**INFECTIOUS NECROTIC HEPATITIS(BLACK DISEASE)**

Etiology An acute toxemia of sheep, cattle and sometimes pigs and horses caused by the toxin of *Clostridium novyi* elaborated in damaged liver tissue. Outbreaks usually associated with fascioliasis

**EPIDEMIOLOGY**

**1-Occurrence**

The disease is worldwide in distribution. In sheep, the incidence rate in a given year is usually about 5% in affected flocks but may be as high as 10-30%. The disease is always fatal in both sheep and cattle. The disease is rare in horses.

**2-Risk factors**

a-Animal risk factors Well-nourished adult sheep in the2-4-year age group are more susceptible than lambs and yearlings

animals.

b-Environmental risk factors

In temperate climates, a seasonal occurrence is marked because of fluctuation in the liver fluke and host snail population.

Exposure to fluke infestation, as occurs when sheep graze on marshy ground during dry summers and drought, is commonly associated with outbreaks of black disease

**Source of infection**

1-infected animals, the soil contaminated by career animal ,symptoms or, carcass.

2-transportion of career animal .

Wild animal (wild ruminant infection, wild carnivores, wild birds) .

4-flooding

**PATHOGENESIS**

After ingested Spores of *C. novyi* are carried to the liver through the lymphatic system. Under local anaerobic conditions, such as occur in the liver when migrating flukes cause severe tissue destruction, the organisms already present in the liver proliferate, liberating alpha toxin, which is necrotoxic and causes local liver necrosis and more diffuse damage to the vascular system. The nervous signs may be observed due to this general vascular disturbance or to a specific neurotoxin.

CLINICAL FINDINGS

**Sheep**

Affected sheep commonly die during the night and are found dead without having exhibited any previous signs of illness.

When observation is possible, some clinical signs on the animal before death.

1. segregate from the flock
2. become behind the flock and fall down if driven.

 3-There is fever (40-42°C) which subsides to subnormal level

 4-some hyperesthesia; respiration is rapid and shallow;

 5-the sheep remains in sternal recumbency and often dies within a few minutes while still in this position.

 6-The course from first illness to death is never more than a few hours and death usually occurs quietly, without evidence of struggling.

**Cattle**

Clinical findings are the same in cattle as in sheep but the course is longer, the illness lasting for 1-2 days

clinical findings include

1- a sudden severe depression

2- reluctance to move,

3-coldness of the skin

4- absence of rumen sounds

5- low or normal temperature.

6-weakness and muffling of the heart sounds.

7- abdominal pain, especially on deep palpation of the liver, and the feces are semi fluid.

**Horses**

In the horse, the syndrome presents as a peritonitis accompanied by severe and progressive toxemia and manifests with depression, reluctance to walk, pain on palpation of the abdomen, frequent straining and recumbency. Fluid from abdominal paracentesis has a profound increase in nucleated cells and protein. Death occurs within 72 hours of onset of

the disease.

**NECROPSY FINDINGS**

1-Bloodstained froth may exude from the nostrils.

2-The carcass undergoes rapid putrefaction.

3-engorgement of the subcutaneous vessels and a variable degree of subcutaneous edema.

4-Bloodstained serous fluid is always present in large amounts in the pericardial, pleural, and peritoneal cavities.

5-The liver is swollen, gray-brown and characteristic areas of necrosis, These are yellow areas 1-2 cm in diameter and are surrounded by a zone of bright red hyperemia.

**Histologically,**

the liver lesion consists of a central tract of eosinophilic inflammation (due to fluke migration) surrounded by a zone of coagulation necrosis

**Diagnosis of Black Disease** requires the

1- culture of C novyi from the typical liver lesion and the demonstration of preformed toxin in the peritoneal fluid and/or the liver lesion from a fresh carcass.

2-Fluorescent antibody techniques(FAT)

3-ELISA for beta toxin in intestinal contents

4-PCR

**Differential diagnosis**

• Acute fascioliasis in sheep can cause

heavy mortality due to massive liver

destruction at the same time and under

the same conditions as does black

disease

• Other clostridial disease - blackleg, malignant edema

• Anthrax

**TREATMENT**

No -effective treatment is available. In cattle and horses the longer course of the disease suggests the possibility of controlling the clostridial infection by the parenteral use of penicillin or broad-spectrum antibiotics

**CONTROL**

1-Vaccination with an alum-precipitated toxoid is highly effective. On an affected farm the initial vaccination is followed by a second vaccination 4-6 weeks later

2- control of the liver fluke. and the flukes eliminated

from the sheep by treatment with flukicides.

3-The host snail must be destroyed in

streams and marshes by the use of a molluscicide

4-Pasture contamination from cadavers should be minimized by burning the carcasses.