History and Physical Examination Findings

A 6-year-old Swedish Warmblood gelding was evaluated by the referring veterinarian because of right mandibular swelling of 24 hours’ duration. The horse was having difficulty eating and was evasive when its head was approached to be examined. On closer examination, the horse resented palpation of the lower jaw and had marked pyrexia. No other abnormalities were found on physical examination. A detailed oral examination was not performed. A radiographic view of the right mandible was obtained (Figure 1). The swelling was considered to be a result of trauma, such as a kick from another horse. Meloxicam (0.15 mg/kg [0.07 mg/lb], PO, q 24 h) was prescribed for 5 days. The horse was reexamined 7 days later. The right mandibular swelling had increased slightly in size. Radiography was repeated, but a conclusive diagnosis was not obtained. Oral administration of meloxicam was continued for another 14 days.

A second opinion was sought 30 days after the first onset of clinical signs. The horse had fewer signs of pain following meloxicam administration, but the right mandibular swelling remained largely unaffected. No other abnormalities were evident on physical examination. Oral examination revealed a nondisplaced sagittal crown fracture passing through the 2 buccal pulp horns (first and second pulp horns) of the right mandibular third premolar tooth. It was not possible to determine the apical extent of the sagittal fracture. A third radiograph was obtained as part of the oral examination (Figure 1). Creation of an occlusal access site to the buccal pulp horns with a high-speed dental drill resulted in drainage of pus.

Determine whether additional images are required, or make your diagnosis from Figure 1—then turn the page →

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Diagnostic Imaging Findings and Interpretation

The left ventral-to-right dorsal oblique extraoral radiographic view of the right mandible (Figure 2) showed portions of the second premolar tooth and first molar tooth as well as the roots, reserve crowns, and most of the clinical crowns of the third and fourth premolar teeth. No radiographic abnormalities of the teeth or surrounding bone were observed. A soft tissue swelling was evident ventral to the mesial root of the third premolar tooth and extended rostrally along the ventral mandibular margin.

On a similar radiographic view obtained 7 days later (Figure 2), a focal radiographic abnormality was observed in the right mandible with the epicenter ventral to the distal root of the third premolar tooth and overlaying the mandibular canal. The intraosseous lesion was moderately large in size, extending from the apices of the third premolar tooth to involve the cortical bone of the ventral mandibular margin. It had elicited a large, immature periosteal reaction. The lesion had an irregular shape with an ill-defined, invasive border (also referred to as a permeative pattern) of bone destruction. No teeth were displaced. Except in the region of the apices of the third premolar tooth, the periodontal space appeared radiographically within normal limits, and the lamina dura appeared to be intact. The lesion extended ventrally from the periapical region. The permeative bone pattern continued through the cortical bone of the ventral margin of the mandible. A large amount of soft tissue swelling was visible ventral to the mandible.

On the radiographic view obtained 30 days after the onset of clinical signs (Figure 2), an irregular periapical lesion of similar shape and dimensions to the lesion seen on the previous radiographic view was evident. However, there was an ill-defined, short, blending border of condensing osteitis or osteosclerosis. The internal structure of the lesion appeared more radiopaque, and a solid periosteal reaction was evident with a large amount of surrounding soft tissue swelling still present. Taken together, the radiographic views illustrated the development of an acquired inflammatory intraosseous lesion (subperiosteal osteomyelitis) secondary to endodontic disease of the right mandibular third premolar tooth.

The only radiographic abnormality detected at the time of initial examination was the soft tissue swelling along the ventral margin of the right mandible. The severity of the clinical signs was suggestive of a mandibular fracture, acute apical periodontitis, or a periapical abscess. It was not possible to rule out a hairline or nondisplaced fracture of the mandible on the basis of the single radiographic view that was obtained, and demineralization of bone associated with an apical abscess may not have been severe enough to be radiographically evident. A neoplastic process was unlikely.

When the horse was reexamined 7 days after the onset of clinical signs, the radiographic appearance of the periapical lesion combined with the cortical bone involvement, associated periosteal reaction, permeative pattern of bone destruction, and lack of displacement of anatomic features were suggestive of an aggressive inflammatory lesion, most likely subperiosteal osteomyelitis as a result of endodontic disease. In the absence of a tooth fracture, malignancy would have been a possible differential diagnosis.

At the time of the third examination 30 days after the onset of clinical signs, the periapical lesion had not increased in size, but the internal structure appeared more radiopaque. There was an ill-defined, short blending border, and new periosteal bone had been formed, which suggested that the inflammatory lesion had become chronic.

Treatment and Outcome

The horse was sedated, and endodontic treatment of the affected tooth was performed. In addition, odontoplasty was performed on the occlusal surface of the fractured tooth to temporarily take it out of occlusion and reduce the occlusal forces acting on the tooth, which was expected to prevent displacement of the buccal fragment and provide immedi-
ate pain relief. Penicillin procaine (25 mg/kg [11.4 mg/lb], IM, q 24 h) was administered perioperatively starting the day before the procedure and continuing for 3 days after surgery. The soft tissue swelling resolved within 1 week after endodontic treatment. Two years after endodontic treatment, radiography revealed resolution of the bony changes in the periapical region of the treated tooth. Five years after endodontic treatment, the horse continued to compete successfully in high-level dressage without signs of oral abnormalities.

The case described in the present report illustrated radiographic changes seen in the development of acute subperiosteal osteomyelitis resulting from endodontic disease over a period of 1 month. At the first visit, the periosteum had separated from the cortical bone owing to an accumulation of inflammatory cells and exudate (the delineation between the periosteum and ventral soft tissue swelling was only appreciated with the image in its original digital format). There was no radiographic evidence of periapical changes. In human mandibles, an estimated 7.1% decrease in total mineral content is required before a focal resorptive bone lesion is radiographically evident. This usually means that detection of a periapical lesion is not radiographically possible until loss of cortical bone starts to occur.4 It is likely that the situation is similar for equine mandibles. Thus, it is possible that osteomyelitis of the cancellous bone was already present, although radiographically undetectable, in this horse at the time of the first examination and that this osteomyelitis resulted in the subperiosteal accumulation of inflammatory cells and exudate.

At the second visit, a permeative pattern of bone destruction was seen radiographically. A permeative pattern reflects the most aggressive form of bony destruction, during which resorption of bone is not matched by formation of new bone. It occurs on the endosteal surface of the cortical bone only and is an extension of a cancellous process, such as osteomyelitis or neoplasia.5 Apical periodontitis is an extension of endodontic disease and has been suggested to be radiographically evident in horses as soon as 15 to 21 days after injury; however, time from infection to development of apical periodontitis is widely variable.5 This is similar to the situation in dogs, in which apical periodontitis has been detected radiographically as early as 14 or 15 days after endodontic infection.6,7 The importance of performing a thorough oral examination cannot be overemphasized, particularly when dental structures may be implicated. In the horse described in the present report, the degree of morbidity, pyrexia, and mandibular swelling in the absence of a mandibular fracture were suggestive of endodontic disease. High-quality radiographs were essential as part of the diagnostic process.

The presence of a complicated crown fracture involving the endodontic system was strong evidence for the origin of the infection in the horse described in the present report. Sagittal slab fractures of the crown of mandibular molariform teeth almost invariably involve the pulp; however, irreversible pulpitis is not always the consequence.8 Fracture planes in equine molariform teeth have been well described,9 and the pattern of fracture through the buccal pulp horns in this horse was typical for a sagittal fracture of a mandibular molariform tooth. Approximately half of the fractures affecting the molariform teeth are idiopathic, but most mandibular tooth fractures are associated with mandibular fracture and involve the premolar teeth.10,11 In equine mandibles, 50% to 90% of tooth fractures have been found to involve the buccal pulp horns, with slab fractures most commonly occurring in middle-aged horses.9,11 Most periapical infections of the mandibular molariform teeth in horses are thought to be due to anachoresis; however, 20% are reported to be due to dental fractures.12

Radiography was important to rule out mandibular fracture as a cause of the mandibular swelling in the horse described in the present report and to confirm endodontic disease as the most likely cause. This case illustrates the radiographic progression of endodontic disease in a horse from acute apical periodontitis to acute focal subperiosteal osteomyelitis to chronic osteomyelitis. It is important to be aware that bony changes may not be radiographically evident even if a large amount of cancellous bone is demineralized, as was possibly the case in the radiograph obtained at the time this horse was first examined. On the radiograph obtained at the time of the second visit, aggressive bony destruction reflective of uncoupled osteoclastic-osteoblastic activity was evident, and on the radiograph obtained at the time of the third visit, the disease process appeared to be in a more chronic phase of inflammation, resulting in increased osteoblastic activity, compared with osteoclastic activity. The importance of a detailed occlusal examination should not be understated for a horse with facial swelling overlying dental structures. Such an examination would have enabled a diagnosis of endodontic disease as the primary cause of clinical signs at the first visit, with appropriate treatment performed at a much earlier stage of the disease.

References