Traumatic pericarditis

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 pericardia! 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, habitual 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 (40-41oC,) 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 diagnostic findings. In the early stages before effusion commences, the heart sounds are normal but are accompanied by a pericardial friction rub, which may wax and wane with respiratory movements.

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.