square independence test with Yates correction. Values with a probability of $p < 0.05$ were considered statistically significant. Correlations between the parameters studied were calculated by the Spearman method.

Tab. 1. Cats included in the retrospective study

Patient no.	VSD type	Sex	Breed	Age	Clinical signs (reduced activity, dyspnoea)
1	1	Female	Mixed-breed	1 year	–
2	4	Male	Maine Coon	6 mo.	+
3	1	Male	Maine Coon	2 mo.	–
4	1	Female	British cat	3 years	+
5	1	Female	Siberian cat	1 year	–
6	1	Male	Norwegian Forest Cat	6 mo.	+
7	4	Male	Mixed-breed	6 mo.	+
8	1	Female	Maine Coon	2 mo.	–
9	4	Male	Norwegian Forest Cat	1 year	+
10	1	Female	Mixed-breed	3 years	+
11	4	Male	Norwegian Forest Cat	2 mo.	+
12	4	Female	Mixed-breed	1 year	+
13	1	Male	Mixed-breed	6 mo.	–
14	1	Female	Maine Coon	3 years	+
15	1	Male	Mixed-breed	2 mo.	–
16	1	Female	Mixed-breed	3 years	–
17	4	Female	Siberian cat	1 year	+
18	1	Male	Mixed-breed	1 year	–
19	1	Male	Mixed-breed	2 mo.	–
20	1	Female	Norwegian Forest Cat	6 mo.	–
21	1	Male	Sphynx	6 mo.	–
22	3	Female	Norwegian Forest Cat	1 year	+
23	1	Male	Mixed-breed	3 years	–
24	1	Female	Mixed-breed	1 year	–
25	1	Male	Siberian cat	3 years	–
26	4	Female	British cat	4 years	+
27	4	Female	Norwegian Forest Cat	4 years	+
28	4	Female	Norwegian Forest Cat	3 years	–
29	4	Female	Mixed-breed	3 years	–
30	1	Female	British cat	4 years	+
31	1	Female	Sphynx	6 mo.	–

Results and discussion

All specimens were found to have a VSD (100%) by cardiac echo examination. By far the most common type of VSD was in the perimembranous part with left-to-right flow, which was diagnosed in 20 animals (64.5%). No VSD of the membranous part with reverse flow was found. The second most commonly diagnosed VSD was a complete atrioventricular canal (CAVC) defect, observed in 10 cats (32.2%). A malalignment outflow tract defect was diagnosed in 1 of the study animals (3.2%). A muscular VSD defect was not diagnosed in any of the cases. For comparison, in Schrope's research (11), 17 cats had an isolated AVSD, which was partial in 13 cats and complete in the other 4. Double outlet right atrium (DORA) was diagnosed in 4 of the 17 cats. Among the animals with a partial AVSD, 7 cats had an atrial communication while the other 6 had a ventricular communication (11).

All animals with diagnosed VSDs were young (the oldest animal was 4 years old). This defect is congenital, which means that it is present from birth. Most animals were diagnosed at one year of age, n = 8 (25.8%) or at three years of age, n = 8 (25.5%). At 6 months of age, a VSD was found in 7 cats (22.5%), at 2 months in 5 cats (16.1%), and in 3 cats at 4 years of age (9.16%).

Statistical analysis showed no significant relationship between the incidence of VSD and the breed of the cats studied. Although the study revealed the presence of VSD in a higher number of pedigree cats (Tab. 1), these results were not statistically significant ($p = 0.4292$). In our study, VSDs were most frequently diagnosed in European (mixed-breed) cats, with 12 individuals (38.7%), followed by 7 Norwegian Forest Cats (22.5%), 4 Maine Coon cats (12.9%), 3 British cats (9.6%), 3 Siberian cats 9.6%) and 2 Sphynx cats (6.4%). These observations are consistent with those by (13), who also did not demonstrate that breed was a predisposing factor for VSD.

Although the VSD in question was found more frequently in females (58.1%) than males (41.9%), statistical analysis showed no significant relationship between the type of VSD or the presence of clinical signs and the sex of the cats ($p = 0.79446$ and $p = 0.524$, respectively).

Not all cats included in the follow-up displayed clinical signs of myocardial failure. The owners noticed changes such as reduced physical activity and dyspnoea in only less than half of the study cats with VSD (n = 14; 45%). Echocardiography showed signs of congestive heart failure and pulmonary oedema. Statistical analysis revealed a significant statistical

relationship (p = 0.00416) between the type of VSD per the Soto classification (groups 1-4) and the incidence of clinical signs in the cats studied. The severity of clinical signs appeared to depend on the location and size of the VSD. This is further confirmed by the positive and high correlation (r = 0.8632) between the study variables, which showed that clinical signs increased with the progression of pathological changes in the ventricular septum (Fig. 3).

A population of cats with VSD was included in this study, taking into account the different VSD morphotic types, the breed and sex of the animals and the clinical signs found in them. There are few reports in the available references concerning epidemiological studies of VSD in this animal population. Bomassi et al. (2) studied a group of 27 cats with VSDs. The most common diagnosis was, as in our study, a VSD in the membranous part.

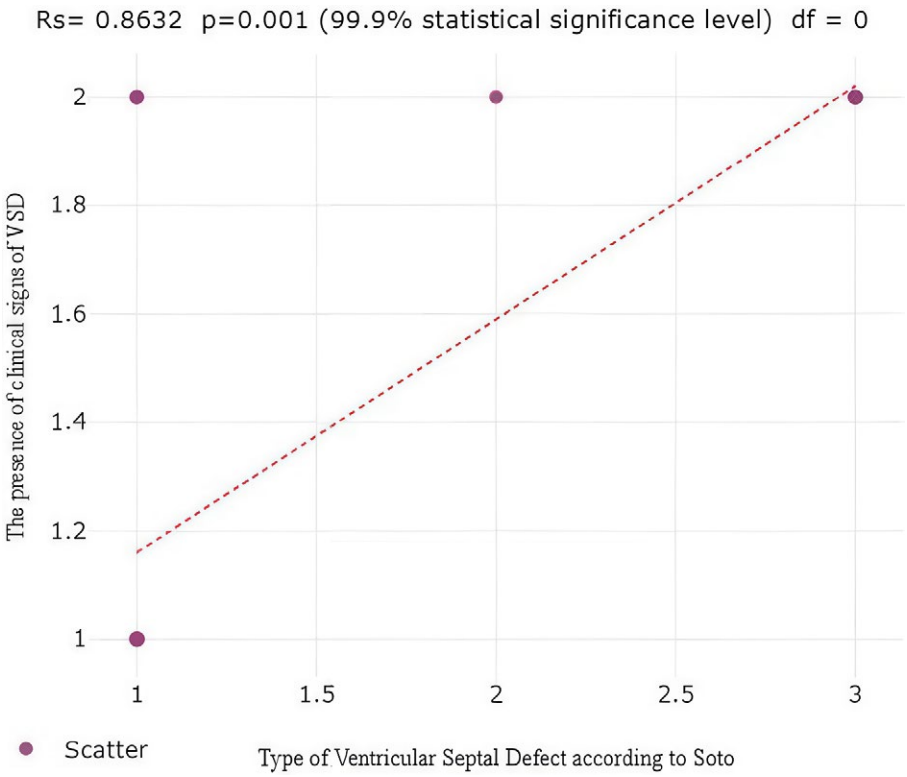

Fig. 3.

A ventricular septal defect is the cause of left-to-right blood flow during left ventricular contraction, with blood flowing into the right ventricle. It can cause increased pulmonary flow, left atrial enlargement and left ventricular overload. As a consequence of these disorders, circulatory failure and pulmonary hypertension can develop. The severity and nature of clinical signs observed in cats with this disorder depend on the size of the VSD and the size of the leak. A small VSD may be revealed by nothing more than a loud systolic murmur. A severe VSD in the first months of life causes increased signs of circulatory failure, with accelerated breathing, tachycardia and stunted weight gain. Such cases are reported by Schrope (11). High pulmonary flow usually results in a murmur over the mitral valve. Some patients have a diastolic murmur due to aortic regurgitation. Such disorders are reported by Riesen (10) and Schrope (11). The results of our own study indicate that, in cats, even a heart defect as extensive as VSD does not always manifest clinically, so owners may be unaware of their pet's disorder. Given this, it seems reasonable that cats should undergo follow-up echocardiography at least once a year.

In addition, the results of our own study show that clinical signs may not appear until Soto Group 4. This should prompt veterinarians to perform echocardiography more frequently and routinely, even for follow-up purposes, which could facilitate faster diagnosis of VSD in cats, even before the onset of clinical signs. Early identification of the condition, in its turn, will allow appropriate therapeutic management to be introduced depending on the location and size of the VSD, thus reducing the risk of clinical signs affecting the comfort and life expectancy of affected cats.

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