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Pathological fracture of a red deer antler secondary to purulent inflammation - a case report

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ABSTRACT

A case of unilateral hard antler fracture in a red deer stag from Croatia is presented. The antler stump that remained, following the loss of the distal fracture fragment, shows evidence of purulent inflammation. The most likely cause of the inflammation is an injury to the velvet antler. We conclude that the fracture resulted from the impaired mechanical stability of the antler caused by the inflammation and its sequelae and thus represents a case of pathological fracture.

Key words: antler pathology, infection, purulent inflammation, red deer (*Cervus elaphus*)

We dedicate this paper to our friend and esteemed colleague, Professor George A. Bubenik, M.D. (Department of Integrative Biology, University of Guelph, Canada), on the occasion of his 70th birthday, May 19th 2012.

Introduction

Their rapid growth and mineralization, and the fact that they are the only mammalian bony organs capable of complete regeneration, make antlers useful models for the study of bone formation and regeneration in mammals (GOSS, 1983; BUBENIK, 1990; PRICE et al., 2005; KIERDORF et al., 2007, 2009; KIERDORF and KIERDORF, 2011). Growing antlers are covered by a special type of skin (velvet), which is richly vascularized, and innervated by sensory nerve fibers that grow out along the route of the major blood vessels in the velvet (WISLOCKI and SINGER, 1946; WALDO et al., 1949; GOSS, 1983; LI et al., 2007). After completion of antler growth and mineralization, the velvet is shed from the antlers

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that thereafter consist solely of bare bone and are referred to as hard antlers. It has been demonstrated in red deer (*Cervus elaphus* L.) that antlers completely dry out after velvet shedding (CURREY et al., 2009). More recently it was shown in the same species that, contrary to injections during antler growth, tetracycline injections given after the velvet had been largely or completely shed from the antlers, produced no labels in antler bone (GOMEZ et al., 2013). These findings corroborate the view (e.g., GOSS, 1983) that hard antlers are dead bony structures. As a consequence of the drop in blood testosterone levels after the rut, the hard antlers are cast and new ones start to grow. In Europe, mature red deer stags cast their antlers in late February/March and the velvet is shed in July/August.

Antlers are collected as trophies by hunters and closely examined at special exhibitions, with additional attention being paid to abnormalities. Abnormal antlers are, however, also of scientific interest, as their study can provide insights into the mechanisms underlying normal antler growth, as well as the pathogenesis of certain bone diseases. We report here a case of a pathological fracture occurring secondary to purulent inflammation in a red deer antler.

Materials and methods

The fractured antler belonged to a stag that was found dead on September 29th, 2010 in the hunting district No VII/15 "Zapadna Garjevica", located partly in the Bjelovarsko-bilogorska County and partly in the Sisačko-moslavačka County, Republic of Croatia. Age at death of the animal was estimated at 10 years. During the antler growth period in 2010, the individual was repeatedly observed and reportedly in good body condition. Antler growth of the stag was judged as 'normal'. At the beginning of the rutting season (early September), the stag was in hard antler (i.e., both antlers were cleaned of velvet), and was observed roaring. When seen the next time, the left antler had been broken and the stag no longer showed rutting behavior. Approximately a week later he was found dead. A necropsy was not performed because the carcass was already in decomposition.

We undertook a macroscopic inspection of the stag's antlers after the skull had been cleaned and prepared for exhibition. Computed tomography (CT) imaging (Somatom Sensation 16, Siemens, Forchheim, Germany, 5 mm slice width and 2 mm reconstruction interval, 120 kV and 138 mAs) was performed to reveal details of the internal structure of the antler stump on the left side.

Results

Both antlers of the stag were brown in color and free of adhering velvet, indicating that they had been cleaned in the normal way. The right antler was well developed with ten points, while on the left side only a stump was present, since the antler had been fractured above the brow tine (Fig. 1). The distal fragment of the left antler was not found.

However, when the stag was observed by game keepers shortly after velvet shedding, the left antler was still intact and judged as being normally developed, that is, of similar size and shape as the antler on the right side.

The fracture plane of the left antler shaft was rough with some large projections. The antler's interior revealed an extended cavitary system and bone areas of varying macroscopic appearance (Fig. 2). These comprised cancellous bone portions with an architecture typical of the spongy core of red deer antlers, but also more densely appearing portions with a rather smooth surface, suggestive of reactive sclerosis of the cancellous bone (Fig. 2). Some cancellous bone areas were sharply demarcated and interpreted as incompletely sequestered portions of necrotic bone. The cavitary system, which was

Fig. 1. Unilateral antler fracture in a red deer stag. The right antler is well developed with ten points. The left antler was fractured above the brow tine, leaving only a stump.

Fig. 2. View of the fracture plane, revealing the macroscopic aspect of the interior of the antler stump. Note extended cavitary system (arrows), a partly sequestered portion of cancellous bone (asterisk), and bone area with a smooth surface (cross), suggestive of reactive sclerosis. B - brow tine; C - coronet.

diagnosed as the remaining part of a larger abscess cavity, reached deep into the antler base (Fig. 3) and apparently even extended into the distal pedicle.

The antler stump exhibited the opening of a large draining sinus in the furcation area between the brow tine and shaft (Figs. 3, 4). Posterior to this opening the antler surface showed a shallow U-shaped groove (Fig. 4). Furthermore, a narrow and shallow furrow originating at the sinus opening in the furcation area between the brow tine and shaft extended over a short distance in the anterior direction (Fig. 4). This furrow presumably originated from bone resorption induced by pressure. Possible causes for this could be the accumulation of blood (hematoma) or pus, kept under the periosteum for some time before it either drained through the velvet or was released by velvet shedding. A second draining sinus, whose lumen was partly occluded by foreign material, opened on the anterior antler surface, immediately proximal to the fracture plane (Fig. 4). Smaller sinus openings were also present at the undersurface of the coronet (Fig. 5).

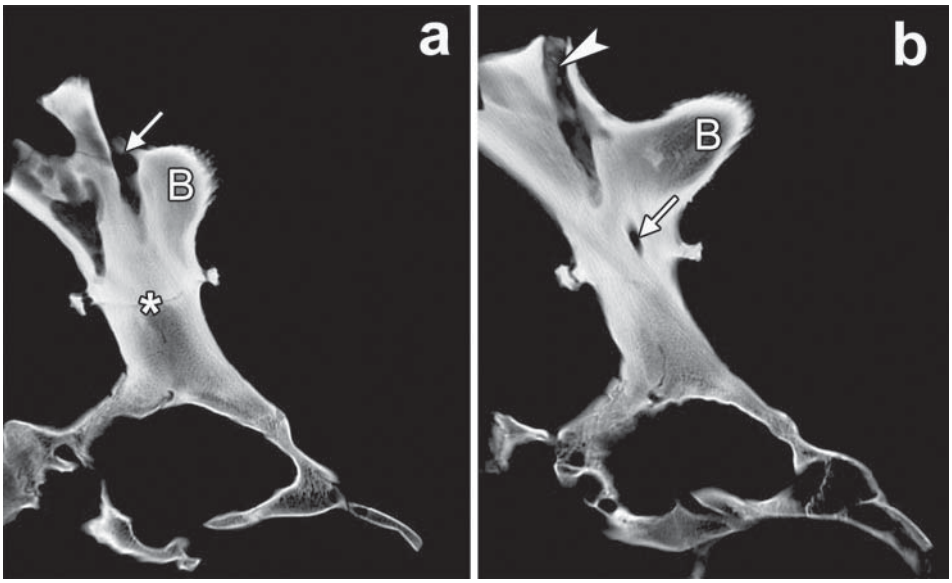

Fig. 3. CT images (a, b) of the stump of the left antler showing parts of the cavitary system. Scan a is positioned medial to scan b. B - brow tine; asterisk - antler-pedicle junction; arrows - draining sinus opening in the furcation area between main beam and brow tine and its extension into the antler base; arrowhead - partly occluded draining sinus that opens immediately proximal to the fracture plane.

Fig. 4. Furcation area between antler shaft and brow tine (B) of the antler stump. Arrowheads mark the opening of a draining sinus in the furcation area and the occluded opening of a second sinus further distally. A shallow furrow (arrow) extends from the proximal sinus opening onto the antler surface, indicating bone resorption caused by pressure exercised by blood/pus that was trapped for some time underneath the periosteum and velvet. F - fracture plane; single asterisk: occluding material in draining sinus; double asterisk - U-shaped groove.

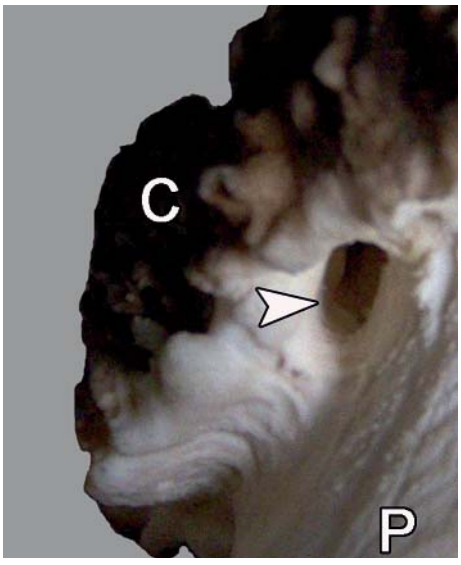

Fig. 5. Opening of a draining sinus (arrowhead) at the undersurface of the coronet (C). P - pedicle.

Discussion

The fractured left antler showed clear evidence of purulent inflammation. Inflammation of bone (osteitis) or bone and marrow (osteomyelitis) commonly results from an infection by pyogenic bacteria, although also other agents (viruses, fungi, parasites) can infect bone and marrow (RESNICK and NIWAYAMA, 1995; WEISBRODE and DOIGE, 2001; THOMPSON, 2007). Antlers lack marrow in the sense of haematopoietic tissue (WISLOCKI, 1942). Therefore the term 'marrow' in the case of antlers can only be used in a topographical sense, as for instance by WISLOCKI (1942).

In wild animals, different routes of bacterial infection of bone are possible. These are: haematogenous spread, spread of infection from an adjacent contaminated site, or direct implantation of bacteria into the bone, in the case of puncture or penetrating wounds. In domestic animals, the most common route of infection is haematogenous, with a predilection for sites of endochondral ossification (THOMPSON, 2007). Given the rapid endochondral growth and rich blood supply of forming antlers (GOSS, 1983), an infection via haematogenous spread is in principle also conceivable in the present case. However, since the stag was reportedly in good physical condition during the antler growth period, it seems more likely to us that the infection resulted from a (puncture) wound to the velvet antler. It might be assumed that the U-shaped groove, located posterior to the more proximally located sinus opening on the anterior side of the antler, formed as a result of a traumatic impact on the antler and that the wounding occurred in the area of the opening, but this must remain speculative.

The observations by the game wardens that the affected antler was normally developed (normal size and shape) prior to fracturing suggests that the inflammation did not start during early antler growth, because this would probably have resulted in some sort of antler malformation. Rather it is assumed that infection and inflammation occurred when the antler had already been growing for some time. In a recent study on red deer, the period between casting of the old antlers and completion of velvet shedding from the regenerated antlers ranged between 134 and 163 days (GOMEZ et al., 2013). Experimental animal studies have demonstrated that osteomyelitis develops within some weeks after inoculation with bacteria. Thus in the rabbit tibial model, disruption of normal bone architecture, bone necrosis and formation of sequestra started two weeks after inoculation with staphylococci, while injection of *Staphylococcus aureus* suspension into the marrow cavity of dog femora, along with the introduction of sclerosing agents and foreign bodies, caused extensive osteomyelitis within 5 to 7 weeks (AN et al., 2006). On the basis of these data, and considering the highly dynamic nature of antler growth, we conclude that there was enough time for the development of extensive purulent inflammation in the antler, even if the process started at a relatively advanced growth stage.

In humans, osteomyelitis is a well-known complication of bite wounds and puncture wounds to the foot, especially its plantar aspect (RESNICK and NIWAYAMA, 1995; CETINUS et al., 2005). Because of their rich sensory nerve supply, growing antlers are very sensitive structures and deer therefore avoid contact between their velvet antlers and other objects. Only after velvet shedding, i.e., when the antlers are dead, do stags use them for fighting, sparring, and marking. Therefore, it is concluded that the infection of the velvet antler presumably occurred as a result of accidental wounding, which may have happened when the alarmed animal tried to escape from a frightening situation. In the hunting district from which the antlers originate, until quite recently fences (made from reinforcing bars) were used to exclude large herbivores and wild boar from areas designed for natural forest regeneration (old forest areas with seed production) and young forest stands. Remnants of these fences are still present in the area, and it seems possible that the stag injured his antler on them.

CT imaging revealed that the inflammatory process in the antler extended deep into the antler base and from there later apparently also into the distal pedicle. In the case of purulent inflammation, accumulation of exudate (pus) increases intra-bone pressure, resulting in compression of blood vessels, that induces thrombosis and infarction of bone and marrow tissues. Osteoclastic bone resorption is stimulated by prostaglandins and cytokines released in the area of inflammation, as well as by a reduced blood flow (WEISBRODE and DOIGE, 2001). In the antler, portions of the cancellous bone became necrotic and were (incompletely) sequestered in the course of the inflammatory process, while other portions appear to have undergone reactive sclerosis. The cavitary system indicates intense resorption activity within the antler. The pus filling the cavities was eventually drained through several sinuses.

Since antlers dry out after velvet shedding (CURREY et al., 2009), we assume that velvet shedding terminated the inflammatory process in the antler. The dry condition of the hard antler rules out the possibility that the inflammation developed secondary to the fracturing of the (hard) antler. This conclusion is corroborated by the fact that accidental damage to hard antlers or the cutting of hard antlers above the coronet, as is often practiced in deer farms to avoid injuries during the rutting period, does not result in infection and inflammation of the remaining antler portion or stump.

In the case presented here, fracturing is thought to have occurred as a consequence of the antler's compromised mechanical properties (weakening of the antler beam) resulting from the preceding bone necrosis and resorption in its interior. The antler fracture can therefore be classified as a pathological fracture, i.e., a fracture developing at the site of a preexisting abnormality, causing a mechanical weakening of the bone (RESNICK et al., 1995).

Fractures caused by trauma to pedicles and growing antlers are frequently observed (GOSS, 1983). Since the fracture fragments are normally held together by the periosteum and skin, such fractures typically heal, with deformation. In contrast, fracturing of hard antlers leads to the loss of the portion distal to the fracture plane, as it also happened in the present case. There is evidence that mineral deficiencies in the diet may impair antler mineralization and cause an increased frequency of hard antler fracture (JOHNSON et al., 2007; LANDETE-CASTILLEJOS et al., 2010).

Cases of pathological bone fracture secondary to osteomyelitis have been described in humans and animals (WALMSLEY, 1983; EMMERSON and PEAD, 1999; LIN et al., 2010; BELTHUR et al., 2012). While several instances of purulent inflammation of antlers have previously been observed (BRANDT, 1901; RAESFELD, 1919), our literature search yielded only a single previous report of a pathological antler fracture secondary to inflammation (SCHULTE, 2011). The case, which was similar to that reported here, occurred in a red deer stag from Germany found dead in the year 2007. Since in our case the stag was found dead and already in decomposition, it remains unknown and open to speculation whether his death was related to the antler inflammation. Haematogenous spreading of the infection from the antler (prior to velvet shedding) or the affected pedicle could have caused severe sepsis, eventually resulting in death (BALK, 2000). However, the stag could also have been lethally injured by another male during an aggressive encounter.

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SAŽETAK

U radu je prikazan slučaj jednostranog prijeloma mineraliziranog roga (lijeva grana) jelena običnog podrijetlom iz Hrvatske. Na preostalom proksimalnom ulomku roga vidljivi su znaci gnojnog upalnog procesa koji je zahvatio rog tijekom faze rasta (rogovlje u bastu). Najvjerojatniji uzrok navedenog stanja je ozljeda rastućeg roga s posljedičnom infekcijom. Iz navedenog zaključujemo da je prijelom posljedica narušene mehaničke stabilnosti roga izazvane upalnim procesom i njegovim posljedicama te stoga predstavlja primjer patološkog prijeloma.

Ključne riječi: patologija rogovlja, infekcija, gnojna upala, jelen obični, *Cervus elaphus*