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Pathology associated with retained fishing hooks in blue sharks, *Prionace glauca* (L.), with implications for their conservation

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Abstract

Fishing hooks retained from previous capture events were found in 6 of 211 blue sharks, *Prionace glauca* (L.), landed in the summers of 1999 and 2000 by recreational fishermen off Long Island (New York, USA). The hooks were embedded within the distal oesophagus ($n = 3$), or perforated the gastric wall ($n = 3$) and lacerated the liver ($n = 2$). The hooks were surrounded by excessive fibronecrotic tissue which ablated the normal anatomical structures and in the three sharks with oesophageal hooks caused partial luminal obstruction. Accompanying lesions included oesophagitis, gastritis, hepatitis and proliferative peritonitis. *Aeromonas* sp. and *Vibrio* sp. were isolated from the peritoneal fluid of one shark with peritonitis and intralesional bacteria were seen on histological examination in all sharks. This is the first report of the prevalence and pathology of retained fishing hooks in a large number of wild-caught sharks.

Keywords: NW Atlantic, pathology, *Prionace glauca*.

Introduction

The blue shark, *Prionace glauca* (L.), is a large cosmopolitan carcharhinid species (Compagno 1984) which is one of the most far ranging and numerous of the large sharks (Lineaweaver & Backus 1970). Although the blue shark is subjected to extensive commercial and recreational fisheries in the USA, most are released alive. In the western North Atlantic, the blue shark is caught on hooks incidental to commercial longline fisheries, but the low marketability of its flesh coupled with a federally imposed blue shark quota (NMFS 1999) results in its predominant release. From 1993 to 1995, the USA pelagic longline catch in the western North Atlantic ranged from 50 to over 120 thousand blue sharks annually, with an average of 81% released alive (Cramer 1997). The blue shark is also the primary pelagic shark species caught by recreational fishermen in the north-east USA from New Jersey to Maine. Although accurate estimates of total blue shark catch and release by the recreational sector are lacking, there is evidence from shark fishing tournament data that these numbers are high. In Massachusetts alone, a single 2-day shark fishing tournament in 1999 resulted in the catch of over 2000 blue sharks and 99% were released alive (G. Skomal, unpublished data).

The high number of blue shark releases results in an unknown number of fish that do not survive as a result of physiological and physical trauma associated with hooking. While it has been shown that the former may not significantly affect post-release survivorship in this species (Skomal & Chase, in press), physical damage from the hook can have lethal consequences (Prince, Ortiz & Venizelos, in press; Skomal, Chase & Prince, in press). This is the first report of documented prevalence of retained fishing hooks with description of associated lesions in a large group of blue sharks.

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Materials and methods

Two hundred and eleven adult male blue sharks were landed during 4 days in July 1999 (81 sharks) and June/July 2000 (130 sharks) by recreational anglers in the western North Atlantic off Montauk, New York. The sharks were necropsied immediately after landing. The date of capture, sex, body mass (BM), fork length (FL), and macroscopic lesions from each shark were recorded. Samples of abnormal organs including the oesophagus, stomach, liver, abdominal wall and testes were preserved in 10% buffered elasmobranch formalin (Prieur, Fenstermacher & Guarino 1976). In the laboratory these samples were processed routinely for bright-field microscopy, embedded in paraffin, sectioned at 4–5 μm, stained with haematoxylin and eosin (H&E), and permanently mounted on glass slides using standard histological techniques. In addition, selected sections were stained with Prussian blue and Brown and Hopps methods (Sheehan & Hrapchak 1980). Samples from the peritoneal fluid from shark no. 6 were collected aseptically for bacterial cultures. These were carried out by streaking the samples onto two selective solid media plates: TSA with 50% filtered ocean water (Micro Technologies, Inc., Richmond, ME, USA), and salt enriched blood agar (Binax-nel, Waterville, ME, USA). Plates were packaged on ice and shipped overnight to Micro Technologies, Inc. Laboratories, Richmond, ME, USA for bacterial isolation.

Results

Six (sharks nos 77, 15, 1, 11, 14, 6) of 211 sharks examined had fishing hooks retained from previous fishing events. The date of capture, total number of sharks examined that day, BM and FL of these six sharks are given in Table 1. All hooks were embedded in yellow-white, firm to semisoft, moderately well delineated fibronecrotic tissue ranging in size from $4 \times 3 \times 1$ to $6 \times 10 \times 12$ cm. Except in shark no. 11, the hooks were corroded and surrounded by a narrow rim (less than 0.1 cm thick) of discoloured, reddish-brown tissue. Three of the hooks were embedded in the distal oesophageal wall (sharks nos 15, 11, 14). In the remaining three sharks (nos 77, 1, 6) the hooks perforated the gastric wall. In addition, in sharks nos 1 and 6, the hooks lacerated the liver.

The macroscopic lesions in all sharks with oesophageal hooks were similar and included mucosal ulceration, and severe mural inflammation and fibrosis ablating the normal architecture and partially obstructing the oesophageal lumen. In all sharks with gastric perforation, there was transmural fibrinonecrotic gastritis and proliferative peritonitis manifested as a diffuse thickening and granularity of the gastric and abdominal serosa. The surface of the hepatic tears in sharks nos 1 and 6 was covered by a hyperplastic serosa with long (0.1–0.3 cm) villus-like projections. In addition, shark no. 6 had a turbid, brownish peritoneal effusion, and a discrete red-brown, sessile, exophytic growth on the otherwise normal appearing serosa of the left testis.

Histological lesions in all sharks with oesophageal hooks were similar. They consisted of mucosal ulceration and transmural fibrosing and necrotizing oesophagitis (Fig. 1). The hooks were surrounded by necrotic amorphous and fibrous material containing numerous colonies of bacteria mixed with extracellular granules staining with Prussian blue (presumably iron from the corroding fishing hooks) (Fig. 2). The normal oesophageal lamina propria which consists of loose, sparsely cellular connective tissue was effaced and expanded by granulomatous inflammation with numerous coalescing foci of necrosis surrounded by granulation tissue and fibrosis at the margins of the lesions. Some of the necrotic foci contained bacterial colonies

Table 1 Field data for the six male blue sharks examined in the study of pathology associated with retained fishing hooks

Total no. of sharks examined	Shark no.	Date of collection	Body mass (kg)	Calculated[a] body mass (kg)	Fork length (cm)
81	77	July 1999	73.1	79.3	230.2
90	15	June 2000	100.8	97.7	246
	1		89.4	76.5	227.5
27	11	June 2000	61.7	68.8	220
	14		98.5	96.4	245
13	6	July 2000	133.0	129.2	269

[a] Calculated from normal range (Kohler *et al.* 1996).

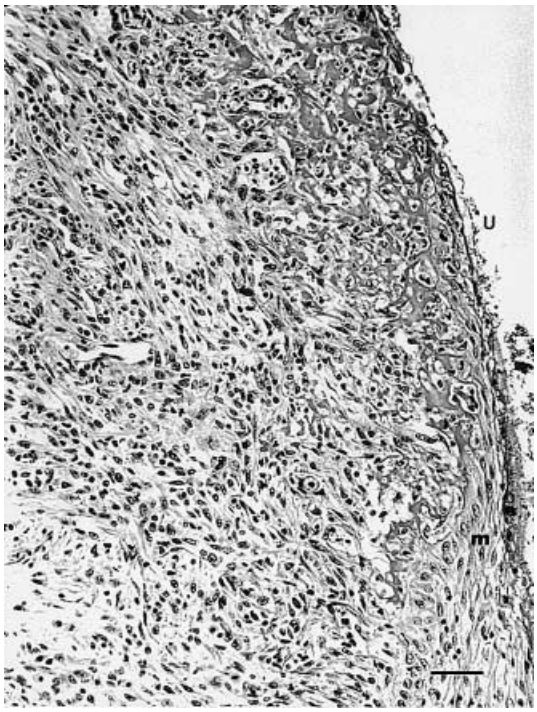

Figure 1 Oesophageal mucosa next to entry point of fishing hook in shark no. 14; diffuse mucosal ulceration (U) with fragments of mucosal epithelium (m) surrounded by fibroproliferative and necrotic tissue. The lamina propria is replaced by granulation tissue and fibrosis (H&E, bar = 40 μm).

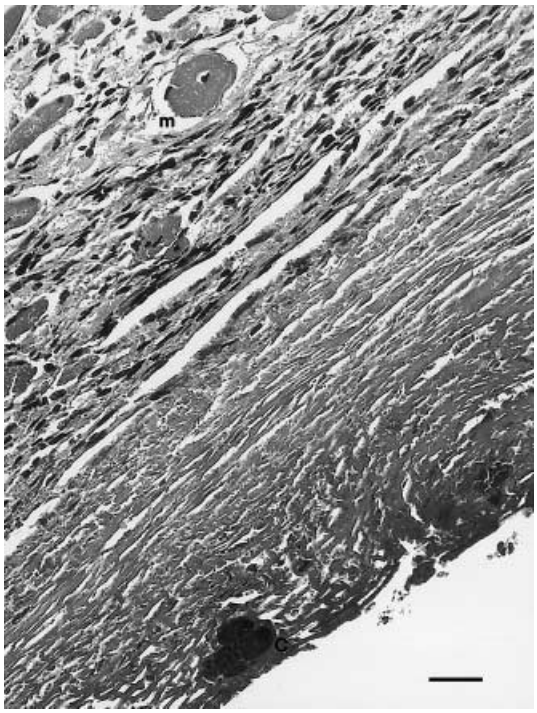

Figure 2 Tissues of the tunnel surrounding the fishing hook embedded within the oesophageal wall in shark no. 11; amorphous and fibrous necrotic tissue with a focus of coccoid bacteria (C); necrotic muscle fibres (m) separated by oedema and fibrosis (H&E, bar = 20 μm).

surrounded by Splendore-Hoeppli material (Fig. 3). Occasional discrete granulomas were centred on macrophages with intracytoplasmic iron and extracellular iron-compatible foreign bodies (Fig. 4). Submucosal lymphofollicular hyperplasia was prominent.

In all sharks with gastric perforation, histological lesions consisted of transmural necrotizing and fibrosing gastritis and proliferative peritonitis. In all of these sharks, the hooks perforated the gastric wall and were lodged within tunnels lined by a narrow rim of iron-impregnated necrotic tissue. The tunnels were surrounded by histiolymphocytic granulomatous inflammation superimposed on granulation tissue. Occasional necrotic or pyogranulomatous foci with intralesional bacterial colonies were seen within the latter. A wide margin of fibrosis with minimal cellularity demarcated the inflammation from the normal gastric wall.

The proliferative peritonitis involved the gastric serosa, the gastric surface of the liver, and the parietal peritoneum adjacent to the above organs. The normal parietal and visceral peritoneum in blue sharks is a simple cuboidal to low columnar mesothelium supported by a narrow, collagenous lamina propria (unpublished data). Within the peritonitis the peritoneum formed papillomatous and branching, cauliflower-like exophytic proliferations (Figs 5–7). The mesothelium was metaplastic and hyperplastic, and changed to simple tall columnar or pseudostratified with rare foci of dysplasia (Figs 5 & 6). The latter was characterized by cellular stratification, disorganisation and loss of cellular polarity (Fig. 6). Occasional mesothelial cells contained intracytoplasmic granules compatible with iron. The lamina propria within the areas of peritonitis was expanded by subserosal fibroplasia with occasional myxomatous foci, mild perivascular plasmacytic and lymphohistiocytic infiltrates, and occasional lymphofollicular structures. Bacterial colonies were found on coelomic serosal surfaces of all sharks with gastric perforation.

The lacerated liver from shark no. 6 had histological lesions consisting of diffuse, marked pyogranulomatous hepatitis and fibrosis, increased numbers of melanophacrophages (mean = 62 per

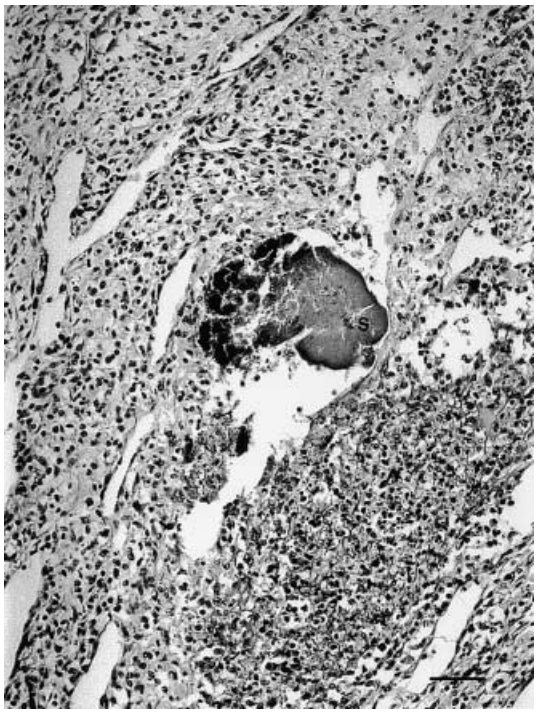

Figure 3 Oesophagitis in shark no. 14; colonies of coccoid bacteria within Splendore-Hoeppli material (S) (H&E, bar = 40 μm).

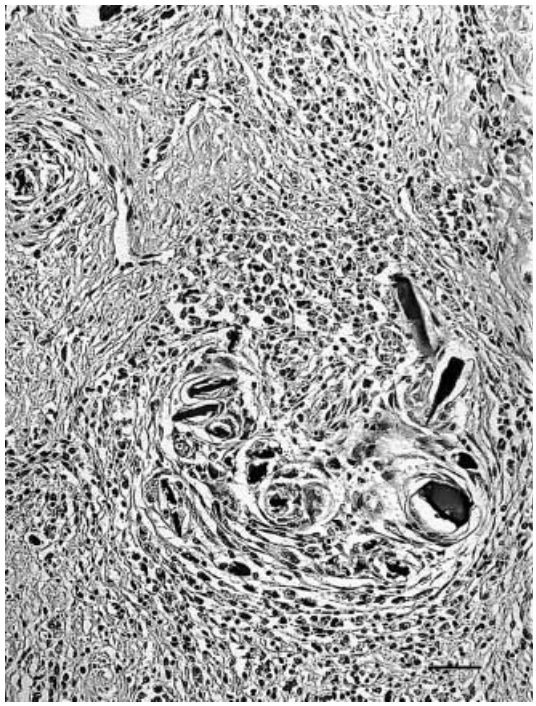

Figure 4 Oesophagitis in shark no. 11; discrete granuloma with extracellular and phagocytized (presumed) iron particles (H&E, bar = 40 μm).

high power field as compared to a mean = 12 found in four normal adult male blue sharks examined for comparison by JDB) and marked, diffuse mesothelial hypertrophy and hyperplasia (Fig. 7). In addition this shark had biliary hyperplasia, an intrahepatic cholangiocarcinoma, and biliary myxosporean organisms compatible with *Chloromyxum* sp. (Kuznetsova 1977), myxosporean cysts within the skeletal muscle of the abdominal wall and a testicular mesothelioma (data reported separately). The liver from shark no. 1 was not examined histologically.

Discussion

We found fishing hooks retained from previous fishing interactions in six (2.84%) of 211 adult male blue sharks landed by recreational fishermen in the western North Atlantic. Considering the numbers of blue sharks caught and released alive each year in the North Atlantic alone, one can estimate the potential number of sharks with retained fishing hooks to number in thousands.

Mild local lesions were described in the integument of sharks in association with another artificial penetrating device – a dart tag (Heupel & Bennett 1997; Heupel, Simpfendorfer & Bennett 1998) and short-term mortality in association with retention gear was reported from an experimental study in teleost fish (Cooke & Hogle 2000). Herein, we report lesions caused by retained fishing hooks in wild-caught sharks. Although the time of the first hooking event is unknown, the granulation tissue and fibrosis found in the lesions indicate a chronic condition. The lesions produced by the hooks were severe and included oesophageal perforation and partial obstruction, gastric perforation, necrotizing and proliferative gastritis and peritonitis, hepatic laceration with hepatitis and secondary bacterial infection. This peritonitis resembles that of traumatic reticuloperitonitis in cattle. Traumatic reticuloperitonitis associated with sharp foreign objects penetrating the wall of the stomach is very common in adult bovines (Barker, Van Dreumel & Palmer 1993), and it is usually complicated by infection with putrefactive environmental micro-organisms. The mechanical injury caused by the fishing hooks was similarly complicated by bacterial colonization of the oesophageal tissues and the peritoneum, as well as by bacterial oesophagitis and gastritis. The

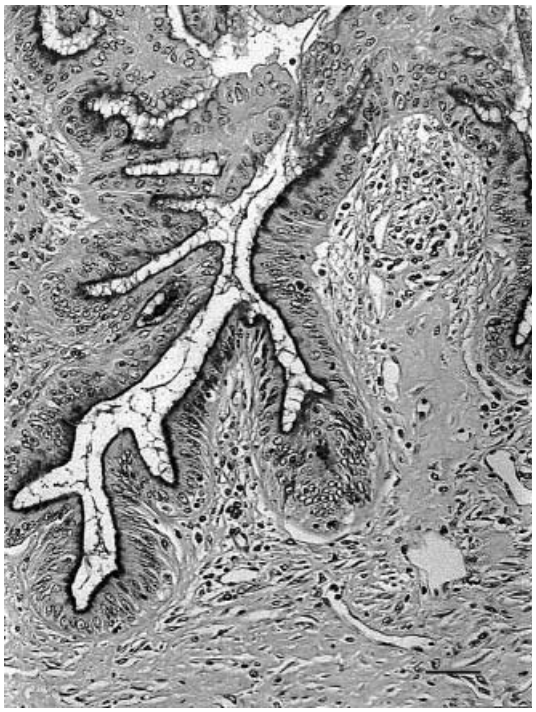

Figure 5 Gastric serosa in shark no. 1; cauliflower-like proliferation of a hyperplastic and metaplastic mesothelium (columnar or pseudostratified) supported by fibrovascular stroma with plasmalymphocytic infiltrates (H&E, bar = 40 μm).

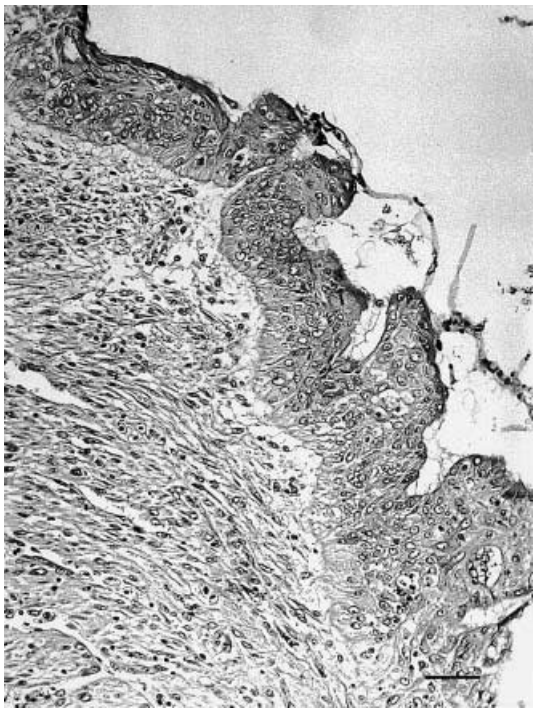

Figure 6 Gastritis with subserosal fibroplasia in shark no. 1; thickened and disorganized serosal mesothelium (H&E, bar = 40 μm).

micro-organisms were most probably introduced with the hook, but one cannot exclude their origin from shark tissues. This is because bacteria can be isolated from apparently healthy sharks (Grimes 1990; J.D. Borucinska, unpublished data). Bacterial cultures were taken only from shark no. 6 with peritonitis, and yielded *Aeromonas* sp. and *Vibrio* sp. These two bacteria are commonly reported from diseased teleosts (Noga 2000). In sharks, *Aeromonas salmonicida* (Briones, Fernandez, Blanco, Ramiro, de Vicente, Mendez Garcia & Goyache 1998) and various *Vibrio* spp. (Grimes, Stemmler, Hada, May, Maneval, Hetrick, Jones, Stoskopf & Colwell 1984; Grimes, Gruber & May 1985; Grimes, Burgess, Crunkleton, Brayton & Colwell 1989) have been associated with septicaemias and *Vibrio carchariae* has been reported as the causative agent of meningitis in brown sharks, *Carcharhinus plumbeus* (Nardo) (Stoskopf 1993).

The mesothelioma-like peritoneal proliferations found in all examined sharks in this study possibly arose from the mechanical trauma and persistent irritation by a foreign body (corroding hooks), and from bacterial infection leading to severe, chronic peritonitis. The mesothelium of sharks is very prone to hyperplastic and hypertrophic changes in response to injury (J.D. Borucinska, personal observation), and has phagocytic properties. Iron particles in hyperplastic mesothelial cells were seen in sharks in this report. The role of chronic foreign body induced serositis progressing to neoplasia has been documented in man and animals exposed to asbestos (Barker 1993) and other inorganic compounds (Evans 1966). The possible progression of proliferative peritonitis to tumours of the peritoneum (mesotheliomas) in sharks with retained fishing hooks warrants further examination.

At this time, the causal relationship between the retained hook in shark no. 6 and the testicular mesothelioma and cholangiocarcinoma is unknown, and these lesions will be described in a separate report. The remaining lesions found in this shark, i.e. myxosporeans within the gall bladder and skeletal muscles were most probably incidental.

The BM of all six sharks examined was within the normal range reported for male blue sharks (Kohler, Casey & Turner 1996), which indicates that none of these sharks suffered from debilitating disease at the time of death. However, active

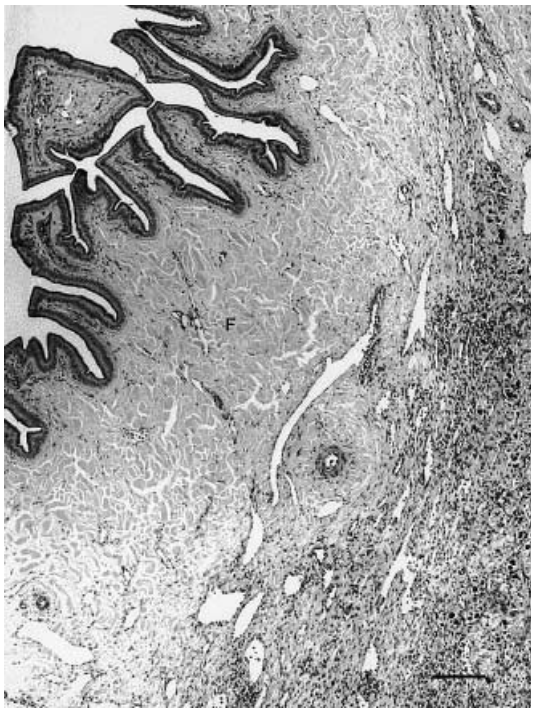

Figure 7 Necrotizing granulomatous hepatitis in shark no. 6; subcapsular fibrosis (F) and papillomatous proliferation of the capsular mesothelium (PAS, bar = 100 μm).

inflammation associated with the hooks and intralesional bacteria were present in each of these sharks, and thus progression of the disease leading to debilitation or death cannot be ruled out. We previously described a case of severe gastritis, peritonitis and pericarditis with cachexia associated with a retained fishing hook in a blue shark (Borucinska, Martin & Skomal 2001). Based on these data, the role of retained fishing hooks in the morbidity and mortality of large pelagic sharks should be examined further. Such studies are warranted because high mortality associated with retained fishing hooks could undermine the efficacy of catch-and-release in shark management and conservation.

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