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Lecture title: **Infectious Necrotic Hepatitis (Black Disease)**

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Summary:

Infectious Necrotic Hepatitis (Black Disease)

ETIOLOGY:

1- The etiologic agent of infectious necrotic hepatitis affecting sheep and cattle and rarely pigs and horses is ***Clostridium novyi,*** **type B.**

2- Spores of ***C. novyi*** are resident in soil and may be present in the liver of normal animals. Clinical disease is triggered by a primary necrotic process in the liver, which causes the organism to proliferate and produce lethal amounts of toxin.

3- The disease has been produced experimentally in sheep by the administration of spores of *C. novyi* after prior infection with fluke metacercaria. Although field outbreaks of the disease are usually precipitated by invasion of the liver by immature liver flukes it is possible that other causes of local hepatic injury, e.g., invasion by cysts of ***Cysticercus tenuicollis***, and **trauma** from liver biopsy may precipitate the disease.

EPIDEMIOLOGY:

Occurrence

1- 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% to 30% and in rare cases up to 50%.

2- The disease is practically always **fatal in both sheep and cattle.** Details of the incidence in cattle are scanty, but the disease is becoming more common in some areas where fluke is being introduced. The disease is rare in horses.

Risk Factors:

Animal Risk Factors

1- Well-nourished adult sheep in the 2-4 years age group are particularly susceptible, and lambs and yearlings rarely are affected.

2- **Environmental Risk Factors** The epidemiologic association between **liver flukes** and ***C. novyi*** has been supported by the observation that both are more prevalent in the soil in areas where black disease occurs than in other areas, and the survival of both the bacteria and the fluke is favored by the same type of soil environment.

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

3- Outbreaks are most common in the summer or autumn months and cease within a few weeks after frosts occur because of destruction of encysted metacercaria.

4- 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, although they can occur in winter.

5- Heavy irrigation of pastures creates favorable conditions for the development of flukes and may predispose disease. Outbreaks in cattle commonly occur on irrigated farms.

Source of Infection:

1- Infection occurs through fecal–oral transmission of ***C. novyi.*** Fecal contamination of the pasture by carrier animals results in ingestion of clostridial spores by herd mates; the cadavers of sheep dead of the disease may cause heavy contamination. Many normal animals in flocks in which the disease occurs carry ***C. novyi*** in their livers.

2- The **spread of infection** from farm to farm occurs via these sheep and probably also by infected wild animals and birds and by the carriage of contaminated soil during flooding.

PATHOGENESIS:

Spores of ***C. novyi*** are ingested and carried to the liver in the lymphatic system; the organism can be isolated from the liver of normal animals. 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 **α-toxin**, which is **necrotoxic** and causes local liver necrosis and more diffuse damage to the vascular system. The nervous signs observed may be caused by this general vascular disturbance or by aspecific **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, **clinically affected sheep are seen** to segregate from the rest of the flock, lag behind, and fall down if driven. There is fever (40–42°C), which subsides to a premortal (subnormal) level, and some hyperesthesia; respiration is rapid and shallow; and the sheep remains in sternal recumbency and often dies within a few minutes while still in this position. 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, with the illness lasting for 1 to 2 days. Outstanding clinical findings in cattle include a sudden severe depression, reluctance to move, coldness of the skin, absence of rumen sounds, a low or normal temperature, and weakness and muffling of the heart sounds. There is **abdominal pain**, especially on deep palpation of the liver, and the feces are semifluid. Per orbital edema may also develop.

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.

CLINICAL PATHOLOGY:

Antemortem laboratory examinations are not usually possible because of the **peracute nature of the disease**, and there is no body of information for this disease.

NECROPSY FINDINGS:

Bloodstained froth may exude from the nostrils. The carcass undergoes rapid putrefaction. There is pronounced engorgement of the subcutaneous vessels and a variable degree of subcutaneous edema. The **dark appearance of the inside of the skin,** particularly noticeable on drying, has given rise to the name **black disease**. **Gelatinous exudate may be present in moderate quantities in the fascial planes of the abdominal musculature**. **Bloodstained serous fluid** is always present in abnormally large amounts in the pericardial, pleural, and peritoneal cavities. Subendocardial and subepicardial hemorrhages are frequent.

The liver is swollen, gray-brown, and exhibits characteristic areas of necrosis.

These are yellow areas 1 to 2 cm in diameter and are surrounded by a **zone of bright red hyperemia**.

DIFFERENTIAL DIAGNOSIS:

• **Acute fasciolosis** in sheep can cause heavy mortality caused by massive liver destruction at the same time and under the same conditions as does black disease.
• **Other clostridial disease** includes blackleg and malignant edema.
• **Anthrax**

TREATMENT:

No effective treatment is available. Although ***C. novyi*** is susceptible to **penicillin,** in sheep parenteral treatment with antimicrobials generally comes too late

once clinical signs are apparent because of the peracute course of the disease. 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, but reported cases have high case fatality.

CONTROL:

Vaccination with an alum-precipitated toxoid is highly effective and can be performed during the course of an outbreak. The mortality begins to subside within 2 weeks.

On an affected farm the initial vaccination is followed by a second vaccination 4 to 6 weeks later and subsequently by annual vaccinations. To provide maximum immunity at the time when the disease is most likely to occur, vaccination as a prophylactic measure should be performed in early summer.

Control of the disease should also be attempted by control of the liver fluke. The host snail must be destroyed in streams and marshes by the use of a molluscicide and the flukes eliminated from the sheep by treatment with flukicides. Pasture contamination from cadavers should be minimized by burning the carcasses.