ESAVS
European School for Advanced Veterinary Studies

Case Report Instructions
EMSAVM / MASVM Cardiology

General instructions

- Case reports, written in prose, must be in a problem-oriented approach and include a complete presentation of the case, illustrations where available and a short discussion of the case with the current literature with references. Candidates must demonstrate a comprehensive understanding of the topic with assessing all obtained diagnostic test results.
- A case report should contain 2000 words +/- 10%, excluding tables, references and appendix. Case reports > 2400 words will automatically be denied (0 points) or sent back for rewriting.
- The 10 cases must be a mixture of various species, problems and diagnosis, all pertaining to the selected master's program. Candidates are required to keep a table of the already submitted cases which shall be send with each new case report submission. The ESAVS Office will provide an Excel template for the table below:

Case Nr.	Species	Problem/s	Diagnosis

- Candidates are advised to submit cases shortly after beginning and throughout the program and not all cases at the end of the program.
- ESAVS cannot guarantee the evaluation of more than 3 case reports per semester. To ensure an evaluation in a specific semester, reports should be submitted no later than the given deadline for the respective semester (please see Important Dates on the ESAVS website).

Cases should be set out under the following headings:

- Title
- Signalement
- Case History and physical Examination
- Case assessment including complete problem list, differential diagnosis with likelihood of what is possible for the case, tests performed and interpretation of these in relation to the case – do not use bullet points but write in prose
- Diagnosis
- Treatment (drugs need exact dosages) and adequate follow up
- Discussion of case in relation to current literature (no repetition of literature but a discussion why the case fits or does not fit what is known)
- References
- Appendix with laboratory results and diagnostic imaging pictures including interpretation (the examination board member reserves the right to see the original results (laboratory, diagnostic imaging) of selected cases)

Each case report is viewed by one member of the Examination Board and graded on a 0-20 scale (<10= fail, 10-11.9 = sufficient, 12-13.9 = fair, 14-15.9 = good, 16-17.9 = very good, 18-20 = excellent).

ESAVS
European School for Advanced Veterinary Studies

Evaluation of a case report

Step 1: Is the case report acceptable?

Is the case described in the report suitable at all? Reasons to reject a case are:

- A case is too simple (e.g. a dog with tetanus)
- Lack of adequate state of the art clinical tests to arrive at a diagnosis (or at least a presumptive diagnosis). The case could be resubmitted when the lacking information can be retrieved.
- The animal's life was endangered by excessive/unnecessary diagnostic tests or treatments (including surgery). Such a case cannot be resubmitted.
- A case that falls not within the specified master program (e.g. a pure internal medicine case or a pure gynecology case for the cardiology master)
- Most diagnostic tests and interpretation are done by referring veterinarian
- Inadequate follow-up of case (e.g. diagnosis reached after euthanasia with no follow-up available)
- Multiple cases all with same problems or diagnosis (e.g. many cases with vomiting and diarrhea in the internal medicine master program)
- Cases not seen during the enrollment in the program of the master student or where the master student is not the primary responsible clinician.
- More than 2400 words.

If a case is rejected the case report is assigned 0 points. The reason will be stated in the evaluation.

Step 2: Grading of the accepted case report

The case report will be evaluated based on a check sheet

An accepted case can reach a maximum of 20 points. A minimum of 10 points is required to pass.

The check sheet (see below) contains a list of 9 potential inadequacies. For each one the examiner can allocate a number of points. At the end a total number of points are given.

Recommendations for the candidate to avoid deduction of points:

- Make sure the history is sufficient
- Give all details of the physical exam
- Reported tests need to be relevant for the animal and interpretation needs to be concise and also relevant.
- Do not just give a list of all potential differentials, but explain why a differential might be more or less likely. Explain why you rule-out some differentials. The assessment of the case is very important, in order that the examiner can follow your thoughts and why you chose which further diagnostic steps
- Discuss your case – do not just repeat text book knowledge. If something has not been done or is abnormal and does not fit, try to explain this with pertinent literature.
- Show all results – missing graphics generally lead to points deducted.
- Treatment must be correct for the dog or cat
- Give information about outcome and therapy. Be specific.

ESAVS
European School for Advanced Veterinary Studies

Case Report Evaluation Check Sheet / Cardiology

Name of candidate: _____

Case report number/ title: _____

For students who have enrolled in a Master of Advanced Studies in Veterinary Medicine (MASVM) or European Master of Small Animal Veterinary Medicine (EMSAVM) program **before the winter semester 2024-2025**, the following grading criteria apply:

- The grades of the individual case reports are averaged to obtain one single grade. When this average grade is **below 10**, candidates are requested to resubmit revised versions of the failed case reports or new cases.
- A case report may not be acceptable and may be rejected if critical concerns in one (or several) areas result in a fail, regardless of whether all other required criteria are adequately met.

For students who have enrolled in a Master of Advanced Studies in Veterinary Medicine (MASVM) program for the first time **from the winter semester 2024-2025 onwards**, the following **new** grading criteria apply:

- 1. Pass = 10 points and more
- 2. Fail (case report insufficient) = below 10 points
  - modifications required - resubmission possible
  - case report insufficient - 0 points resubmission of this case report not possible – a new case report needs to be submitted

- **IMPORTANT:** the **average grade** for the module must be **13 points or higher** and none of the case reports must be graded below 10 points.
- A case report may not be acceptable and may be rejected if critical concerns in one (or several) areas result in a fail, regardless of whether all other required criteria are adequately met.

ESAVS
European School for Advanced Veterinary Studies

The maximum grade of a case report is 20 points. The second column indicates the maximum number of points that can be reached. In the third column the examiner indicates the number of achieved points, half points may also be allocated.

	Maximum points	Allocated points
Complete signalment, history and physical examination *Comments:*	1	
Problem list *Comments:*	1	
Differentials/ assessment for the problem list (the candidate should tailor and rank the differentials to the individual patient, not list every possible differential for each problem) *Comments:*	2	
Adequate and/or appropriate tests – and **assessment** of the test results (available results must be assessed for the submitted case) *Comments:*	3	
Diagnostic tests adequately graphically presented (radiographs, ECG, endoscopy, etc. **must** be shown in adequate size and quality) *Comments:*	3	
Correct and justified diagnosis *Comments:*	2	
Adequate or appropriate therapeutic management including generic drug names and dosages *Comments:*	3	
Discussion pertaining to submitted case, adequately referenced *Comments:*	4	
Special features not covered above (i.e. Language and word count adequate, adequate follow-up)	1	
TOTAL POINTS/ GRADE	20	

There is no “perfect” case and thus the attached example should be viewed more as how to present your case. If you have questions, please ask them during one of the courses early on – the course masters are ready and willing to help.

Case report # 4

Title:

A mature dog with Mitral Valve Stenosis (MVS), a congenital disease that decompensated later in life.

Signalement and history:

An 8-year-old mix breed FI dog with a BW of 26kg was presented for cardiac evaluation. The main complaint from the owners has been increasing bouts of muscle weakness and syncope-like episodes with a short duration. Additionally, there was an acute severe episode of gastrointestinal problem requiring hospitalization at another veterinary clinic, where a heart disease was suspected and therapy with high doses of diuretic (furosemide 4mg/kg/12h PO) and atenolol (2mg/kg/12h PO) was already started. According to the owners, the episodes decreased after the therapy start, but due to the lack of a specific diagnosis they sought a second opinion.

Physical examination:

Clinical examination of the patient revealed good general condition, preserved mental status and slightly increased body mass index of 6-7/10. Normal body temperature of 38.5^0C and peripheral perfusion indices - within normal limits. On auscultation there was a soft plateau 3/6 diastolic murmur best appreciated at the left heart base, and a soft 1-2/6 systolic one with a PMI at the MV area. No jugular pulse or abnormalities on palpation of the abdomen detected.

Case assessment, tests performed and their interpretation:

The initial cardiological examination consisted of electrocardiography, thoracic radiography, echocardiography, blood work and thyroid hormones profile.

The ambulatory ECG was performed in right lateral recumbence and documented a normal sinus rhythm, sinus arrhythmia and a HR of 70bpm. Normal MEA with positive R waves in leads I and aVF, normal in amplitude of the QRS complexes, but borderline as width – with 0.6s duration which is the upper normal limit, which could be attributed to some degree of conduction disturbance or left ventricle enlargement; wide and notchy P waves compatible with “p-mitrale” (p-wave = 0.08s, normal < 0.04s), an ECG sign for left atrial enlargement (Fig 1).

Chest radiographs were obtained in two orthogonal views - left lateral and ventro-dorsal. Again, the suspicion of left atrial enlargement from the ECG was confirmed - with a rentgenographic sign of isolated left atrial size increase – a "backpack sign" in the lateral view and a "cowboy leg sign" in the ventro-dorsal view, where there was also marked local cardiomegaly at the level of the left auricle (at 3 o'clock position). Interestingly, the rest of the cardiac silhouette margins were normal and the elevated VHS of 12.5 (normal < 11.3), as a sign of generalised cardiomegaly, was attributed only to the size of the left atrium. The caudal vena cava, aorta and the cranial pairs of pulmonary vessels were considered normal. Lung parenchyma with slightly increased "sand-glass" radiographic density, with diffuse interstitial and bronchial pattern - mainly due to age-related changes and the patient's conformation. Presence of several thin pleural fissures without clinical significance were recorded. No evidence of pleural effusion, good detail in the abdominal region available for interpretation. The rest of the study was unremarkable (Fig 2).

Transthoracic echocardiography was performed with a low frequency 2-5 MHz phase array probe. In B-mode, moderate atrial dilatation was noted (AO 19.1mm, normal 15.0-28.7mm; LA 37.3mm, normal 19.0-34.2mm, LA/AO ratio – 1.95, normal 0.83-1.13)[1] with a normal left ventricle; markedly reduced mitral valve orifice excursion with leaflet doming into the left ventricle during diastole. Both MV leaflets were thickened and nodular, with reduced orifice. In addition, M-mode echocardiographic findings showed a decreased mitral valve E to F slope and abnormal mitral valve leaflet motion. Color Doppler imaging showed color jets with aliasing, mosaic signal originating from the stenotic mitral orifice and extending into the left ventricle during diastole. Only mild and central MV insufficiency was recorded in the left atrium during systole. Spectral Doppler recordings showed transvalvular mitral valve gradients of up to 18 mmHg and prolonged pressure half-times. In B mode the MV orifice was assessed in all available US windows and the maximum width measured was around 4.5mm. The systolic function of the heart was preserved with a good wall to chamber indices and adequate FS of 40%. The normalized LV end-diastolic dimension was normal (LVIDdN 1.64, normal<1.85). The right atrium and ventricle were unremarkable and there were no pleural and/or pericardial effusions (Fig 3).

A standard blood work was done and apart from a mild prerenal azotemia with BUN 13mmol/L (normal 3.6-8.9mmol/L) all other parameters in the referral limits. The free T4 was also normal – 29nmol/L (normal 13-51nmol/L).

Diagnosis:

Mitral Valve Stenosis (MVS) as a single congenital cardiac defect.

Treatment and follow-up:

Despite the patient's advanced age, the diagnosed heart disease was clearly defined as a congenital pathology. This was the guiding factor in assessing whether the clinical picture that was actually observed (gastrointestinal signs and seizures) was due to this long standing compensated condition or was a coexisting finding. The decision was made to modify the assigned therapy in the context of the absence of signs of heart failure and to follow the patient's response over time.

The prescribed diuretic was replaced by another one from the same group - Torasemide instead of Furosemide, and the dose reduced to 0.2mg/kg SID. Despite the lack of direct evidence of congestive heart failure, the decision for a diuretic therapy was in the context of the increased size of the left atrium and the increased pressure within it. Atenolol was discontinued and replaced with Pimobendan – again, to benefit the left atrial pump function, at a dose of 0.25mg/kg BID. The owner was advised to count and log the SRR and, if possible, to video record the "episodes" to better assess their nature and try to distinguish between syncope and seizure.

Over the next two months, the dog was followed by phone, and the owner reported a good general condition, normal respiratory rate, and only two brief episodes consistent with seizure activity - with tonic/clonic limb movements, preserved consciousness, and a short post-ictal phase. The dog was then presented to the clinic as an emergency - with acute weakness and tachypnea. According to the owner, she had deteriorated over one night and was unable to walk normally, with shallow and rapid breathing. Clinical examination revealed a fast and irregular heart rate, tachypnea with RR of 60/min and otherwise good general condition. The ECG showed a fast and irregularly irregular rhythm consistent with atrial fibrillation with a HR of 240 bpm (Fig 4), and the chest X-rays were consistent with left-sided CHF - with marked cardiomegaly with a VHS of 14.0, severe left atrial enlargement, prominent pulmonary veins and a general increase in lung parenchyma lucency, especially in the hilar region (Fig 5). The focused echocardiography showed increased atrial dimension with an abnormal LA/AO ratio of 2.65 (AO 18.8mm and LA 49.9mm).

In-hospital therapy for the signs of CHF was started with flow-by oxygen and a bolus of furosemide at 4mg/kg IV, then two consecutive boluses of 2mg/kg IV each. The clinical signs

resolved rapidly and the patient had a normal RR after the first 6 hours of hospitalisation. Digoxin was also started as a rate control antiarrhythmic at 0.033mg/kg/12h PO. On day two the dog was discharged and the therapy was adjusted accordingly: Torasemide 0.2mg/kg/12h PO, Pimobendan 0.5mg/kg/12h PO, Benazepril 0.25mg/kg/24h PO and Digoxin 0.033mg/kg/12h PO. Again, because of the benign presentation of the seizures (less than once per month and with a short duration) a therapy in that direction was postponed and only strict monitoring was advised.

The patient was discharged on the second day with normal RR and good general condition. No seizures were observed during the hospital stay. Over the next two weeks, several follow-up consultations were made by telephone and the owner reported a good clinical response without any concerns. Then, 14 days later, a new clinical examination was carried out and the ECG was repeated - showing a much slower heart rate of 110 bpm, still consistent with AF (Fig 6). Due to intermittent vomiting in the recent past, the blood was checked for digoxin levels, which came back as optimal - 1.49ng/ml (normal 0.9-2.0ng/ml). The blood tests were also normal with only mild prerenal azotemia with BUN 17.0mmol/L (normal 3.6-8.9mmol/L).

Over the next three years, we were able to manage the patent heart failure with gradual adjustments in therapy, reaching a full spectrum of sequential nephron blockade with torasemide 0.3mg/kg/12h PO, hydrochlorothiazide 2mg/kg/24h PO and spironolactone 2mg/kg/24h PO; pimobendan 0.5mg/kg/12h PO; benazepril 0.25mg/kg/24h PO; digoxin 0.033mg/kg/12h PO, diltiazem 2mg/kg/8h PO and some adjuncts like taurine, L-carnitine and coenzyme Q10. The dog was re-examined over 15 times and the heart size progression was very well documented with a VHS of 16.0 at the last presentations (Fig 7).

The patient's seizures were also treated because of increasing episodes lasting more than one minute. Initially, only levetiracetam was prescribed at 20mg/kg/8h PO, then phenobarbital was added at 2.5mg/kg/12h PO, which completely controlled them. Unfortunately, a number of other diseases were eventually identified, such as endometritis, mammary gland carcinoma and hypothyroidism. This constellation of pathologies was treated with so many drugs that the owners became less and less compliant and when renal failure started to develop, the decision was made to euthanise – more than three years after the initial patient presentation. A necropsy was done and part of the left atrial myocardium (severely distended and modified) together with the stenotic MV lesion were sent for histology (Fig 8).

Histopathology revealed fibromyxomatous degeneration of the mitral valve and neutrophilic atrial myocarditis with severe chronic lesions: Diffusely, the mitral valve was severely expanded by an increased fibromyxomatous matrix consisting of nodules of interstitial cells in interlacing fascicles into a myxomatous stroma, and with central metaplasia into hyalinised chondrocytes, surrounded by mature connective tissue with occasional neovascularisation and diffuse infiltration by scattered lymphocytes, plasma cells, erythrocytes and haemosiderin-laden macrophages.

There was moderate, multifocal interstitial, predominantly neutrophilic with occasional scattered lymphocytes and plasma cells infiltration of the left atrial wall and multifocal perivascular infiltration by lymphocytes and plasma cells accompanied by mild fibrosis. In the atrial lumen remnants of fibrinonecrotic thrombus were also observed (Fig 9).

Discussion:

In our patients, mitral valve stenosis (MVS) is defined as a narrowing of the mitral valve orifice due to an abnormal mitral valve. This creates a pressure gradient between the ventricle and the atrium, resulting in abnormally high left atrial pressure. MVS is a rare cardiac abnormality and is almost always congenital, unlike in humans where it can result from rheumatic fever (a condition not reported in dogs)[2]. MVS is also extremely rare in humans, accounting for only 4 in 1000 babies with congenital heart disease[3]. It usually involves abnormalities of one or more components of the valve apparatus. The leaflets may be thickened, the chordae tendineae shortened or the papillary muscles hypoplastic. Although it is a progressive disease, MVS can be asymptomatic for many years and may not decompensate until later in life[2,3].

MVS is most commonly diagnosed in dogs and cats as an incidental finding or when the patient presents with heart failure[2]. If it is an isolated pathology, only a soft systolic and/or diastolic murmur will be found in the area of the MV. From the diagnostic tests, the ECG and XR may give the impression of a huge left atrium - with a wide and notched P wave on the ECG and characteristic rentgenographic bulges where the left atrial borders are expected on both orthogonal XR views. However, echocardiography is routinely used to make the final diagnosis - it shows the morphological changes in the valve apparatus and assesses the degree of blood flow obstruction. In humans, the grading system of disease severity is based on the pressure gradients across the stenotic area and is classified as mild (between 8-10mmHg), moderate (between 11-15mmHg) and

severe (when > 15mmHg)[3]. This finding, together with the patient's clinical symptoms, usually determines the treatment plan - medical or surgical.[3,4,5,6].

The treatment of the patients with MVS is generally medical and depends on the degree of the left heart failure clinical signs – from mild to moderate[4,7,10]. In contrast with humans, where the increased pulmonary vein pressure could lead to pulmonary hypertension and right heart failure, in dogs no such complications were reported associated with this condition. The drugs used are diuretics, ACE inhibitors and inotropes. If an arrhythmia is present – with atrial fibrillation one of the most common ones – an appropriate antiarrhythmic drug is used. The treatment in the severe cases in humans is generally surgical or interventional. In dogs there are some reported cases where a successful intervention is done in order to alleviate the stenosis – commissurotomy or valvuloplasty[5,6,8].

The treatment plan and especially the prognosis in patients with MVS both in human and in veterinary medicine strongly depends on the specific anatomical findings of the defect, the concurrent congenital conditions if any and the age and clinical signs at presentation. In some patients with a severe stenosis it is difficult to find the balance between the diuretic dose and the congestion control[9]. An excessive reduction in left atrial pressure with aggressive diuretic regimen could be actually bad for the patient, reducing the cardiac output precipitously. Generally, an aggressive preload reduction of the left atrium should be avoided[3]. This is a disease with known progressive nature in people, so even as an accidental finding, it should be followed strictly over time in dogs too.

In this particular case, the disease was a typical incidental finding and did not cause clinical symptoms for many years. I suspect that the treatment that was started (including a beta-blocker) may have altered the fine balance between volume and pressure overload and triggered the cascade for the disease to worsen[10,11]. Once on therapy, the dog was completely dependent on it and required constant monitoring and drug adjustments. Even though, due to the morphology of the valve leaflets (thick and fibrotic) no surgical or interventional treatment plan was proposed to the owner, we were able to give the patient a good quality of life for another three years.

References:

(1) Boon June A., BA MS, Two-dimensional and M-mode Echocardiography for the small animal practitioner, Textbook, Teton New Media, 2002, New York

(2) Kittleson MD. Other Congenital Cardiovascular Abnormalities. In: Kittleson MD, Kienle RD, editors. Small Animal Cardiovascular Medicine. St. Louis, Missouri: Mosby; 1998

(3) American College of Cardiology/American Heart Association Task Force on Practice Guidelines for management of patients with valvular diseases; Society of Thoracic Surgeons; Bonow RO, Carabello BA, Kanu C, de Leon AC Jr, Faxon DP, et all. Circulation. 2006 Aug 1;114(5): e84-231. doi: 10.1161/CIRCULATION AHA.106.176857. Erratum in: Circulation. 2010 Jun 15;121(23) PMID: 16880336.

(4) Lehmkuhl, L. B., Ware, W. A., & Bonagura, J. D. (1994). Mitral Stenosis in 15 Dogs. Journal of Veterinary Internal Medicine, 8(1), 2–17

(5) Borenstein, N., Daniel, P., Behr, L., Pouchelon, J. L., Carbognani, D., Pierrel, A., … Laborde, F. (2004). Successful Surgical Treatment of Mitral Valve Stenosis in a Dog. Veterinary Surgery, 33(2), 138–145

(6) Trehiou-Sechi, E., Behr, L., Chetboul, V., Pouchelon, J.-L., Castaignet, M., Gouni, V., Borenstein, N. (2011). Echoguided closed commissurotomy for mitral valve stenosis in a dog. Journal of Veterinary Cardiology, 13(3), 219–225

(7) Mitral Stenosis in Dogs. (1994). Journal of Veterinary Internal Medicine, 8(4), 310–314

(8) J.W. Arndt, M.A. Oyama, Balloon valvuloplasty of congenital mitral stenosis, J Vet Cardiol, 15 (2013), pp. 147-151

(9) M. Rishniw, H.N. Erb, Evaluation of four 2-dimensional echocardiographic methods of assessing left atrial size in dogs, J Vet Intern Med, 14 (2000), pp. 429-435

(10) Oyama, M. A., Weidman, J. A., & Cole, S. G. (2008). *Calculation of pressure half-time. Journal of Veterinary Cardiology, 10(1), 57–60*

(11) Meisner, J. S., Keren, G., Pajaro, O. E., Mani, A., Strom, J. A., Frater, R. W., Yellin, E. L. (1991). Atrial contribution to ventricular filling in mitral stenosis. Circulation, 84(4)

Appendix with figures and tables:

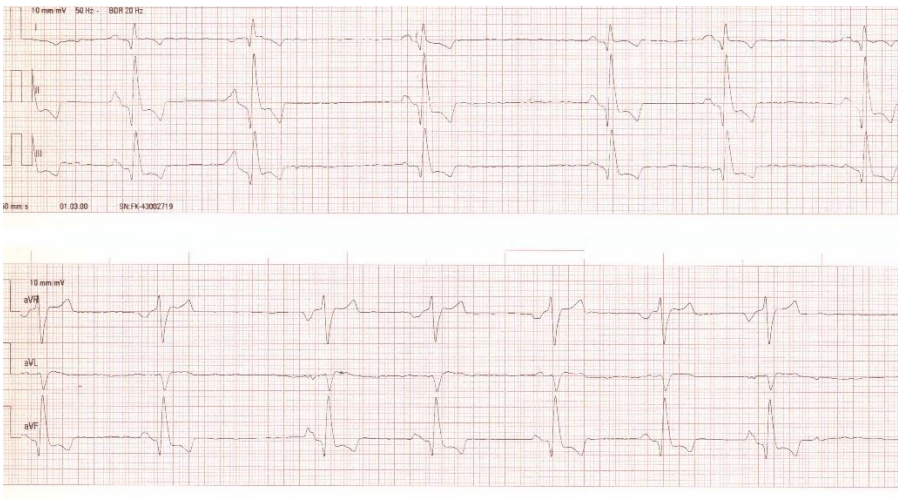


Fig.1 Six-lead ECG with sinus rhythm at 70bpm; 10 mm/mV, 25 mm/sec. Wide and notched P waves compatible with "p-mitrale"

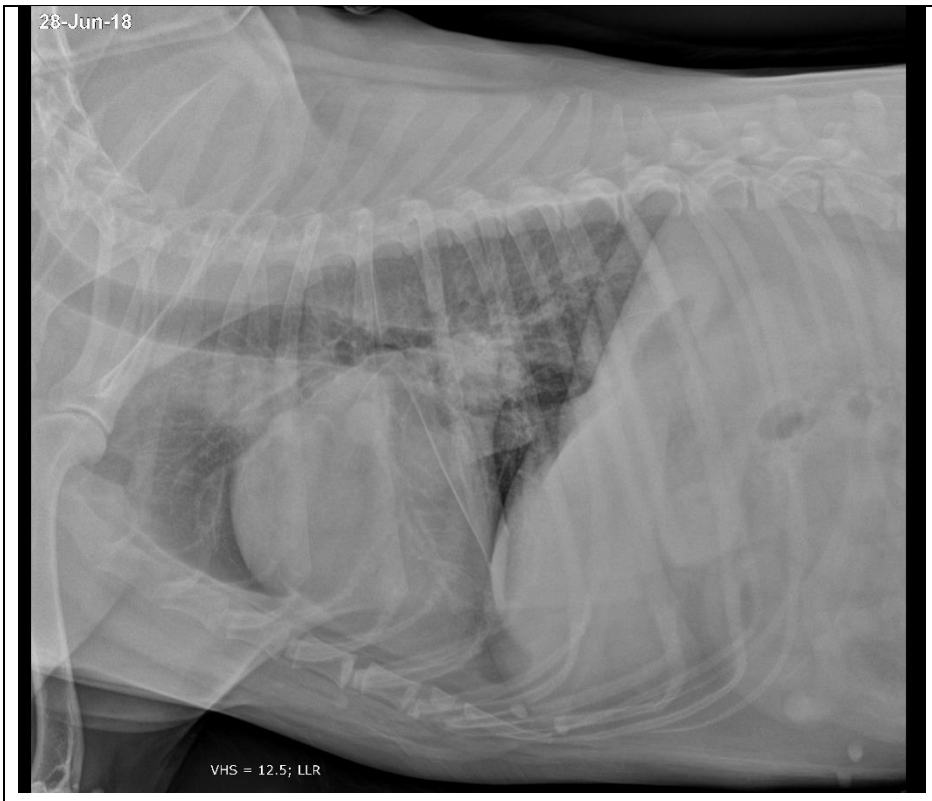
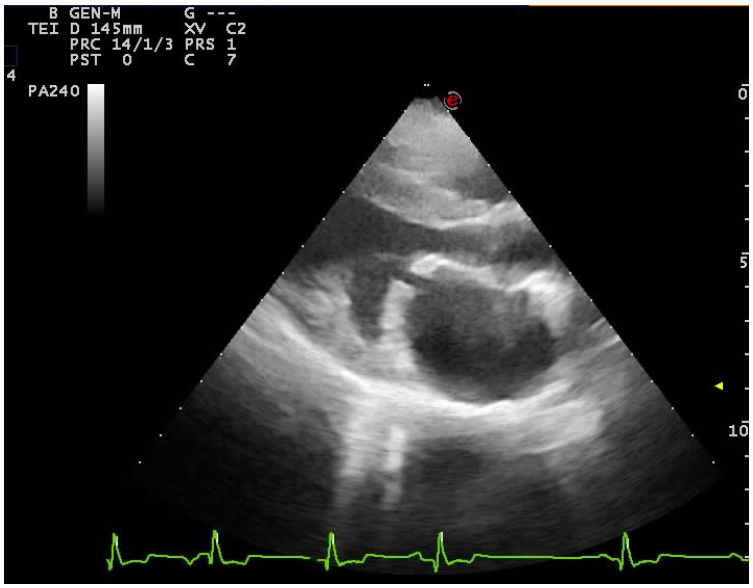
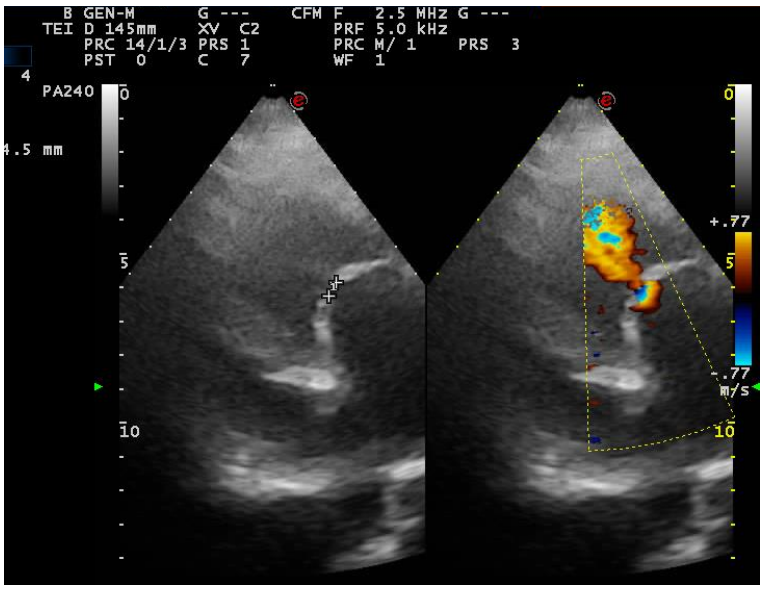


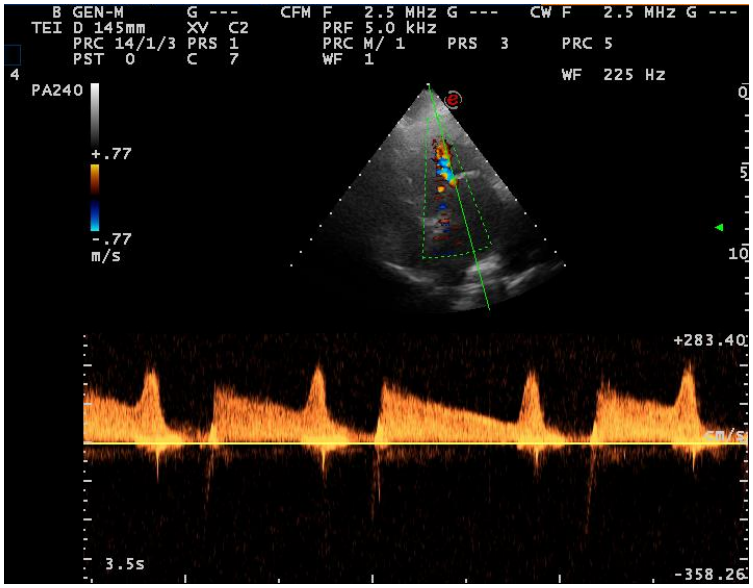
Fig.2. Left lateral thoracic X-ray demonstrating a local cardiomegaly at the level of the left atrium – a bulge between 12 and 3 o'clock positions, a "back-pack" radiographic sign


B GEN-M G ---
TEI D 145mm XV C2
PRC 14/1/3 PRS 1
PST 0 C 7
PA240
0
5
10

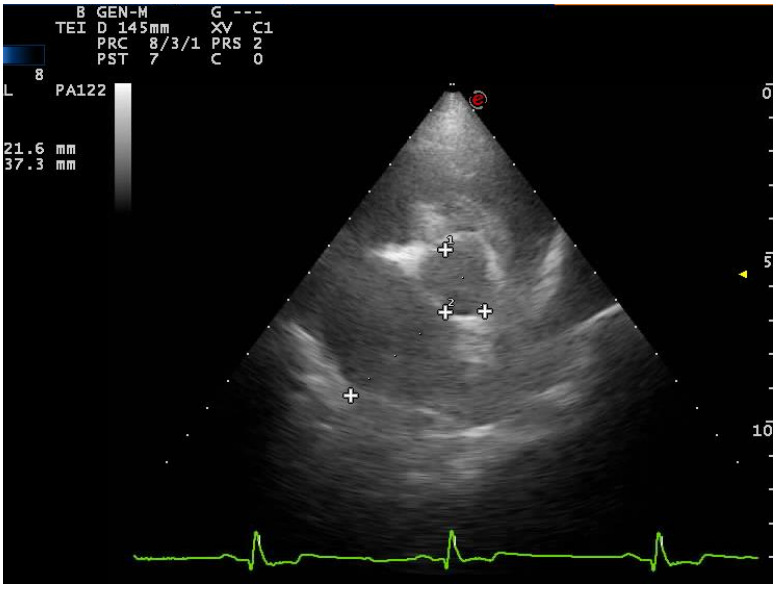
(A)


B GEN-M G --- CFM F 2.5 MHz G ---
TEI D 145mm XV C2 PRF 5.0 kHz
PRC 14/1/3 PRS 1 PRC M/ 1 PRS 3
PST 0 C 7 WF 1
PA240
4.5 mm
+.77
-.77
m/s
0
5
10

(B)

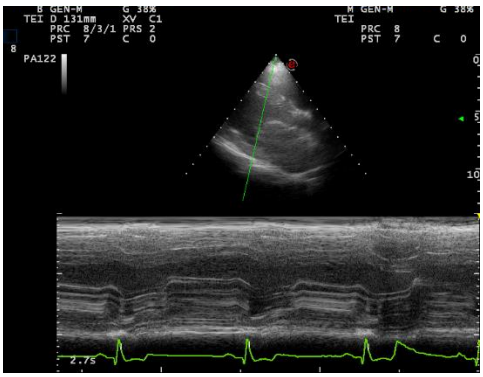


(C)

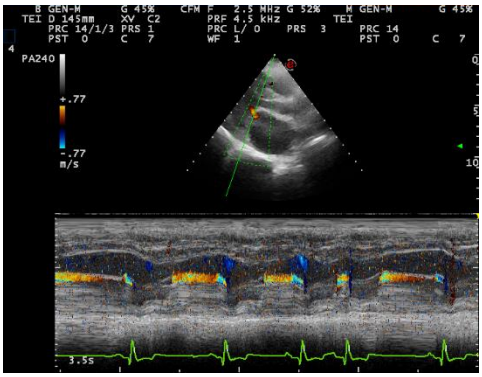


(D)

Fig.3 The mitral valve apparatus was severely deformed and the leaflets fused, and dooming into the left ventricle during diastole leaving only a small, incomplete orifice (A); the lack of sufficient MV opening was assessed and demonstrated in B mode where a turbulent, aliased signal was seen on CD mode protruding deep in the left ventricle chamber during diastole (B); the spectral Doppler profile of the MV was also abnormal; CWD profile revealed a long E wave deceleration time and high A wave with a velocity of 2.14m/s translating in a 18mmHg gradient across the valve (C); LA/AO ratio assessment in B mode (D).



(E)



(F)

(E and F) The M mode and the Color M mode of the MV were also severely abnormal – with indistinguishable E and A peaks and turbulent, mosaic flow throughout diastole

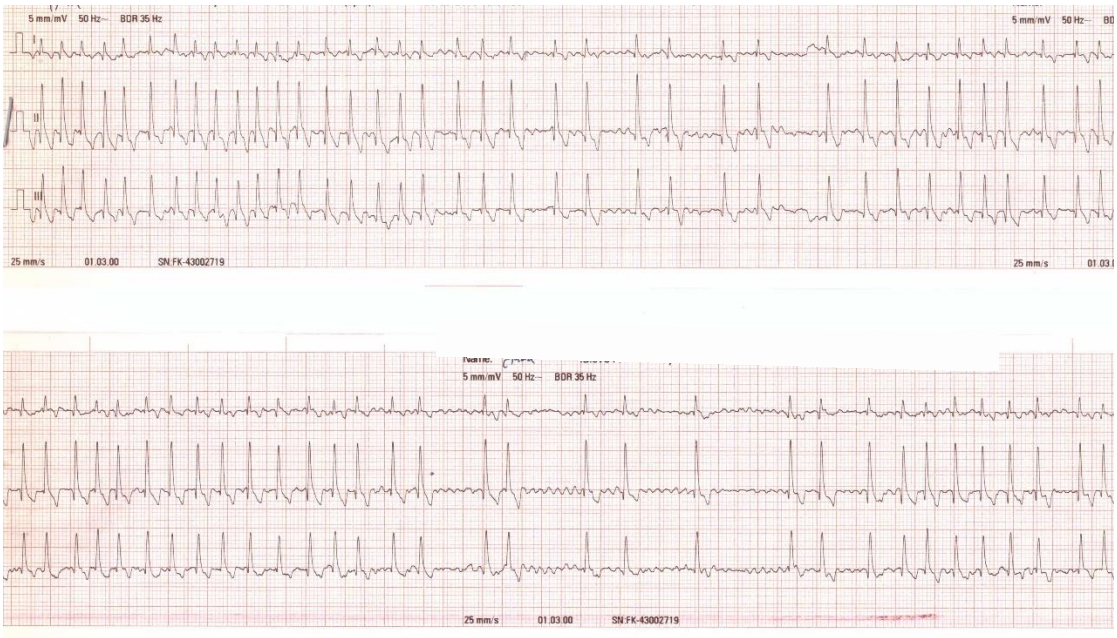


Fig.4 Six-lead ECG with atrial fibrillation and HR up to 260bpm; 5 mm/mV, 25 mm/sec. Fast, supraventricular and irregularly irregular rhythm without P waves; a possible interpretation of the ECG finding could be an atrial flutter, but the irregularity of the rhythm on the long runs of ECG recorded were more in favor of atrial fibrillation

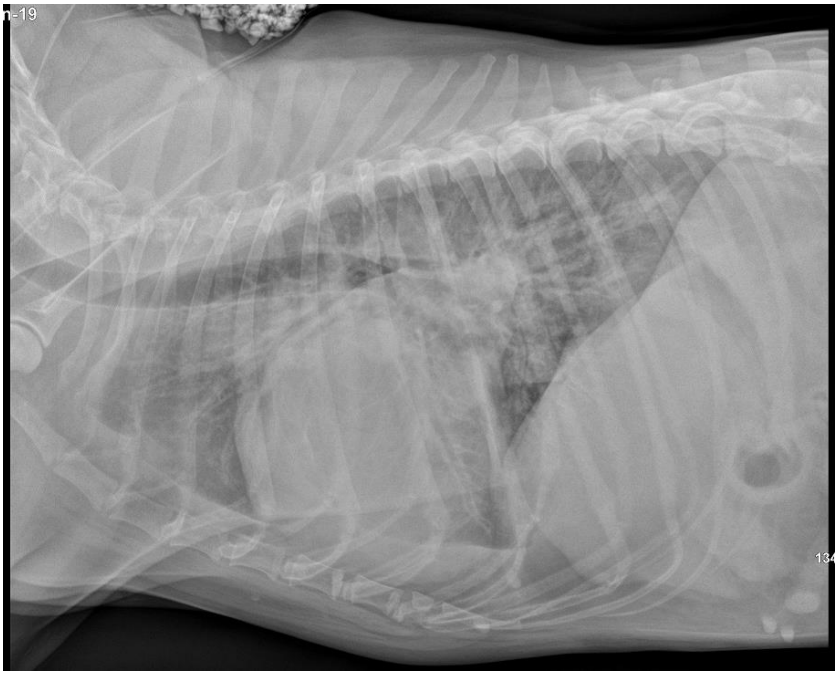

September 2018

Fig.5 Left lateral thoracic X-ray with radiographic signs compatible with LCHF – severely enlarged left atrium, prominent pulmonary veins and diffusely increased lung opacity starting from the perihillar area with cardiac borders and vascular edge effacement. Together with the clinical findings like tachypnea and dyspnea this is suggestive for a pulmonary edema and needs an aggressive management.

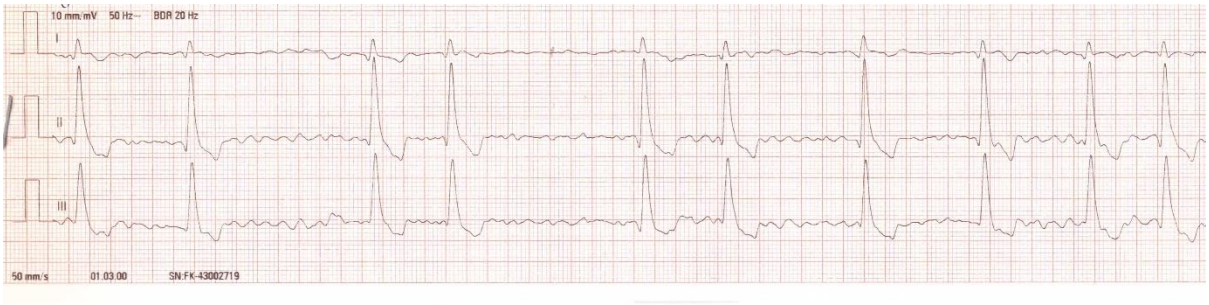
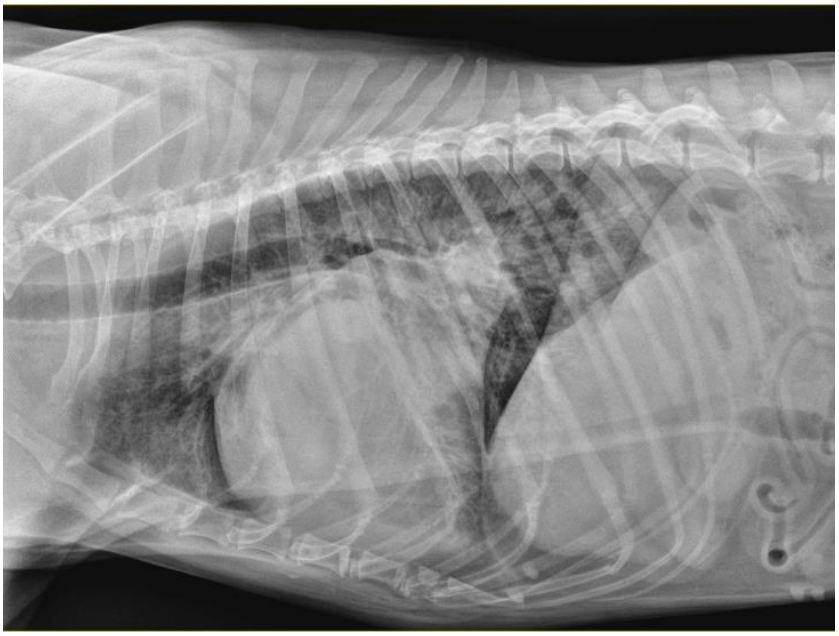
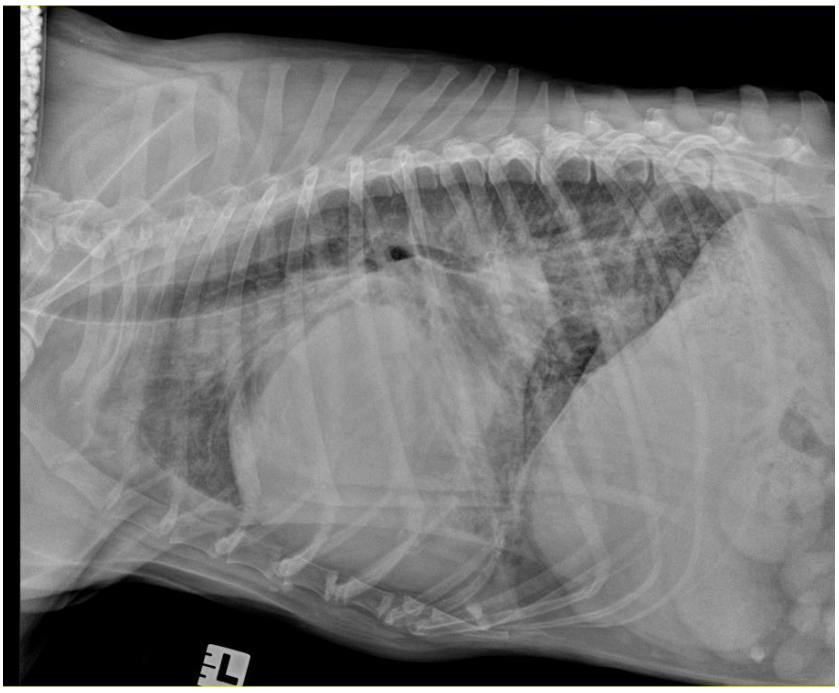


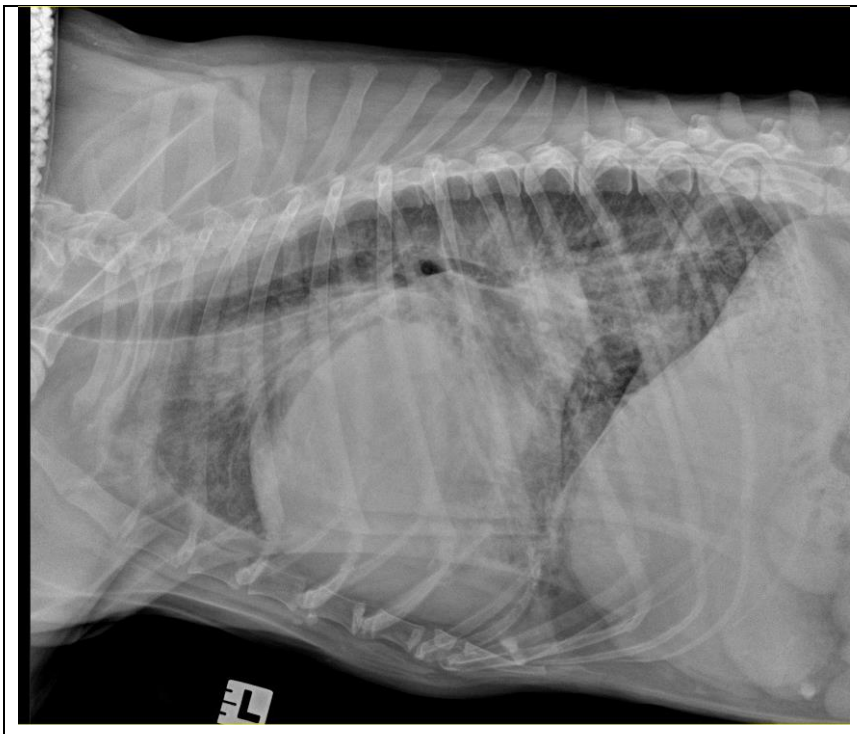
Fig.6 ECG in lead I, II and III; still with atrial fibrillation but the HR is decreased to average of 140bpm; 10mm/mV, 50mm/sec. The patient is receiving Digoxin at 0.033mg/kg/12h PO for the last three weeks as a single antiarrhythmic drug.



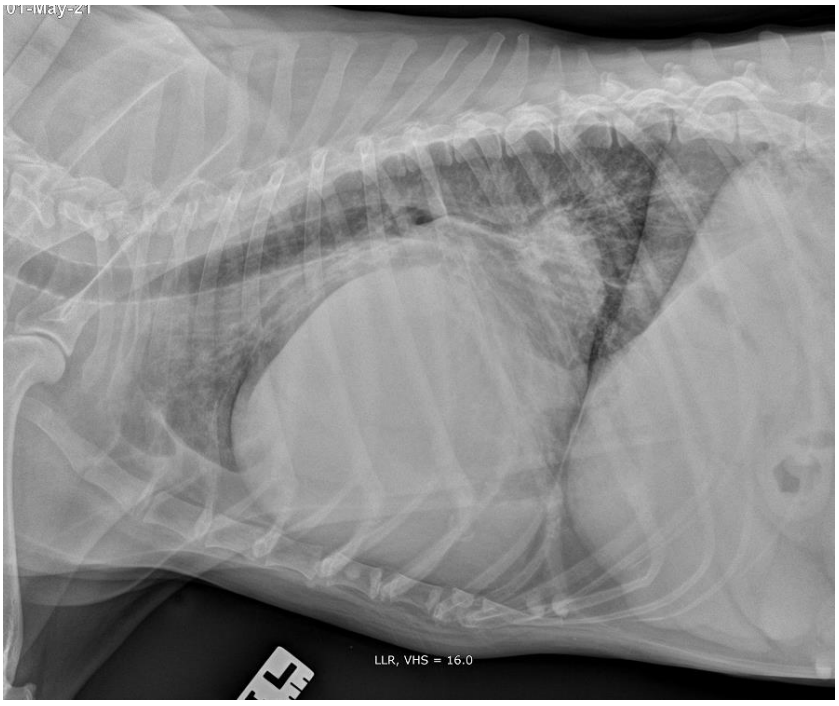
June 2019



March 2020

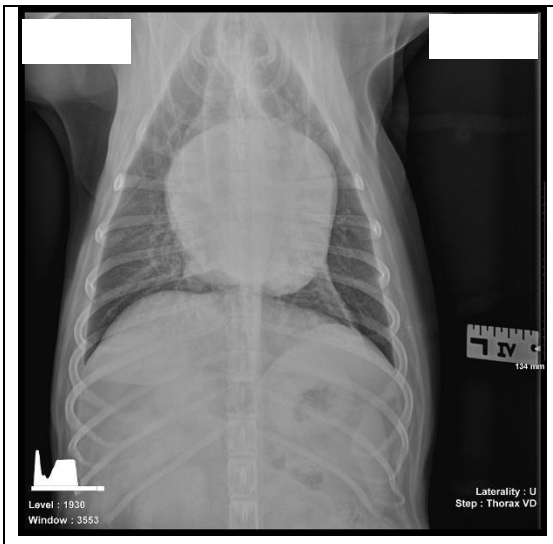


September 2020

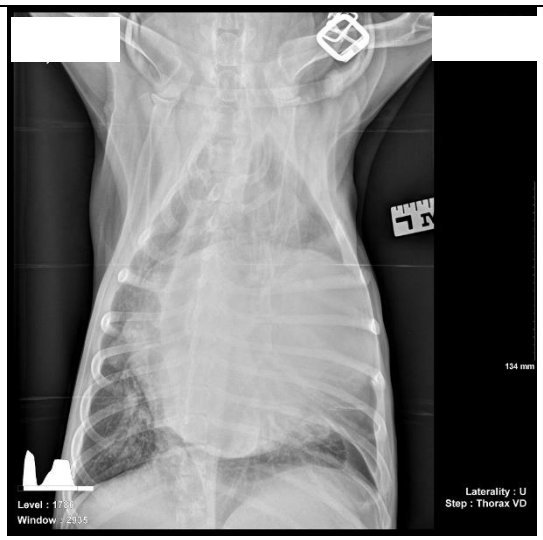


May 2021

Fig.7 It was possible to follow the patient through these two years both as a clinical exams and as an X-ray changes evolution. The owners were very strictly following the SRR and when it was going up they presented the dog for reevaluation. The left ventricle was not volume overloaded, but the left atrium was huge and the intraatrial pressure high.



(A)



(B)

The difference between the VD view from June 2018 (A) and May 2021 (B); here, too, it is clearly seen that the left atrium occupies a large part of the cardiac silhouette and contributes most to this generalized cardiomegaly.

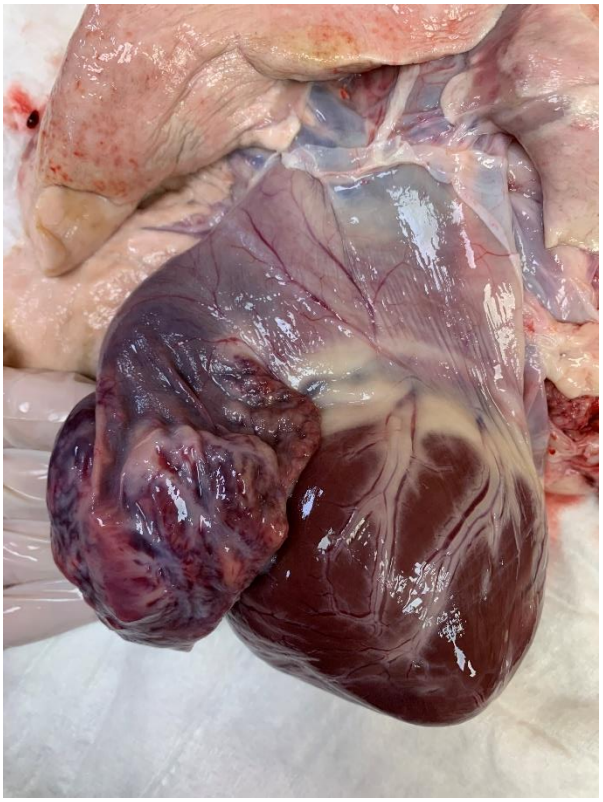
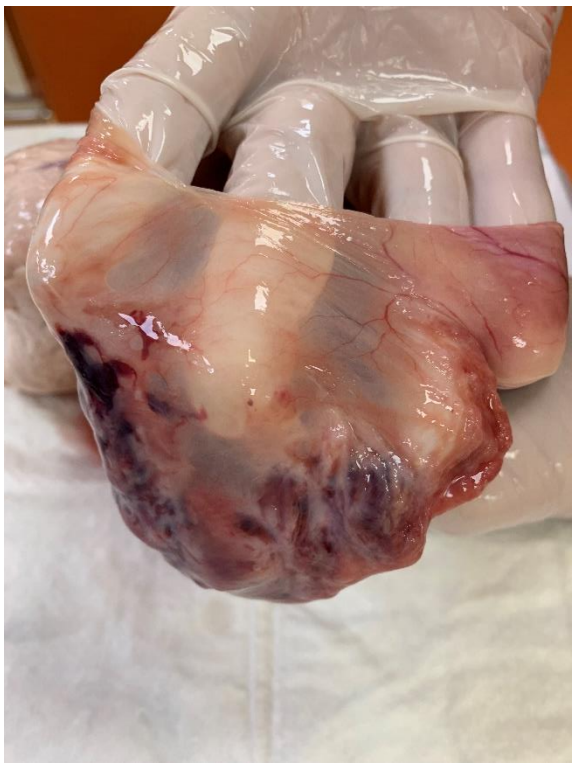
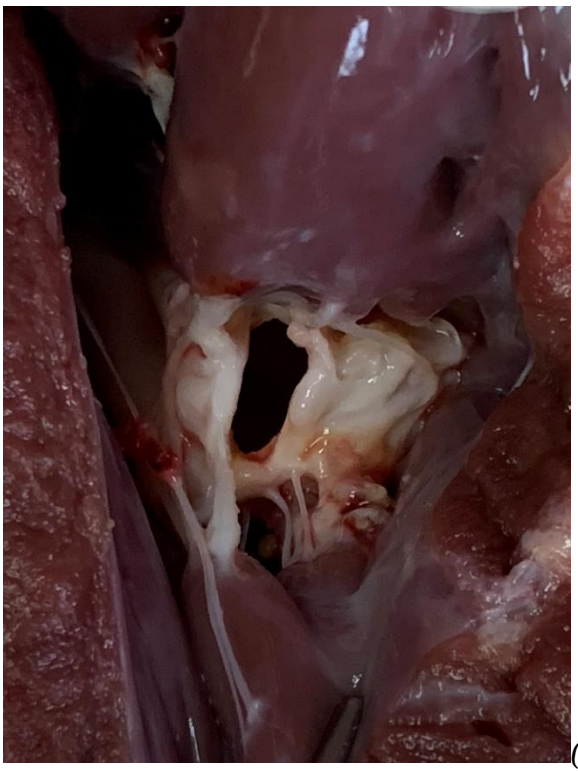


Fig. 8.1 The gross appearance of the heart with both left ventricle and left atrium exposed; the left auricle is severely enlarged and partly filled with stiff thrombi – recognized in that area echcardiographically pre-mortem.

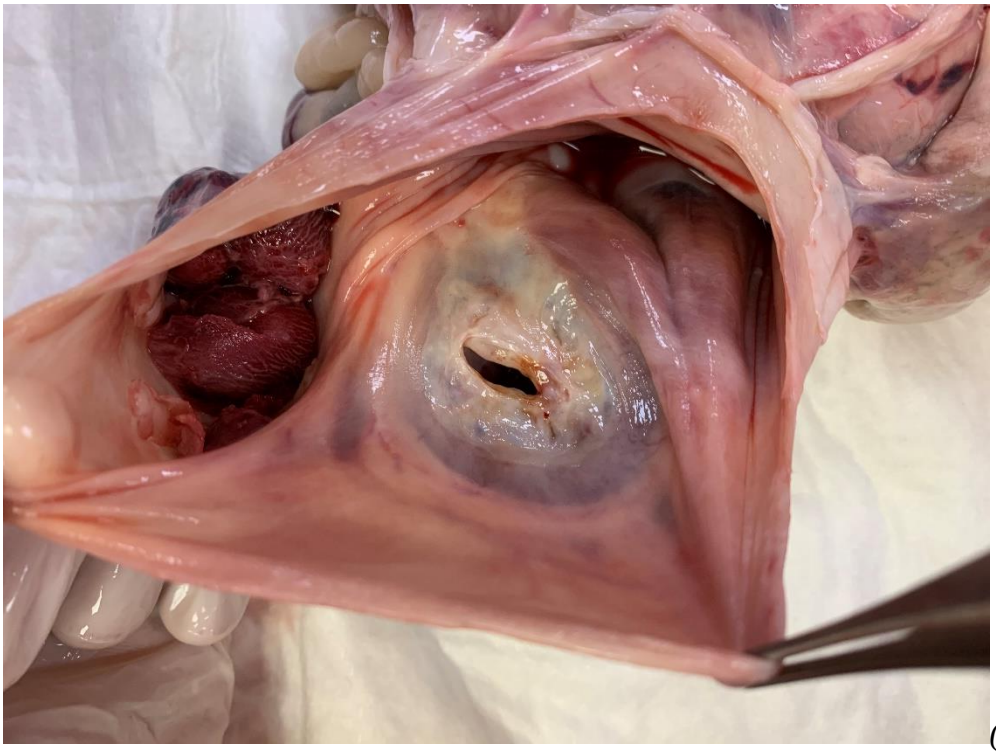


(A)

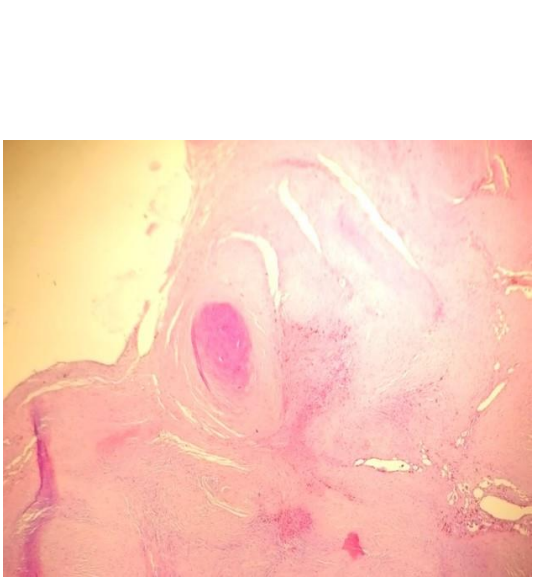


(B)

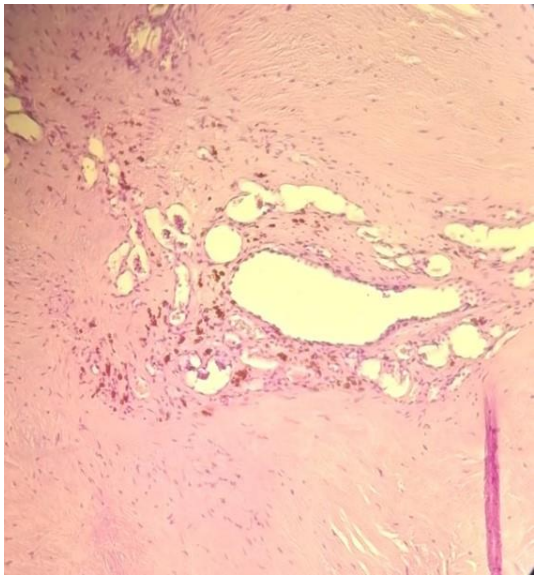
Fig. 8.2 Severely stretched and transparent left atrial wall at the level of the left atrial appendage (A), the MV from the ventricle side (B); and the MV "surgical view" from the left atrial side (C).



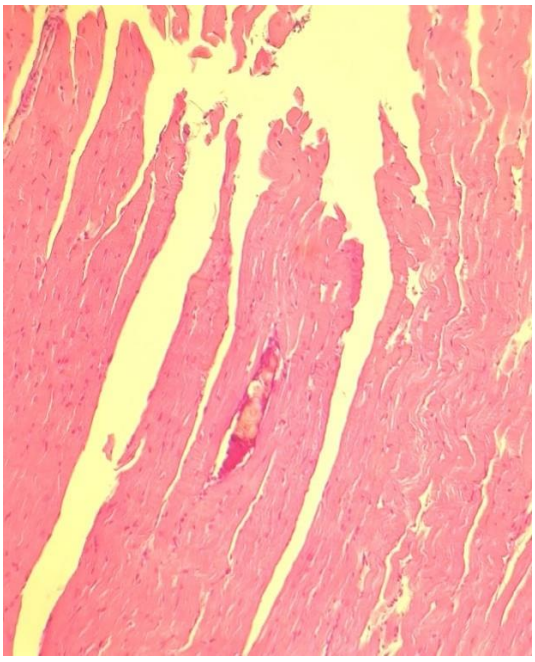
(C)



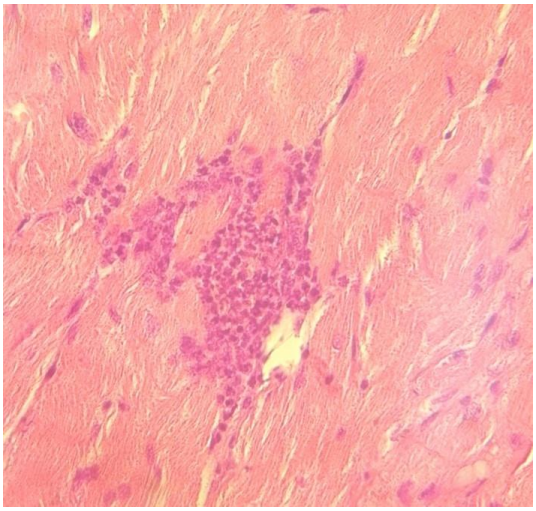
(A)



(B)



(C)



(D)

Fig. 9 The histopathology micro photos from the MV stenotic lesion (A and B) demonstrating the severe fibromyxomatous degeneration; atrial myocardium (C and D) with evidence of a chronic neutrophilic myocarditis.