1.2.7 Furunculosis

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A. Name of Disease and Etiological Agent

Furunculosis is caused by *Aeromonas salmonicida.*

B. Known Geographical Range and Host Species of the Disease

1. **Geographical Range**
   This disease is known to occur in North America, South America, Europe, Asia, and Africa.

2. **Host Species**
   Although it is usually associated with freshwater fishes, marine fishes are also considered to be susceptible.

C. Epizootiology

This organism has been known for almost 100 years, but the exact route of transmission is not completely understood. The primary points of controversy are: 1) the ability of the organism to exist in the natural environment outside the fish host, 2) the role of the natural environment in transmission, and 3) the mechanism(s) of entry into the fish. It is known that the organism can be transmitted horizontally both among and within fish populations, and is present at extremely low levels in carrier fish. However, vertical transmission of the bacterium has not yet been shown to occur in naturally infected fish populations. Studies on the ecology of the pathogen have been hampered by the lack of a suitable sensitive selective medium, although the differential Coomassie Brilliant Blue agar medium (Markwardt et al. 1989) may prove useful.

The occurrence of clinical disease is known to follow generally the seasonal temperature patterns. At water temperatures of 20^{o}C, susceptible fishes can develop furunculosis within 4 to 12 days after viable bacteria are released into their water supply. At temperatures below about 13^{o}C, chronic infections are more likely to develop, with incubation periods up to several weeks. At temperatures below 8 to 9^{o}C, overt disease signs may never develop in infected fish. Adverse conditions such as high water temperatures (within the limits tolerated by the bacterium) and low dissolved oxygen may precipitate clinical disease. There is some evidence that the organism can be transmitted in seawater.

D. Disease Signs

Typical furunculosis in salmonids is caused by *Aeromonas salmonicida* var. *salmonicida* and may occur in one of several forms:

1. **Peracute**
   Noted especially in fingerlings; the fish usually appear dark and die readily. Internally, the gross pathological changes resemble the acute disease.

2. **Acute** (not size-specific)
   Generally some indication of disease is noted 2 to 3 days before mortality (fish darken and go off feed). Internally, the viscera are hemorrhagic, the kidney tissue is very soft, the spleen is enlarged, and the liver is pale or mottled with petechiae.

3. **Subacute**
   The onset of mortality is more gradual. Internal lesions are present but the fish also commonly have skin lesions.

4. **Chronic**
   Similar to subacute, but is distinguished by evidence of healing around lesions.

5. **Latent**
   No mortality or clinical signs associated with *Aeromonas salmonicida* infections are evident.

Figure 1. Skin lesions typical of chronic furunculosis.

Histopathological examination of fish with acute infections often reveals foci of bacteria in the heart, kidneys, and spleen, and in the vasculature of other organs. Hematopoietic tissue necrosis, focal hepatic necrosis, and degeneration of myocardial and renal tubular tissues are often observed. In chronic disease, the heart and spleen are the organs most consistently affected. In fact, the presence of large colonies of small rod-shaped bacteria within the myocardial trabeculae is considered almost pathognomonic for furunculosis in salmonids in fresh water. The “furuncles” occurring in the skeletal musculature of some chronically diseased fish consist of necrotic tissue, tissue fluid exudate, and some macrophages. These lesions differ from true furuncles of homoiothermic vertebrates, which characteristically contain numerous polymorphonuclear leukocytes in addition to the necrotic debris and tissue fluid.

E. Disease Diagnostic Procedures

Diagnosis is based on the observation of clinical signs characteristic of the disease, and isolation of the causative organism. Primary isolation is best made from the kidney either on TSA or BHIA at 20 to 25^{o}C for 24 to 48 hours. There is some indication that certain strains of *Aeromonas salmonicida* cannot be readily grown on TSA.

1. Presumptive Diagnosis

When grown on the above media, the organism is a very short (1-2 x 0.8 μm), nonmotile, gram-negative rod that is oxidase positive, glucose positive, and gelatinase positive. Most strains produce a brown diffusing pigment. An oxidase-negative isolate has been described (Chapman et al. 1991).

Several immunological techniques have been used for the rapid diagnosis of furunculosis directly in the tissues of clinically diseased fish. These techniques include latex bead agglutination, staphylococcal coagglutination and the enzyme- linked immunosorbent assay (ELISA, Austin et al. 1986).

2. Confirmatory Diagnosis

Aeromonas salmonicida isolated in culture can be identified rapidly by serological procedures such as the FAT or agglutination tests (microtiter or slide). Care must be exercised in the use of agglutination tests, because many strains of *Aeromonas salmonicida* autoagglutinate in saline. Modified agglutination tests, such as latex bead agglutination (McCarthy 1975b) or staphylococcal coagglutination (Kimura and Yoshimizu 1983, 1984), have been used to avoid the problem of autoagglutination. The organism can also be identified by extensive phenotypic identification. In some cases this may be desirable because several subspecies of *Aeromonas salmonicida* are described in the current Bergey's Manual of Systematic Bacteriology (Holt et al. 1984).

F. Procedures for Detecting Subclinical Infections

Detection of latent furunculosis is significantly enhanced by the FAT and culture of intestinal material. The kidney should be used as a second organ for culture.

G. Procedures for Determining Prior Exposure to the Etiological Agent

No reliable methods are available at present.

H. Procedures for Transportation and Storage of Samples to Ensure Maximum Viability and Survival of the Etiological Agent

See Section 1, 1.1.1 General Procedures for Bacteriology.

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