Periosteal osteosarcoma of the scapula in a horse

Jeffrey F. Zaruby, J. Wesley Williams, Sandra L. Lovering

Osteosarcomas are highly malignant tumors of skeletoblastic mesenchyme in which the tumor cells produce osteoid or bone (1,2). These products, called tumor osteoid or tumor bone, are not considered malignant since it is the tumor cells that possess the characteristics of malignancy, not the matrices they produce (1,2).

While the incidence and nature of osteosarcoma are well documented in domestic animals generally, this is not the case for horses. Osteosarcomas are the most common type of primary bone tumor reported in domestic animals, accounting for over 80% of malignant skeletal neoplasms in dogs, and greater than 50% of those in cats (1–4). In horses, the reported incidence is much lower, perhaps due to the fact that large animals rarely live to "cancer-forming age".

A search of databases (The Commonwealth Agricultural Bureau International Abstracts, and Medline-National Library of Medicine) for the years 1983 to 1992, inclusive, failed to produce a single reported case of primary scapular osteosarcoma in horses. We describe herein the clinical, radiographic, and pathological evaluation of a unique case of primary periosteal osteosarcoma involving the scapula of a horse.

An eight-year-old Arabian gelding was presented for evaluation of a lameness of unspecified duration. While walking, the horse was noticeably lame on the left forelimb, with the lameness becoming markedly worse while trotting. The horse exhibited a shortened anterior swing phase with decreased range of motion in the shoulder.

The horse was in good body condition except for atrophy of the musculature of the left shoulder and upper forelimb. The heels of the left forefoot were longer than the heels of the right forefoot, and the gelding stood more upright through the proximal interphalangeal and metacarpophalangeal joints on the left in comparison to the right foreleg.

Cranial protraction, with extension of the scapulohumeral joint, demonstrated a decreased range of motion in the shoulder joint and elicited a painful response from the horse. Digital palpation of the shoulder joint and bicipital bursa revealed soft tissue enlargement.

Can Vet J 1993; 34: 742–744

Department of Clinical Studies, Ontario Veterinary College, Guelph, Ontario N1G 2W1 (Zaruby), Las Colinas Veterinary Clinic, 600 Royal Lane, Irving, Texas, USA 75039 (Williams), and Texas Veterinary Medical Diagnostic Laboratory, Drawer 3040, College Station, Texas, USA 77841 (Lovering).

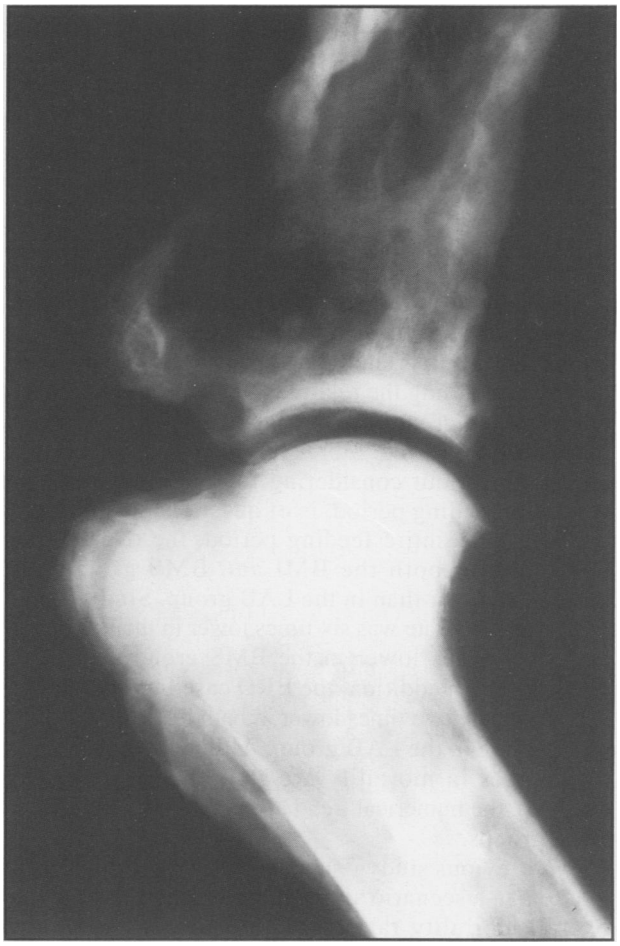

Figure 1. Postmortem lateromedial radiograph of the dissected scapulohumeral joint. There is loss of normal metaphyseal architecture in the scapula with areas of lysis, sclerosis, and periosteal osteoid production. There is significant productive periosteal reaction present on the proximal humerus.

Standing mediolateral radiographs of the scapulohumeral joint were obtained despite the horse's reluctance to have the leg extended in front of him. Radiographic changes included marked periosteal proliferation on the distal neck of the scapula, supraglenoid tubercle, and metaphyseal region of the humerus. These changes were accompanied by decreased bone density in the distal scapula.

On the basis of the clinical and radiographic findings, a tentative diagnosis of malignant neoplasia was made. Differential diagnoses included severe degenerative joint disease and/or osteomyelitis. Owing to the severity and apparent chronicity of the lameness, a poor prognosis for return to function was given, and the horse was humanely destroyed at the owners' request.

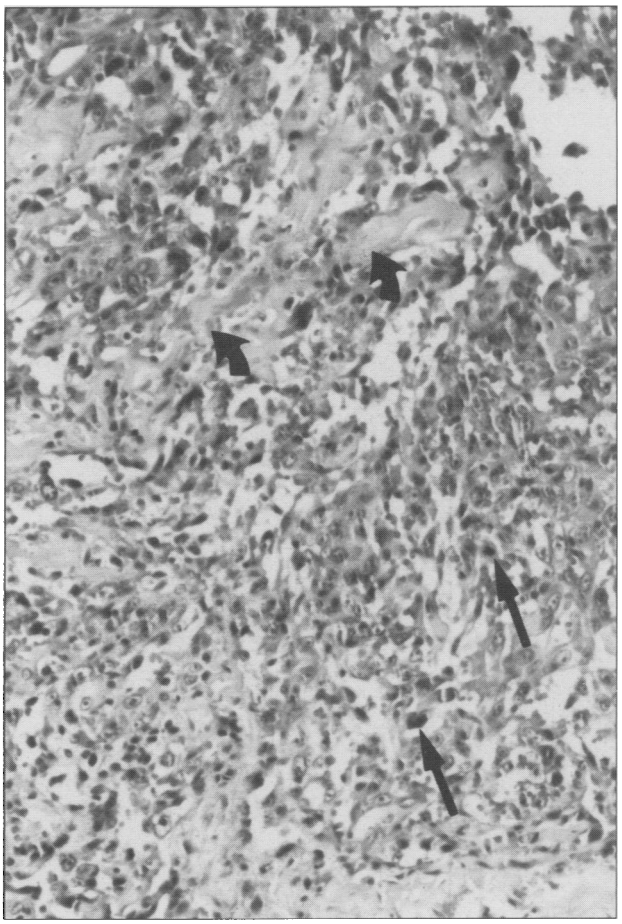

Figure 2. Photomicrograph of a transverse section of the distal scapula. The tumor is comprised of spindle-shaped cells which are variable in size and in which mitotic figures are frequent (straight arrows). Irregularly arranged collagen comprises the tumor osteoid (broad, curved arrows). H&E. ×120.

Significant necropsy findings were confined to the scapulohumeral region and adjacent soft tissues. The joint capsule and bicipital bursa were thickened, and the musculature of the shoulder was atrophied. There was generalized proliferation of the periosteum, starting at the distal neck of the scapula and extending distally to the metaphyseal region of the humerus. A lateromedial radiographic view of the dissected shoulder was taken (Figure 1). The articulating surfaces of the shoulder joint were not involved. Transverse sectioning of the scapula revealed soft, hemorrhagic areas, with focal patches of light yellow necrosis. Destruction of the endosteal surface of the cortical bone was evident but the cortices remained intact. Macroscopic separation between normal and neoplastic bone was poorly delineated.

Microscopically, the bone marrow exhibited mild hemosiderosis. There were areas of dense reactive cortical bone overlaid with irregularly thickened periosteum. The periosteum was composed of spindle-shaped cells characterized by variation in nuclear size and frequent mitoses (Figure 2). The histopathological results confirmed the tentative diagnosis of malignant neoplasia. The final diagnosis was periosteal osteosarcoma of the scapula.

No pulmonary, visceral, lymphoid, or other soft tissue metastases were apparent at postmortem examination. Although early hematogenous metastasis is characteristic of osteosarcomas, such soft tissue lesions are usually not appreciable grossly or radiographically at the time the primary tumor is detected (1).

In dogs, cats, and humans, osteogenic sarcomas localize largely in the appendicular skeleton with the most common predilection sites being the proximal humerus and tibia, and the distal femur, radius, and humerus (2,5–7). In a study of 75 cases of canine osteosarcoma involving the appendicular skeleton, only four cases (5.3%) involved the scapula (2). Three of those four were located in the blade region and one in the distal neck of the scapula, which demonstrates the rarity with which the scapula is involved in cases of primary osteosarcoma in domestic animals.

In horses, osteosarcomas have a predilection for the skull (1,2,4,8,9), with greater than 80% of reported cases occurring in the jaw. Whereas in dogs, tumors involving the skull tend to appear later in life than tumors of the appendicular skeleton (3), in horses there is evidence to suggest that the opposite is true (3). If appendicular osteosarcomas truly do have a later chronological expression than tumors of the head, the average lifespan of horses may limit the natural expression of osteogenic sarcomas in the appendicular skeleton. This would account for the rarity with which osteosarcomas are documented in the appendicular skeleton of horses (1,5).

Although most osteosarcomas originate in the metaphyseal region of long bones (2,3), some arise in the periosteum and occasionally in extraskeletal tissues. The two primary osteosarcomas arising from the periosteum are (i) periosteal osteosarcomas, and (ii) juxtacortical (parosteal) osteosarcomas (6). Juxtacortical osteosarcomas demonstrate a high degree of structural differentiation with slow growth rates. In contrast, periosteal osteosarcomas tend to extend into the epiphysis rather than the diaphysis, and demonstrate marked periosteal reaction with cortical bone erosion, but seldom is the articular cartilage perforated (6).

Although no specific cause of osteosarcoma has been established, various etiological theories exist. These include trauma, viral agents, exposure to ionizing radiation, bone infarcts, and hereditary/genetic etiologies (10). Support for multiple trauma as an inciting factor stems from the observation in man and domestic animals that primary osteosarcoma tends to occur in major weight-bearing bones and late-closing physes. The increase in cell turnover arising from multiple minor traumatic episodes may result in increased cell mutagenesis, and the development of neoplastic cell characteristics (2,3,6,7). A viral etiology of osteosarcoma has been suggested by the observed occurrence of osteosarcoma in littermates, and the ability to induce the disease in fetal puppies injected with osteosarcoma cells (10). Ionizing radiation has been shown to produce osteosarcomas in beagles exposed to radon, plutonium, and strontium (10). Evidence for a genetic component stems from the observation that children with genetically transmissible retinoblastomas have a significantly increased incidence of femoral osteosarcoma (11). It is proposed that the retinoblastoma gene predisposes these individuals to secondary neoplasms (11).

The typical radiographic appearance of a primary osteosarcoma is that of an aggressive metaphyseal

lesion in which the normal metaphyseal architecture is lost (3,7). Osteosarcomas may be completely lytic or predominantly sclerotic, but they usually exhibit a combination of these features (6). The lesion is usually poorly delineated, with a long zone of transition between normal and abnormal bone, and no sclerotic border at the margin of the lesion. Periosteal elevation on either side of the lesion can be irregular and discontinuous over the lesion, with abundant periosteal new bone formation and production of neoplastic bone that is spicular or disorganized (3,7). Proliferating bone produced by the neoplastic cells characteristically has a streaked texture radiating out at right angles from the primary lesion; this is called the sunburst effect (5,6).

Historically, it has been accepted that the physis acts as a barrier to the spread of neoplasms. Extension of neoplastic tissue across the growth plate to include the epiphysis of long bones was considered an unusual event (12). Recently, several reports have documented direct extension of long bone osteosarcomas across growth plates, regardless of whether the growth plate was functionally mature or not (12). If a joint does become involved, the opposing bone will not be invaded by the neoplastic process in most cases (7).

Our case was particularly interesting in that the production of tumor osteoid had crossed the scapulohumeral joint to involve the proximal humerus, which is uncharacteristic of this type of neoplasm. CVJ

References

1. Gillette EL, Thrall DE, Lebel JL. Carlson's Veterinary Radiology. 3rd ed. Philadelphia: Lea & Febiger, 1977: 360–362.
2. Jaffe HL. Tumors and Tumorous Conditions of the Bones and Joints. Philadelphia: Lea & Febiger, 1958: 256–278.
3. Pool RR. Tumors of bone and cartilage. In: Tumors in Domestic Animals. 3rd ed. Moulton JE, ed. Berkeley: University of California Press, 1990: 181–195.
4. Jubb KVP, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals. 4th ed. Vol 1. San Diego: Academic Press, 1993: 133–136.
5. Gibbs C, Denny HR, Kelly DF. The radiographical features of osteosarcoma of the appendicular skeleton in dogs: A review of 74 cases. J Small Anim Pract 1984; 25: 177–192.
6. Dahlin DC. Bone Tumors. General Aspects and Data on 6221 Cases. 3rd ed. Springfield, Illinois: Charles C. Thomas, 1978: 226–260.
7. Jacobsen SA. The Comparative Pathology of the Tumors of the Bone. Springfield, Illinois: Charles C. Thomas, 1971: 164–211.
8. Dahlin DC, Coventry MB. Osteogenic sarcoma: A study of 600 cases. J Bone Joint Surg 1967; 49A: 101–110.
9. Livesey MA, Wilkie IW. Focal and multifocal osteosarcoma in two foals. Equine Vet J 1986; 18: 407–410.
10. Withrow SJ, MacEwen EG. Clinical Veterinary Oncology. Philadelphia: J.B. Lippincott, 1989: 234–252.
11. Huvos AG. Bone Tumors. Diagnosis, Treatment, and Prognosis. Philadelphia: W.B. Saunders, 1979: 47–93.
12. Norton KI, Hermann G, Abdelwahab IF, Klein MJ, Granowetter LF, Rabinowitz JG. Epiphyseal involvement in osteosarcoma. Radiology 1991; 180: 813–816.