Traumatic pericarditis

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- Perforation of the pericardial sac by a sharp foreign body originating in the reticulum causes pericarditis with the development of toxemia and congestive heart failure.
- Tachycardia, fever, engorgement of the jugular veins, anasarca, hydrothorax and ascites, and abnormalities of the heart sounds are the diagnostic features of the disease.

- **Etiology**

  Traumatic pericarditis is caused by penetration of the pericardial sac by a migrating metal foreign body from the reticulum.

- The incidence is greater during the last 3 months of pregnancy and at parturition than at other times.

- Approximately 8% of all cases of traumatic reticuloperitonitis will develop pericarditis.

- Most affected animals die or suffer from chronic pericarditis and do not return to completely normal health.

- **Pathogenesis**

1-The penetration of the pericardial sac may occur with the initial perforation of the reticular wall. However, the animal may have had a history of traumatic reticuloperitonitis some time previously, followed by pericarditis,

2- usually during late pregnancy or at parturition. In this case it is probable that the foreign body remains in a sinus in the reticular wall after the initial perforation and penetrates the pericardial Sac at a later date.

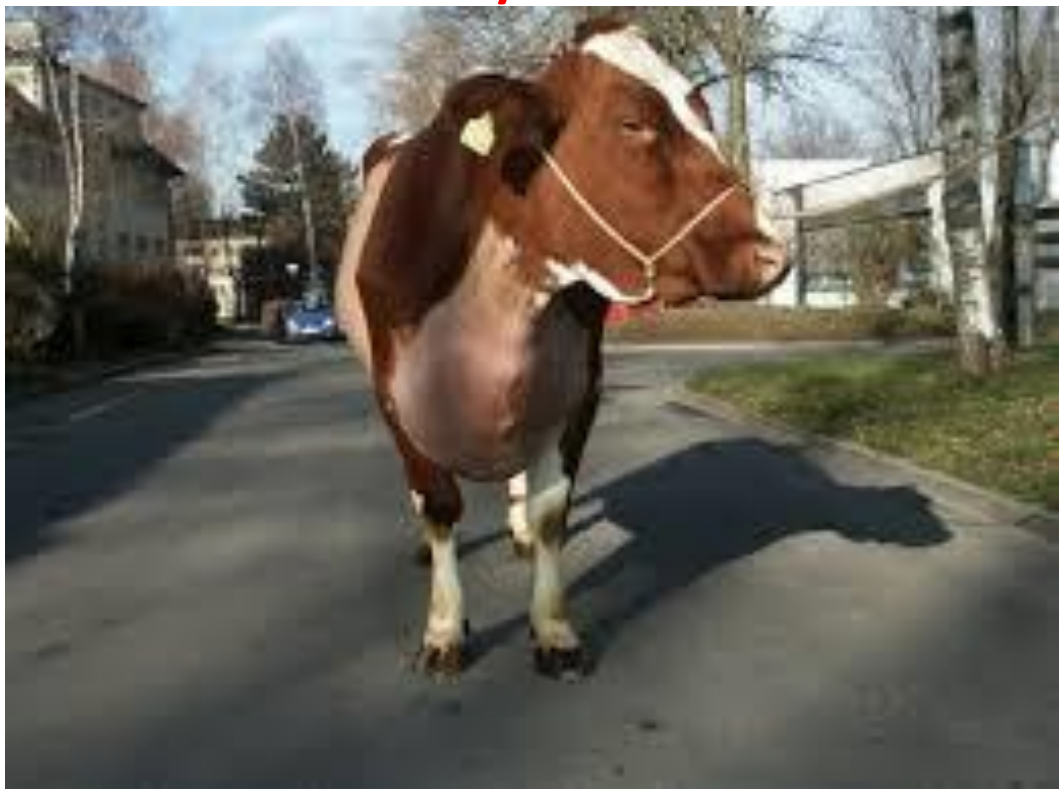
3-Physical penetration of the sac is not essential to the development of pericarditis, infection sometimes penetrating through the pericardium from a traumatic mediastinitis.

4-Introduction of a mixed bacterial infection from the reticulum causes a severe local inflamation , and persistence of the foreign body in the tissues is not essential for the further progress of the disease.

- 5-The first effect of the inflammation is hyperemia of the pericardial surfaces and the production of friction sounds synchronous with the heart beats.
- 6-Two mechanisms then operate to produce signs: the toxemia due to the infection and the pressure on the heart from the fluid which accumulates in the sac and produces congestive heart failure.

- 6- an affected animal may be severely ill for several weeks with edema developing only gradually, or extreme edema may develop within 2-3 days.
- The rapid development of edema usually indicates early death.

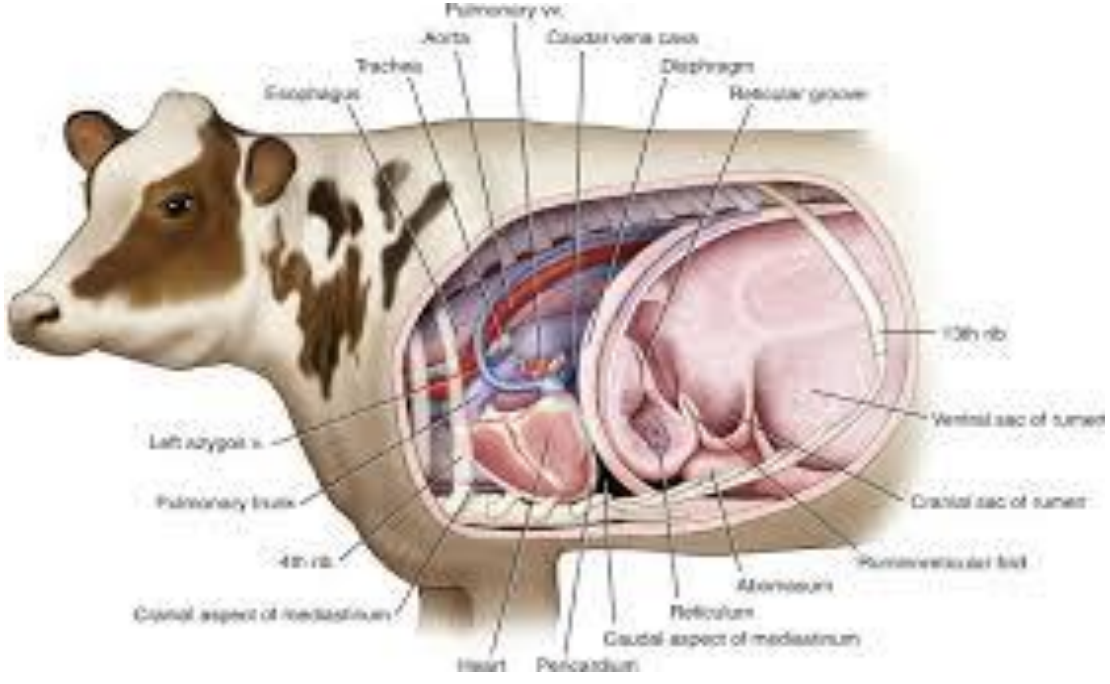
7-If chronic pericarditis persists there is restriction of the heart action due to adhesion of the pericardium to the heart.



- 8-Congestive heart failure results in most cases but some animals may recover.

  9- An uncommon sequel after perforation of the pericardial sac by a foreign body is rupture of a coronary artery or the ventricular wall.

  Death usually occurs suddenly due to acute, congestive heart failure .



- **Clinical findings**

1-Depression, anorexia, habitualy recumbency and rapid weight loss are common.

2-Diarrhea or scant feces may be present and grinding of the teeth, salivation and nasal discharge are occasionally observed.

3-The animal stands with the **back arched and the elbows abducted**.

4- Respiratory movements are more obvious, being mainly abdominal, shallow, increased in rate to 4O- 50/min and often accompanied by grunting.

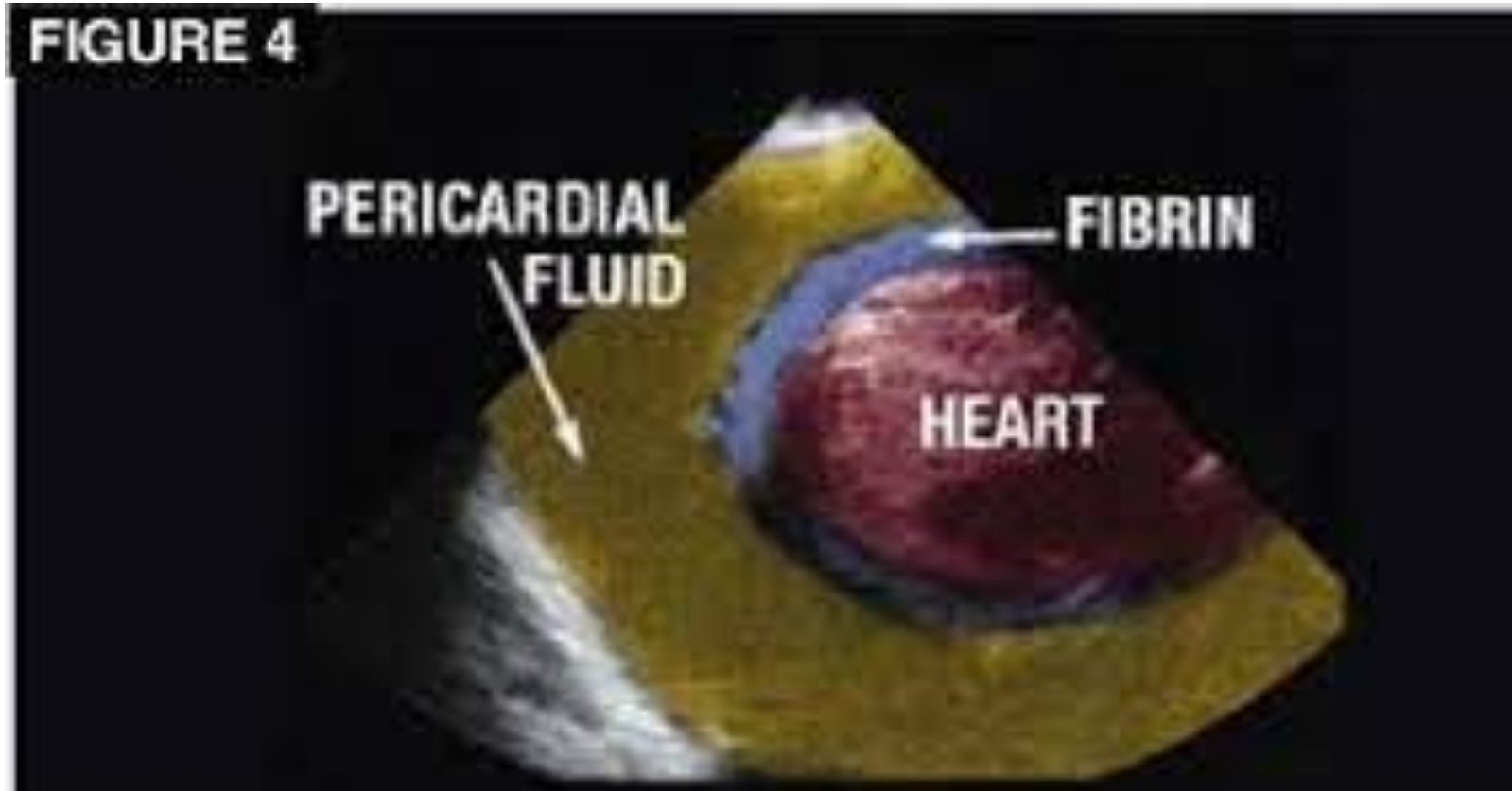
5-Engorgement of the jugular veins, and edema of the brisket and ventral abdominal wall are common

5-A prominent jugular venous pulse is usually visible and extends proximally up the neck.

6-Pyrexia (40-41C,) is common in the early stages and an increase in the heart rate to 100/min and a diminution in the pulse amplitude are constant.

7-Rumen movements are usually present but depressed. **Pinching** of the withers to depress the back or deep palpation of the ventral abdominal wall behind the xiphoid sternum commonly elicits a marked painful grunt.

8-Auscultation of the thorax reveals the heart sounds are normal but are accompanied by a pericardial friction rub. Care must be taken to differentiate this from a pleural friction rub due to inflammation of the mediastinum. In this case the rub is much louder and the heart rate will not be so high.



9-Several days later when there is marked effusion, the heart sounds are muffled and there may be gurgling, splashing or tinkling sounds.

10-Most affected animals die within a period of 1-2 weeks, although a small proportion persist with chronic pericarditis.

11-The obvious clinical findings in the terminal stages are gross edema, dyspnea, severe watery diarrhea, depression, recumbency and complete anorexia.

12-Enlargement of the liver may be detectable by palpation behind the upper part of the right costal arch in the cranial part of the right paralumbar fossa.

13-Death is usually due to asphyxia and toxemia. Animals which have recovered from an initial pericarditis are usually affected by the chronic form of the disease.

14-Body condition is poor, the appetite is variable, there is no systemic reaction and the demeanor is bright.

- **Clinical pathology**

  1-Hemogram A pronounced leukocytosis with a total count of 16 000-30 OOO/pL accompanied by a neutrophilia and eosinopenia is usual.

Necropsy findings

1-In acute cases there is gross distension of the pericardial sac with foul-smelling, grayish fluid containing flakes of fibrin, and the serous surface of the sac is covered by heavy deposits of newly formed fibrin.

2-A cord-like, fibrous sinus tract usually connects the reticulum with the pericardium.

3-Additional lesions of pleurisy and pneumonia are commonly present.

4-In chronic cases the pericardial sac is grossly thickened and fused to the pericardium by strong fibrous adhesions surrounding loculi of varying size which contain pus or thin straw-colored fluid.

- **Treatment**

The results of treatment are usually unsatisfactory but salvage of up to 50% of cases can be achieved by long-term treatment with antimicrobials. Selected cases of traumatic pericarditis have been treated satisfactorily by pericardiotomy.