ECVP/ESVP Summer School in Veterinary Pathology

Summer School 2014 – Mock Exam

CASE 4 Fish, gills amebiasis

Histologic Description	*Points*
Style	0,5
Gills	1
Approximately 60% (0,5) of gills are characterized by inflammation that variably expands (0,5) the subepithelial tissue of primary (0,5) and secondary lamellae (0,5)	2
Primary and secondary lamellar goblet cell hyperplasia	1
Primary lamellar lamina propria expanded by	0
Prevalence of Lymphocytes (0,5) and Plasma cells (0,5)	1
Reactive macrophages	0,5
Secondary lamellae expanded and fused	0,5
Secondary lamellar epithelial hyperplasia (only 0,5)/ Pilar cell hyperplasia (1 point)	1
Secondary lamellar lamina propria expanded by	0
Prevalence of macrophages	0,5
Lymphocytes and plasmacells	0,5
Lesser numbers of large cells with abundant intensely eosinophilic granular cytoplasm (mast cells)	1
Occasional neutrophils	1
Hyperhaemia	0,5
Myriads of protozoa on the surface between secondary lamellae	0,5
Protozoa description:	0
Round, 10 to 20 micron (0.5) oval to pear shaped (0,5) trophozoites (0,5)	1,5
Abundant granular lightly eosinophilic cytoplasm	0,5
5-7 micron round hyperchromatic cetral nucleus	0,5
Protozoa interpretation as Ameba	1
Morphological diagnosis: Moderate (0,5), chronic, diffuse (0,5), lymphoplasmacytic (0,5) and granulomatous or histiocytic (0,5) branchitis (1) with itrnalesional protozoa	3
Etiological diagnosis Branchial amebiasis (amebic branchitis)	2
	20

HD Approximately 80% of the gills are affected and severely expanded by diffuse inflammation involving prevalently primary lamellae with shortening and fusion of secondary lamellae. Within the surface among secondary lamellae there are multiple round to pear shaped, 10 to 20 micron protozoal (trophozoites) organisms characterized by a thin membrane, granular lightly eosinophilic cytoplasm and a 5-7 micron round hyperchromatic central nucleus (Amoeba). Secodanry almellae are shortened, fused and often characterized by Pilar cells and globlet cell hyperplasia. At the base of secondary lamellae and in primary lamellae the lamina propria is severely expanded by a prevalence of lymphocytes, plasmacells dn reactive macrophages associated with lesser numbers of heterophils.

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Causative agent *Neoparamoeba perurans*,

COMMENT

Amoebic gill disease (AGD) is a potentially fatal disease of some marine fish. It is caused by *Neoparamoeba perurans*, the most important amoeba in cultured fish. It primarily affects farm raised fish of the Salmonidae family, most notably affecting the Tasmanian Atlantic Salmon (Salmo salar) industry, costing the A$20 million a year in treatments and lost productivity. Turbot, bass, bream, sea urchins and crabs have also been infected. The disease has also been reported affecting the commercial salmon fisheries of the United States, Australia, New Zealand, France, Spain, Ireland and Chile.http://en.wikipedia.org/wiki/Amoebic_gill_disease - cite_note-2 It was first diagnosed in the summer of 1984/1985 in populations of Atlantic Salmon off the east coast of Tasmania and was found to be caused by the *Neoparamoeba perurans*

REFERENCES AND ANATOMY

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Clinical Communication

Nodular gill disease causing proliferative branchitis and mortality in Chinook salmon (*Oncorhynchus tshawytscha*)

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Abstract

CASE HISTORY: A period of escalating mortality occurred among Chinook salmon (1–5 g) held in freshwater raceways at a commercial culture facility. The mortality rate peaked at 1.5% of the population per day, water temperature was 9–10°C, and water quality had recently deteriorated due to an influx of suspended solids. The affected fish did not respond to several chloramine-T bath treatments (10 ppm for 1 hour).

CLINICAL FINDINGS: The dead fish did not exhibit flared opercula typical of infection with bacterial gill disease (BGD) nor did the remaining fish show obvious signs of respiratory distress. Clinical lesions were limited to the gills of the fish, and were consistent with a proliferative branchitis, including substantial hyperplasia and fusion of lamellae.

DIAGNOSIS: Numerous angular amoebic organisms (approximately 10 x 8 µm), with a central, large vesicular nucleus and faintly acidophilic cytoplasm, visible on H&E-stained sections, were located on the hyperplastic epithelia of the lamellae, filaments, and remaining lining of the branchial cavity. Morphologically, the amoebae were similar to representatives of the genus *Cochliopodia* spp. described from North American salmonids,

Introduction

'Proliferative gill disease' is a loose term that has historically been used to describe a characteristic group of lesions in which branchial epithelial hyperplasia, and hypertrophy and fusion of lamellae dominate. This term has not necessarily been associated with a particular aetiology; in fact, numerous agents are capable of producing this range of lesions, including viruses, bacteria, and protozoan and metazoan parasites (Roubal *et al.* 1989; Pote *et al.* 2000; Nylund *et al.* 2008). For the purposes of the study presented here the more correct term 'proliferative branchitis' will be used to describe these lesions. A range of fish species in both salt and fresh water are affected by this condition, and these lesions can be economically important to aquaculture because of the associated morbidity and mortality.

In sea-cage aquaculture of salmonids in Australia, the most economically significant proliferative branchitis is amoebic gill disease (AGD), caused by infections with the amphizoic amoeba *Neoparamoeba perurans* (Young *et al.* 2007). This disease is known to affect a wide range of sea-farmed fish from several countries, including New Zealand (Munday *et al.* 2001; Young *et al.* 2008). Although present in New Zealand, AGD is not considered a

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cant mortality (Mun-

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Effects of gill abrasion and experimental infection with *Tenacibaculum maritimum* on the respiratory physiology of Atlantic salmon *Salmo salar* affected by amoebic gill disease

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ABSTRACT: The effects of gill abrasion and experimental infection with *Tenacibaculum maritimum* were assessed in Atlantic salmon *Salmo salar* with underlying amoebic gill disease. The respiratory and acid-base parameters arterial oxygen tension (P_aO_2), arterial whole blood oxygen content (C_aO_2), arterial pH (pH_a), haematocrit and haemoglobin concentrations were measured at intervals over a 48 h recovery period following surgical cannulation of the dorsal aorta. Mortality rates over the recovery period were variable, with gill abrasion and inoculation with *T. maritimum* causing the highest initial mortality rate and unabraded, uninoculated controls showing the lowest overall mortality rate. Fish with abraded gills tended to show reduced P_aO_2 and lower C_aO_2 compared with unabraded fish. Infection with *T. maritimum* had no effect on P_aO_2 or C_aO_2. All fish showed an initial alkalosis at 24 h post-surgery/inoculation which was more pronounced in fish inoculated with *T. maritimum*. There were no significant effects of gill abrasion or infection upon the ratio of oxygen specifically bound to haemoglobin or mean cellular haemoglobin concentration. Histologically, 48 h following surgery, abraded gills showed multifocal hyperplastic lesions with pronounced branchial congestion and telangiectasis, and those inoculated with *T. maritimum* exhibited focal areas of branchial necrosis and erosion associated with filamentous bacterial mats. All fish examined showed signs of amoebic gill disease with multifocal hyperplastic and spongious lesions with parasome-containing amoeba

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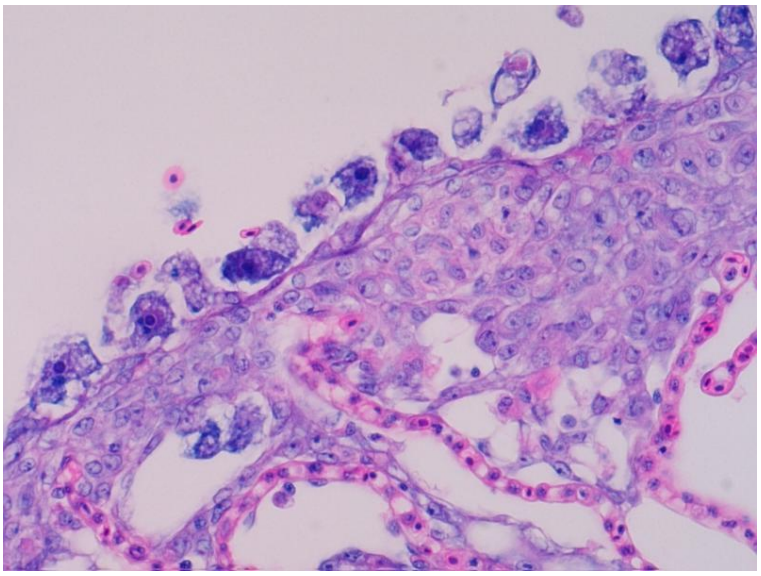
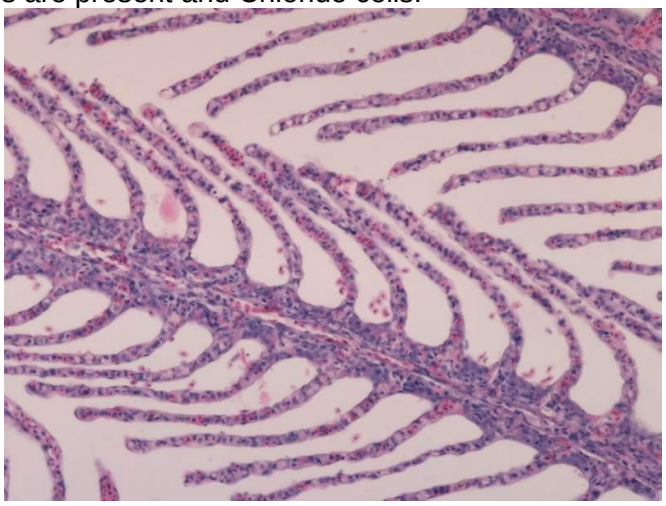


Primary lamellae originate perpendicularly from the emibranchial tissue. Primary lamellae represent the base and the origin of secondary lamellae.
The respiratory epithelium is constituted by flattened epithelial cells and by vessels composed of pilar cells (modified contractile endothelial cells). At the base of secondary lamellae mucous cells are present and Chloride cells.



Secondary lamellae

- A single or double layer of epithelial cells= Pillar cell
- An interstitial space
- Basement membrane
- Marginal channel
- Fingerprint-like microridges of lamellar epithleium

Base of secondary lamellae

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Goblet cell
Chloride cell
Progenitor cell
Afferent and efferent filamental arterioles
Filamental cartilage/ Negative charge of mucus sialic acid group/ Lymphocytes/

Macrophages
Neuroepithelial cell
Rodlet cell/ X-cell (large ovoid pale staining),