ICES Identification Leaflets for Diseases and Parasites of Fish and Shellfish

Leaflet No. 59

Piscine myocarditis (cardiomyopathy syndrome)

David Bruno

ICES International Council for the Exploration of the Sea
CIEM Conseil International pour l'Exploration de la Mer

International Council for the Exploration of the Sea
Conseil International pour l'Exploration de la Mer

H.C. Andersens Boulevard 44–46
DK-1553 Copenhagen V
Denmark
Telephone (+45) 33 38 67 00
Telefax (+45) 33 93 42 15
www.ices.dk
info@ices.dk

Recommended format for purposes of citation:

ICES. 2013. Piscine myocarditis (cardiomyopathy syndrome). ICES Identification Leaflets for Diseases and Parasites of Fish and Shellfish. Leaflet No. 59. 4 pp.

Series Editor: Stephen Feist. Prepared under the auspices of the ICES Working Group on Pathology and Diseases of Marine Organisms.

ISBN 978-87-7482-133-5

ISSN 0109–2510

Piscine myocarditis (cardiomyopathy syndrome)

David Bruno

Piscine myocarditis (previously known as cardiomyopathy syndrome (CMS)) is an economically important disease of Atlantic salmon, *Salmo salar* in seawater. Characteristic heart lesions primarily involving the myocardium are reported in natural outbreaks with associated mortality. This disease should be regarded as chronic which is associated with a necrotizing severe myocarditis involving the atrium and the spongious region of the heart ventricle and primarily observed in adult or maturing fish after 12 to 18 months in seawater.

Susceptible species

Piscine myocarditis was originally described from farmed Atlantic salmon, *S. salar* in 1985 but has subsequently been recorded in wild Atlantic salmon along the Norwegian coast. It is suggested that cardiac function would be impaired and therefore could compromise such migratory fish.

Disease name

The original name 'cardiomyopathy syndrome' was used to refer to a condition where the aetiology was unknown. However, successful transmission of the condition using tissue homogenates from Norway and Scotland from mild or severely affected fish with the development of typical lesions; together with the identification of a causative virus resulted in the name piscine myocarditis to become adopted.

Aetiological agent

Piscine myocarditis is caused by a double-stranded RNA virus named piscine myocarditis virus (PMCV) with structural similarities to the *Totiviridae* family.

Geographical distribution

Since the first diagnosis of Piscine myocarditis in Norway the disease has also been diagnosed in sea farms rearing Atlantic salmon in Scotland, the Faroe Islands, Denmark and Canada.

Associated environmental conditions

Studies suggest that fish are infected through horizontal transfer of virus as salmon injected intramuscularly transfer the virus through water to cohabitating fish which then develop heart changes typical of Piscine myocarditis.

Significance

Mortality can occur without prior clinical signs and typically just prior to slaughter and consequently with significant economic losses. Affected groups show significantly increased mortality, causing a direct annual loss for the industry from data published in 2003 of €4.5 to 8.8 million on fish farms. Evidence suggests that Piscine myocarditis is increasing in Norway but stable in Scotland.

Gross clinical signs

Fish may go off feed and swim sluggishly with little external signs or show skin haemorrhage and oedema, raised scales and exophthalmia.

Internally a fibrinous peritonitis, ascites, and blood clots surrounding the atrium and sinus venosus or the dorsocranial surface of the liver may be recorded.

Control measures and legislation

There is no prophylactic measure but outbreaks maybe helped through coordinated eradication strategies. This disease is not reportable under current statutory regulations.

Diagnostic methods

Histopathology is used extensively to diagnosis Piscine myocarditis and is based on observation of cardiac lesions, characterized by severe inflammation and necrosis of the spongy myocardium of the atrium and ventricle. Both the atrium myocardium and ventricle spongiosum show epicarditis, muscular degeneration, proliferation of the endocardial cells with mononuclear cell infiltration and lymphocytes subendocardially, and a moderate to marked thickening of myofibres with loss of striation within the sarcolemma.

Focal necrosis in the hepatic parenchyma may also occur with hyalinization and occasional nuclear enlargement, extending from the outer compact layer of the myocardium into the spongy layer. In some sections of liver, a mild, fibrosis of the central vein can be recorded.

At 6–7 weeks post challenge viral genome levels peak as shown by real-time reverse transcription (RT)-PCR. Detection of the virus genome can be achieved through in situ hybridization in degenerate cardiomyocytes from clinical cases. Viral levels reach a plateau between 6 and 10 weeks which is consistent with the presence of histopathological changes typical of Piscine myocarditis.

Supplementary literature

Brun., E., Poppe, T., Skrudland, A., and Jarp, J. 2003. Cardiomyopathy syndrome in farmed Atlantic salmon *Salmo salar*: occurrence and direct financial losses for Norwegian aquaculture. Diseases of Aquatic Organisms, 56: 241–247.

Bruno, D. W., and Nogueram P. A. 2009 Experimental transmission of cardiomyopathy syndrome (CMS) in Atlantic salmon *Salmo salar.* Diseases of Aquatic Organisms, 87: 235–242.

Haugland, O., Mikalsen, A. B., Nilsen, P., Lindmo, K., Thu, B. J., Eliassen, T. M., Roos, N., Rode, M., and Evensen, O. 2011. Cardiomyopathy Syndrome of Atlantic Salmon (*Salmo salar* L.) is caused by a double-stranded RNA virus of the Totiviridae family. Journal of Virology, Vol. 85: no 11: 5275–5286.

Løvoll, M., Wiik-Nielsen, J., Grove, S., Wiik-Nielsen, C. R., Kristoffersen, A. B., Faller, R., Poppe, T., Jung, J., Pedamallu, C. S., Nederbragt, A. J., Meyerson, M., Rimstad, E., and Tengs, T. 2010. A novel totivirus and piscine reovirus (PRV) in Atlantic salmon (*Salmo salar*) with cardiomyopathy syndrome (CMS). Virology Journal, 7: 309–315.

Poppe, T. T., and Seierstad, S. L. 2003. First description of Cardiomyopathy syndrome (CMS)-related lesions in wild Atlantic salmon *Salmo salar* in Norway. Diseases of Aquatic Organisms, 56: 87–88.

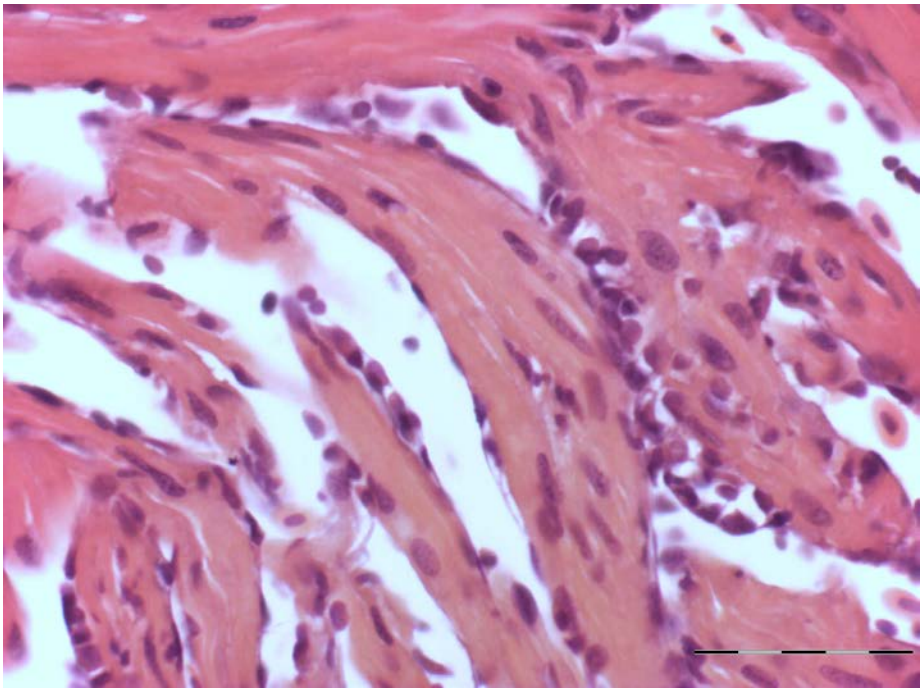

Figure 1. Piscine myocarditis in Atlantic salmon showing proliferation and hypertrophy of the endocardial cells with subendocardial infiltration. H&E, bar scale = 50µm.

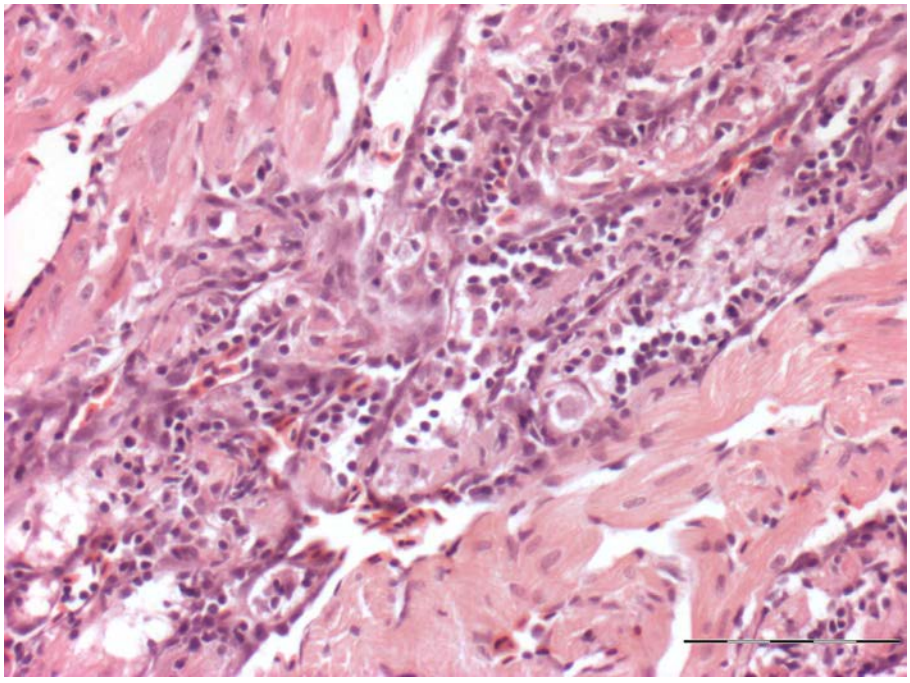

Figure 2. Piscine myocarditis in Atlantic salmon. Widespread hypertrophy adjacent to areas of infiltration, degeneration and necrosis of the myocardium. H&E, bar scale = 100µm.

Author Contact Information

David Bruno
Marine Laboratory
PO Box 101
375 Victoria Road
Aberdeen AB11 9DB
UK
David.Bruno@scotland.gsi.gov.uk