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A SYNDROME OF ISCHEMIC LEG NECROSIS IN NORTHERN GANNETS (*MORUS BASSANUS*)

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ABSTRACT: The Northern Gannet (*Morus bassanus*) is a large marine bird whose whole North American population breeds in waters of eastern Canada. Opportunities to identify causes of morbidity and mortality in recently hatched birds of this species are therefore limited to this region of North America. During the three decades since 1990 of wildlife health surveillance at the Atlantic regional center of the Canadian Wildlife Health Cooperative, what appears to be a previously undescribed syndrome of ischemic leg necrosis affecting mainly hatch-year Northern Gannets has emerged, which may relate to some unique aspects of the life history of these birds. This syndrome, observed in 14 birds, is characterized by severe necrosis and fibrinopurulent inflammation of soft tissues of the feet extending along the whole tarsometatarsus. An infectious cause is proposed to explain the pattern of lesions observed in these birds, possibly favored by a specialized and rich vascular system in their legs and feet. An acute or subacute cardiomyopathy, thought to be secondary to the severe leg lesions, was also observed microscopically in six of these birds.

Key words: Cardiomyopathy, distal extremities, ischemic necrosis, *Morus bassanus*, Northern Gannet, *Salmonella* spp.

INTRODUCTION

The Northern Gannet (*Morus bassanus*) is a coastal marine bird that is rarely seen far offshore and feeds primarily by plunge diving in the first few surface meters of the water column. Its western Atlantic population overwinters in waters along the east coast of the US from Maine to Texas and breeds on islands at only six locations, all in eastern Canada (three in the Gulf of St. Lawrence, three off insular Newfoundland), being seen in Canadian waters from late March to late October (Chapdelaine 1996; Montevecchi et al. 2012; Chardine et al. 2013). Two of the breeding colonies—Bonaventure Island, 3.5 km off the southern coast of Gaspé Peninsula, Québec, and Bird Rocks, 40 km northeast of the Magdalen Islands, Québec, both in the southern Gulf of St. Lawrence—contain up to 77% of the total North American population, with a combined number of close to 90,000 breeding pairs in 2009 (Chardine et al. 2013). According to Garthe et al. (2007), the mean foraging range and total distance travelled per foraging trip by these birds from Bonaventure Island are 138 and 452 km, respectively, indicating that they can range within a wide expanse of the Gulf of St. Lawrence.

Various causes of morbidity and mortality have been reported in Northern Gannets, identified mainly through examination of injured or dead birds found opportunistically ashore. Most of these causes are human-related, including oiling and, especially, interactions with gillnet and longline fisheries (Nelson 1978; Wilhelm et al. 2009; Lucas et al. 2012; Hedd et al. 2016). Starvation is probably a common cause of natural mortality, particularly in postfledging young birds, which spend the first 2 wk at sea unable to fly (Nelson 1978). Infectious causes of mortality have not been commonly reported in this species, although Nelson (1978) mentioned salmonellosis and Spalding et al. (2002) described a single case of encephalitis caused by *Sarcocystis* sp. in an adult Northern Gannet on the east coast of Florida. We describe what appears to be a unique syndrome of ischemic necrosis in this species, involving the distal region of the legs and primarily affecting hatch-year (HY) birds.

MATERIALS AND METHODS

Since 1990, the Canadian Wildlife Health Cooperative (CWHC) has been conducting health surveillance in free-living wildlife (Leighton et al. 1997). As part of this mandate, fresh or frozen carcasses of Northern Gannets found sick, injured, or dead along the shores of the three Canadian Maritime provinces (New Brunswick, Nova Scotia, Prince Edward Island) were submitted for postmortem examination by conservation officers, park wardens, biologists, wildlife rehabilitation center staff, and the public to the Atlantic regional center of the CWHC, based at the Atlantic Veterinary College, University of Prince Edward Island. The following information was collected from each carcass: collection site, month and year when the bird died or was euthanased or when the carcass was found, history of the circumstances under which the bird was found, age class (HY; immature, <5 years old; adult) by plumage (Harrison 1983; Sibley 2014), degree of development of the gonad(s) and presence or absence and relative size of a cloacal bursa, sex, subjective assessment of nutritional condition (good, moderate, poor) from relative size of pectoralis muscles and relative amount of fat in subcutis and coelomic cavity (Daoust et al. 2021), and final diagnoses from gross and microscopic examination to the extent that the state of preservation of the carcass allowed.

For microscopic examination, tissue samples were fixed in 10% buffered formalin, dehydrated in graded alcohol and xylene, and embedded in paraffin; 5-μm-thick sections were stained with H&E. Samples submitted for bacteriologic examination were cultured at 35 C in 5% CO_2 on MacConkey agar and on Columbia agar with 5% sheep blood. Bacterial isolates were identified with the Bruker microflex LT matrix-assisted laser desorption/ionization time-of-flight mass spectrometry system (Bruker Daltonik GmbH, Bremen, Germany) with the MBT Compass version 4.179 reference library. Presumptive isolates of *Salmonella* sp. were sent to the World Organisation for Animal Health Salmonella Reference Laboratory (National Microbiology Laboratory, Guelph, Ontario, Canada) for confirmation and serotyping by slide agglutination (White-Kauffmann-Le Minor Scheme; Grimont and Weill 2007).

RESULTS

Between April 1990 and October 2017, a total of 314 carcasses of Northern Gannets (33 HY, 39 immature, 238 adult, four of undetermined age; 144 female, 140 male, 30 of undetermined sex) were examined. Several of these carcasses, often parts of small-scale mortality events associated with fisheries interactions, were too scavenged for a complete postmortem examination but could still provide some information, such as age, sex, and nutritional condition. A syndrome became gradually apparent in this species, consisting of moderate to severe inflammation and necrosis of one or both legs from the tarsometatarsus distally. Between June 1991 and October 2016, a total of 14 birds with this putative syndrome were identified (Table 1). All were found along shores of the southern Gulf of St. Lawrence, all except one were found alive, and a majority involved HY birds in the fall, after they had fledged. Both legs were usually involved, although not necessarily with the same degree of severity. In most cases, lesions were considered subacute and generally involved birds that were still in moderate to good nutritional condition (Table 1). Macroscopic lesions consisted, in general, of accumulation of edema fluid and exudate in soft tissues along the tarsometatarsus and marked edema of the interdigital foot webs, with characteristic sloughing of the superficial epidermal layers over affected webs and loss of some toe nails. In the most severe case, all interdigital soft tissues of the left foot had sloughed off, exposing phalangeal bones (Fig. 1). Other than the leg lesions and poor nutritional condition in four birds, macroscopic pathologic processes identified included generalized petechiae and ecchymoses interpreted as terminal, multifocal to confluent subcutaneous granulomatous cellulitis involving the left flank and severe necrotizing enteritis in birds 3, 6, and 14, respectively (Table 1).

Although often hampered by autolysis and freezing artifacts, microscopic examination was performed on all 14 birds with leg lesions, confirming the presence of fibrinopurulent exudate associated with large areas of necrosis in soft tissues (including portions of epidermis) and muscles along the tarsometatarsus, these areas of necrosis being often bordered by multinucleated giant cells. Fibroblastic tissue suggestive of more chronic lesions was

identified within the exudate in a few instances. Thrombosed, inflamed, or necrotic small blood vessels could also be found within the exudate in several cases (Figs. 2, 3). In two cases, a sharp zone of demarcation between viable and necrotic tissues could be detected microscopically in soft tissues along the tarsometatarsus (Fig. 4). In the single instance in which the tarsometatarsal bone was examined microscopically, much of the marrow fat appeared necrotic and included several small aggregates of heterophils and occasional bacterial colonies; viability of the osseous tissue itself could not be evaluated properly because of autolysis. In the more severe cases, the interdigital foot webs were completely necrotic, with total loss of differential staining affinity and infiltration by variable numbers of small bacterial colonies with no associated inflammation. Lesions in foot webs in some other cases resembled those in soft tissues along the tarsometatarsus. In a few birds, the severity of lesions differed among webs of the same foot.

Microscopic lesions of cardiomyopathy that were not evident macroscopically were identified in six birds, all HY (Table 1). These lesions consisted of multifocal to confluent areas of hydropic degeneration, fragmentation, and occasional necrosis and mineralization of myocardial fibers associated with an infiltration of small numbers of macrophages. All were considered of acute to subacute duration and were of various degrees of severity, most of them mild to moderate, but in one bird marked.

Bacterial culture of soft tissues along affected tarsometatarsi was attempted in nine of the 14 birds. *Aeromonas hydrophila* was isolated in several of these birds (Table 1). This bacterium was also isolated in very small numbers from the liver, but not the kidneys, of bird 3, with generalized petechiae and ecchymoses. Bacterial culture from lesions of granulomatous cellulitis in the left flank of bird 6 yielded *Aeromonas* spp., alpha-hemolytic *Streptococcus* spp., and coagulase-negative *Staphylococcus* spp. in small numbers. Monophasic *Salmonella enterica* subsp. *enterica* serovar Typhimurium phage type 41 was isolated in small to moderate numbers from the more severely affected leg of birds 13 (antigenic formula, 4:i:-) and 14 (antigenic formula, 4,5:i:-). This same bacterium was also isolated from lesions of severe necrotizing enteritis in bird 14. Interestingly, this bird had an abundant amount of fibrinopurulent exudate in soft tissues along its tarsometatarsi, but intact foot webs.

DISCUSSION

The pattern of gross and microscopic lesions identified in birds included in this report strongly suggests various degrees of ischemic injury to the distal regions of the legs. Other than Northern Gannets, such a pattern has not been observed in any species of marine birds submitted to the Atlantic regional center of the CWHC during three decades of wildlife health surveillance. Two unique aspects of the biology of this species may be relevant to this apparent syndrome. First, these birds, both male and female, incubate their single egg by covering it with the highly vascularized webs of their feet (King and McLelland 1984; Chapdelaine 1996). This may suggest a specialized and rich vascular system in the feet and legs of these birds, possibly more susceptible to interference with blood perfusion. To our knowledge, no specific information is available on this particular aspect of the Northern Gannet's anatomy. Second, HY Northern Gannets do not fledge before mid-September or later (Lagueux 1986; Chapdelaine 1996) and spend the following 1–2 wk on water at sea, relying on their fat reserves accumulated on the nest, before they gain flight and start foraging (Nelson 1978; Lagueux 1986). Average sea surface water temperature in the Gulf of St. Lawrence in summer is around 15 C, decreasing to 12 C or less as fall progresses (Galbraith et al. 2012), which may promote vasoconstriction.

Ischemic necrosis of extremities has been described in other species, with various proposed causes. Yates et al. (1969) reported on 22 free-living Mute Swans (*Cygnus olor*) of

TABLE 1. Epidemiologic data, morphologic diagnoses, and bacteriologic results in 14 Northern Gannets (*Morus bassanus*) with ischemic leg necrosis found moribund or dead along shores of the southern Gulf of St. Lawrence, Canada, 1990–2020.

Bird no.	Month and year found	Age, sex[a]	Nutritional condition	Diagnosis	Bacterial isolate(s) from soft tissues of affected legs[b]
1	June 1991	Imm, F	Moderate	Subacute unilateral fibrinopurulent pododermatitis.	NA
2	October 1994	HY, M	Good	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and myositis and pedal necrosis.	NA
3	October 1996	HY, M	Good	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis.[c] Moderate acute cardiomyopathy. Terminal generalized petechiae and ecchymoses.	*Aeromonas hydrophila* in large numbers (also in very small numbers from liver)
4	October 1997	HY, F	Good	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis.[c] Mild acute cardiomyopathy.	*A. hydrophila* in large numbers
5	October 1999	HY, F	Good	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and myositis and pedal necrosis.[c] Moderate subacute cardiomyopathy.	NA
6	October 1999	HY, F	Moderate	Subacute unilateral (right) fibrinopurulent and necrotizing tarsometatarsal cellulitis and myositis and pedal necrosis. Multifocal to confluent subcutaneous granulomatous cellulitis (left flank).	NA (mixed flora in small numbers from left flank)
7	December 2000	HY, F	Poor	Chronic unilateral granulomatous tarsometatarsal cellulitis and multifocal to confluent pedal necrosis.	NA
8	October 2002	HY, F	Good	Subacute bilateral fibrinopurulent tarsometatarsal cellulitis and myositis and pedal necrosis.[c] Marked subacute cardiomyopathy.	*A. hydrophila* in large numbers
9	October 2003	HY, M	Good	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis. Moderate subacute cardiomyopathy.	*A. hydrophila* in large numbers
10	November 2003	HY, M	Moderate	Subacute bilateral fibrinopurulent tarsometatarsal and pedal cellulitis.[c]	Mixed flora in very small numbers
11	October 2004	HY, M	Moderate	Chronic bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and myositis and pedal necrosis. Mild acute cardiomyopathy.	*A. hydrophila* and *Shewanella putrefaciens* in moderate numbers
12	May 2005	Ad, F	Poor	Chronic bilateral fibrinopurulent tarsometatarsal cellulitis and pedal necrosis.[c]	*Enterobacter* spp. in moderate numbers

TABLE 1. Continued.

Bird no.	Month and year found	Age, sex[a]	Nutritional condition	Diagnosis	Bacterial isolate(s) from soft tissues of affected legs[b]
13	November 2016	HY, M	Poor	Subacute bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis.[c]	Monophasic *Salmonella enterica* subsp. *enterica* serovar Typhimurium phage type 41 (antigenic formula, 4:i:-) in small numbers
14	October 2016	HY, F	Poor	Subacute bilateral fibrinopurulent tarsometatarsal cellulitis.[c] Subacute necrotizing enteritis.	Monophasic *Salmonella* Typhimurium phage type 41 (antigenic formula, 4,5:i:-) in moderate numbers (also from intestine)

[a] Imm = immature <5 years old; HY = hatch-year; Ad = adult; F = female; M = male.

[b] NA = not available.

[c] Different degrees of severity of lesions between the two legs. The diagnosis provided pertains to the more severely affected leg.

undetermined age found in August and early fall with severe necrotic lesions of the feet, in a few cases extending to the tarsometatarsal joints. No cause for these lesions was suggested. Petrak (1982) described an idiopathic syndrome of dry gangrene of one or both legs in captive Budgerigars (*Melopsittacus undulatus*), the necrosis progressing up the foot and usually terminating at the tarsometatarsal joint. Calle et al. (1982, p. 473) described "distal extremity necrosis" in several captive birds of various orders, most cases secondary to frostbite, but with five cases of spontaneous gangrene involving mainly birds of the order Ciconiiformes (two Marabou Storks, *Leptoptilos crumeniferus*, one Lesser Adjutant, *Leptoptilos javanicus*). Lesions included gangrene of the toes and feet with loss of toes and, less often, loss of the upper or lower beak. Although the primary cause of the five cases of spontaneous gangrene was not determined, the authors identified myocardial lesions in two of these five birds and in several

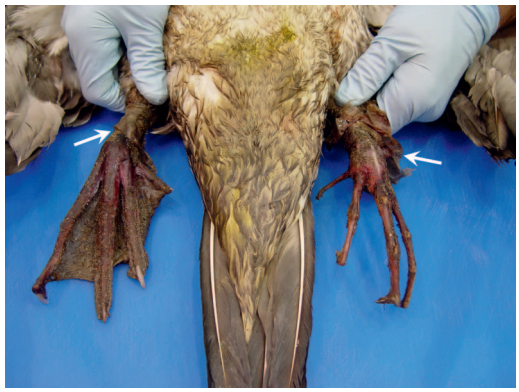

FIGURE 1. Hatch-year Northern Gannet (*Morus bassanus*) found along shores of the southern Gulf of St. Lawrence, Canada, with severe bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis (bird 11; Table 1). The left foot has complete loss of interdigital soft tissues, with widespread exposure of phalangeal bones and loss of all toe nails and some distal phalanges. Soft tissues over digits of the right foot are hyperemic. The arrows point to portions of the epidermis that have lifted off the feet.

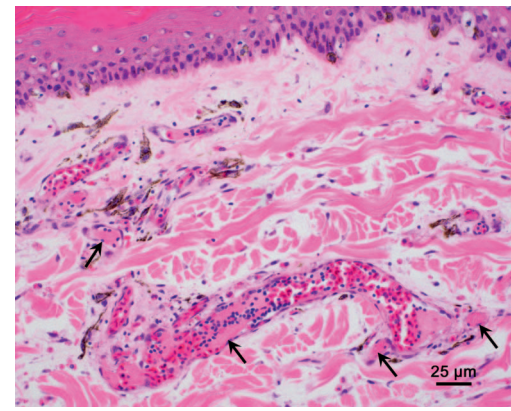

FIGURE 2. Hatch-year Northern Gannet (*Morus bassanus*) found along shores of the southern Gulf of St. Lawrence, Canada, with bilateral fibrinopurulent tarsometatarsal and pedal cellulitis (bird 10; Table 1). Multiple thrombi (arrows) in small blood vessels of a foot web. H&E stain.

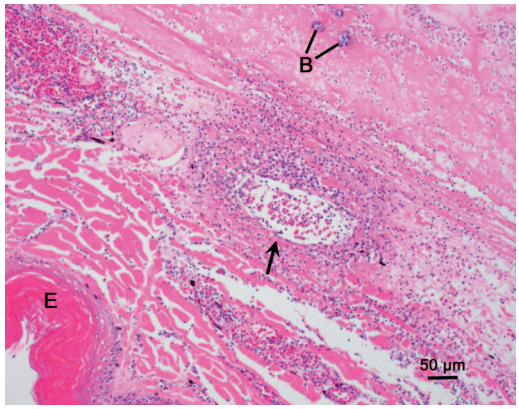

FIGURE 3. Hatch-year Northern Gannet (*Morus bassanus*) found along shores of the southern Gulf of St. Lawrence, Canada, with bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis (bird 9; Table 1). Severely necrotic and inflamed arteriole (arrow) in tarsometatarsal mesenchymal tissue close to the epidermal surface (E). Necrosis and edema more deeply in mesenchyme (top right corner); several bacterial colonies (B) with no associated inflammation. H&E stain.

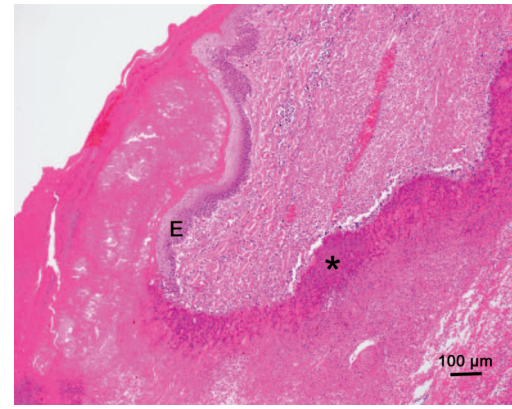

FIGURE 4. Hatch-year Northern Gannet (*Morus bassanus*) found along shores of the southern Gulf of St. Lawrence, Canada, with bilateral fibrinopurulent and necrotizing tarsometatarsal cellulitis and pedal necrosis (bird 4; Table 1). Sharp zone of demarcation, defined by a thick layer of necrotic inflammatory cells (*), between viable metatarsal epidermis (E) and mesenchymal tissue (above), and completely necrotic tissue (below). H&E stain.

of the birds that suffered from frostbite. They noted that most of the affected birds have long legs and suggested that the myocardial lesions may have been a predisposing factor by compromising blood circulation in the birds' extremities. It is therefore interesting that lesions of cardiomyopathy were identified in six of the 14 Northern Gannets with ischemic leg necrosis. However, care must be taken to suggest the same kind of cause-effect relationship in these birds because, as far as could be determined microscopically, myocardial lesions generally appeared more recent than the leg lesions in the same birds. Alternatively, these myocardial lesions may have been a result of the severe leg lesions, either because of the increased production of catecholamines associated with stress or because of toxemia associated with severe and extensive ischemic necrosis complicated by bacterial infection (Robinson and Robinson 2016).

Mechanical strangulation of part of a limb caused by entrapment in a snare targeting furbearing animals occurs with particular frequency in Bald Eagles (*Haliaeetus leucocephalus*), which, as semiobligate scavengers, are attracted to unconcealed baits in proximity to the snares (Fitzgerald et al. 2015). By analogy, an earlier consideration in the present series of Northern Gannets was possible entanglement in gillnets and subsequent release by the fishers. This was eventually dismissed as highly improbable because no remnant of gillnet was ever found entangled in the legs of these birds and because little fishing activity occurs in October and later in waters where these birds have been found. In cervids, adults and microfilariae of the nematode *Elaeophora schneideri* inhabit arterial beds, particularly those of the head, and can cause vascular thrombosis followed by dry gangrene of the ears and necrosis of the muzzle and nostrils, as well ischemic brain necrosis. This condition is seen most often in elk (*Cervus canadensis*), mainly calves and yearlings (Anderson 2001). No parasite was ever identified in soft tissue of affected legs in birds of this series.

In domestic animals, possible causes of ischemic necrosis of extremities include some forms of poisoning, bacterial embolism, disseminated intravascular coagulation (DIC), and cold agglutinin disease. Ergotism and fescue toxicosis are two classic examples of

poisoning causing gangrene of the extremities, including limbs, ears, and tail, in cattle. These toxicoses are associated with ingestion of plants contaminated by certain species of fungi and their alkaloids, resulting in peripheral vasoconstriction and thrombosis, possibly exacerbated by cold weather (Mauldin and Peters-Kennedy 2015). Poisoning would seem implausible in wild birds that strictly feed on live fish.

An infectious cause should be considered as a serious candidate to explain the pattern of lesions observed in the legs of these Northern Gannets. This interpretation is best exemplified by bird 14, which had a severe necrotizing enteritis caused by *Salmonella* Typhimurium, which was also isolated from soft tissues of one of its legs and may suggest a propensity for bacteria circulating in the blood to localize to some parts of the vasculature of Northern Gannets' legs along the tarsometatarsus, possibly related to a special configuration of the vasculature at this level. By analogy, bacteria tend to localize to sites of active endochondral ossification in growing animals because of the unique nature of the vascular architecture at these sites, coupled with other factors such as sluggish circulation and relatively inefficient phagocytic cells (Craig et al. 2015). The kidney is also a frequent site of infarction and subsequent necrosis from bacterial emboli because of the poor collateral circulation provided by its vascular architecture and the large volume of blood that continuously flows through this organ (Junqueira and Carneiro 2005; Cianciolo and Mohr 2016). The intact foot webs of bird 14, concurrent with an abundant amount of fibrinopurulent exudate in soft tissues along its tarsometatarsi, suggested an earlier stage of the syndrome that would have been followed by ischemic necrosis of the webs had this bird lived longer. A similar isolation of *Salmonella* Typhimurium from one of the legs of bird 13 also suggests a propensity of bacteria circulating in the blood to localize to this anatomic location, although the source of infection by this bacterium in this bird was not determined. Interpretation of the significance of isolation of *A. hydrophila* from the legs of several of the other birds is complicated by the fact that this bacterium is commonly found in the aquatic environment and therefore could easily have been a secondary invader in tissues devitalized by ischemia. The hemorrhagic diathesis in bird 3, characterized by petechiae and ecchymoses in multiple tissues and organs, could have resulted from DIC triggered by bacteremia or septicemia. Gangrene of the extremities can be a clinical manifestation of DIC (Robinson and Robinson 2016). However, the hemorrhagic diathesis in this bird was considered terminal, and it was not possible to determine whether the process of DIC that could have triggered it would have been a result or a cause of the leg lesions.

A comparison between lesions described in this group of Northern Gannets and a syndrome known as cold agglutinin disease is pertinent. Cold agglutinin disease is a rare type of immune-mediated hemolytic anemia caused by binding of autoantibodies (cold agglutinins) to the surface of red blood cells at lower than core body temperatures and, thus, usually associated with cold exposure (Pedersen 1999). Dry gangrene of the extremities resulting from the agglutination of red blood cells in smaller blood vessels can be a manifestation of this disease. This condition has been described in bovine calves in association with systemic infection by *S. enterica* subsp. *enterica* serovar Dublin and exposure to cold temperature in winter (Loeb et al. 2006), which can occur during the acute stage of infection, long after the calf has recovered from the infection, or in otherwise asymptomatic calves. Resulting from the production of antibodies in response to the infection and their cross-reaction with red blood cells, it typically affects the ears, the tip of the tail, and the distal part of the hind legs. Vascular thrombosis and vasculitis can be seen microscopically within the exudate and granulation tissue near the junction between affected and normal tissues. Interestingly, no case has been described in calves older than 3 mo, possibly because of an age-related difference in antibody response, a higher concentration of cold agglutinins in younger animals,

or a greater susceptibility of young animals to cold environmental temperature (Loeb et al. 2006). An analogy can also be made with a syndrome of wingtip edema and necrosis, with occasional involvement of the feet, in captive birds of prey (Simpson 1996; Chitty 2008). The etiology of this syndrome is still unknown. It affects mainly young birds in cold weather, especially if tethered close to the ground. Other factors besides cold temperature may predispose to this condition, and some have suggested the involvement of vasculitis from infection or toxin (Chitty 2008). Additionally, a succinctly described syndrome known as dermal septic necrosis has been observed in the Virginia opossum (*Didelphis virginiana*), affecting the ears and tail tips of animals with clinical signs of septicemia (Heatley 2009; McRuer and Jones 2009).

The small number of Northern Gannets observed with this syndrome over a period of 25 yr prevented us from detecting any annual change in the incidence of this syndrome. Sea surface temperature in the Gulf of St. Lawrence during ice-free months (May–November) has seen a warming trend of 0.9 C per century (Galbraith et el. 2012), likely accelerating in recent decades. This warming may already be enough to affect the marine ecosystem in this region, but perhaps not sufficient to decrease the influence of cold water temperature on the development of this syndrome in fledgling gannets, if this is a significant contributing factor.

Progress in explaining the potential cause of this proposed syndrome in Northern Gannets, particularly HY birds, would require better access to live affected birds. Obtaining affected HY birds is a difficult prospect, considering the very opportunistic nature of these observations and the fact that, when found, live affected birds are rarely in proximity to a research institution such as a veterinary college for further clinical assessment and most often need to be euthanased quickly for humane reasons. Access to live affected birds would provide an opportunity for detailed clinicopathologic investigation before euthanasia, such as potential evidence of DIC; more extensive bacteriologic examination could be done more reliably in fresh carcasses, and fresh carcasses could be used for basic studies of the vascular anatomy of the legs in this species.

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