Craniomandibular Osteopathy

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Craniomandibular osteopathy (CMO) is a nonneoplastic, proliferative bony disease of the dog affecting primarily the mandible, tympanic bullae, and occasionally other bones of the head. In certain instances other long bones may be affected also. The disease predominates in Scottish terriers,(4,6) West Highland white terriers,(13) and cairn terriers; however, boxers, (15) Labrador retrievers,(16) Great Danes,(5) and Doberman pinschers(17) have also been reported in the literature.

The disease is seldom recognized until signs of discomfort due to chewing and eating are observed. This usually occurs when the dogs are from 4 to 7 months old: the mandible is bilaterally thickened and the angular processes of the mandible and tympanic bullae are usually so large and tender that the mouth cannot be fully opened. The tenderness of the affected mandible is associated with intermittent fever (as high as 104°F) of 3 or 4 days’ duration. This tenderness and fever may recur every 2 to 4 weeks during the bony proliferation phase.(14)

Although the primary disease process does not seem to cause death, euthanasia has been performed because of severe pain and malnutrition resulting from the inability to eat. It has been a common observation that when the affected dog is approximately 11 to 13 months of age, the disease may become self-limiting. The growth of abnormal bone slows, often regresses, and sometimes recedes completely. This period of self-limitation coincides with the time of completion of regular endochondral bone growth and ossification.

Radiography is the best method for demonstrating the lesions of CMO. At present, the cause of the disease is not known.

HISTORY
The first written description of CMO appeared in 1958.(9) It was described in five West Highland white terriers affected within a 2-year period. In this instance the disease was thought to be neoplastic and the affected dogs were destroyed. Histologically, however, it was diagnosed as abnormal benign osseous and chondromyxomatous proliferation.(3)

In 1959 in Canada the same disease was reported in Scottish and West Highland white terriers 4 to 6 months of age. It was described as mandibular periostitis associated with dysfunction of the temporomandibular joint.(3) The chief clinical signs were an inability to open the mouth completely, weight loss, and intermittent fever.

Another report described CMO in Scottish and West Highland white terriers as a disease of extensive newbone formation of
the mandible and of the temporal and occipital bones. (1)

In one text, reference was made to the three reports mentioned above, and a brief description of the disease was given. (8)

Riser reported the histologic findings of 18 affected dogs in 1967 and proposed an infectious process based on the clinical signs and presence of inflammatory cells in some areas of new bone. (13)

Putnam and Archibald described the disease in 1968 as mandibular periostitis and concluded that while the cause is unknown, CMO is suspected to be congenital and possibly inherited. (12)

Watson described two atypical Doberman pinschers with CMO in 1975. Although both animals had firm enlargements of the mandible bilaterally, the swellings were painless. One dog had an elevated serum calcium level. (17)

Alexander and Kallfeiz reported a case of CMO in a 6-month-old Labrador retriever in 1975. They injected the dog with 99mTc-MDP (methylene diphosphonate), which is a bone-seeking radio-pharmaceutical. A subsequent bone scan demonstrated that uptake of the drug was increased significantly in the mandibular rami, indicating increased new-bone formation. (2)

Schulz in 1978 described a boxer with CMO from whose affected mandibular bone he cultured Enterobacteriaceae sp. The dog responded to high-dose corticosteroids combined with specific antibiotic therapy. (15)

Reviews of the disease by Alexander in 1972 (2) and Newton in 1982 (10) did not offer new insight into the etiopathogenesis of the disease.

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CLINICAL SIGNS

Onset of the disease is usually between 4 and 10 months of age. Both sexes are affected equally. The animal may be presented because of obvious mandibular swelling, drooling, inability to open the mouth, or pain on opening or manipulation of the mouth.

On physical examination, there is usually a bilateral, symmetric firm or bony swelling of the horizontal, and occasionally vertical, rami of the mandible. The bony enlargement may be painful on direct palpation of the swelling. Discomfort is elicited when attempting to open the animal's mouth; in advancing disease the mouth may not open more than 1 cm to 2 cm (Fig. 54-1, A). Dogs may be febrile during the period of bone proliferation. (1) Riser described dogs with fevers of up to 104°F that lasted 3 to 4 days and occurred every 10 to 14 days from the time the disease was discovered until the animals were 8 months old. (14) Lymphadenopathy or temporal muscle atrophy may also be present. Hypertrophic bone may be present in the soft tissues adjacent to long bones. The new bone is similar in appearance to the bone found in hypertrophic osteodystrophy and may result in lameness (Fig. 54-2). (14)

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RADIOGRAPHIC FINDINGS

The diagnosis of CMO usually requires radiographic demonstration of the bony proliferative lesions. Survey radiographs of the skull and mandible should include oblique, dorsoventral, and lateral views to adequately assess the presence or extent of the disease. The dog may need to be anesthetized or sedated to obtain proper positioning and radiographic detail.

The radiographic lesions of a noninfectious, nonneoplastic bony proliferation usually involve the cranial bones of the skull and the mandibles (Fig. 54-1, B). The horizontal rami and angles of the mandibles and tympanic bullae of the temporal bones are affected most commonly. The lesions may involve other bones, including the occipital bone, parietal bone, frontal bone, maxilla, and rarely an appendicular bone (Fig. 54-2).

The bony proliferations are usually bilateral and symmetric. Often the bone cortices and medullary cavity are not visible owing to the superimposed new bone. The teeth are normal. The abnormal bony changes may be localized or generalized for any affected bone. Occasionally, no radiographic abnormality is present at the time of the disease onset. The poorest clinical prognosis involves dogs who have radiographic evidence of partial or complete bony ankylosis of the temporomandibular joints.
The proliferation of new bone of the head and jaws decreases as the endochondral ossification of the long bones slows after 7 to 8 months of age. By the time the growth plates close at 11 to 13 months of age, the bony proliferation of the skull becomes radiographically static and the beaded edges of the affected bone recede and become smooth. In nine dogs the size of the bony proliferation had decreased by half by 13 months of age (Fig. 54-3)(14)

![FIG. 54-1 Craniomandibular osteopathy in an 8-month-old West Highland terrier. (A) In this macerated skull specimen the mandible and tympanic bullae are thickened and fused by extraosseous bone. (B) Radiograph of the skull in A. In addition to the osseous changes of the mandibles and tympanic bullae, the parietal crest is thickened. (Riser WH, Parkes LJ, Shirer BS: Canine craniomandibular osteopathy. J Am Vet Radiol Soc 8:23, 1967)](image)

![FIG. 54-2 Craniomandibular osteopathy in a 5-month-old Scottish terrier. Radiograph of the radius and ulna. (A) The ulna is increased in diameter and more radiopaque than the radius. Heterotopic bone is present in the soft tissue adjacent to the ulna. (B) The same foreleg at one year of age. The heterotopic bone had resorbed and the size and density of the ulna are reduced. (Riser WH, Parkes LJ, Shirer BS: Canine craniomandibular osteopathy. J Am Vet Radiol Soc 8:23, 1967)](image)

![FIG. 54-3 Craniomandibular osteopathy in a 16-month-old Scottish terrier. The edges of the mandibles, although still radiopaque, have diminished in size. The edges are well defined and smooth. The tympanic bullae are still enlarged. (Riser WH, Parkes LJ, Shirer BS: Canine craniomandibular osteopathy. J Am vet Radiol Soc 8:23, 1967)](image)

LABORATORY SIGNS
In blood specimens of West Highland white terriers studied, no abnormalities were observed in the number of erythrocytes or leukocytes. The hemoglobin, calcium, phosphorus, and alkaline phosphatase determinations were also normal. Bacterial growth was negative in 26 blood culture inoculations from four West Highland white terriers.(14)

GROSS ANATOMICAL APPEARANCES OF THE SKULL LESIONS
Necropsy material from eight dogs with CMO has been studied.(14) The dogs ranged in age from 4 to 13 months.

In one dog, 4 months of age, early exostoses were observed radiographically in the midshaft of the femur at the time the leg was examined for lameness. At necropsy, additional exostoses were also described in the mandible and frontal bones.

The remaining seven dogs were put to sleep because of their inability to eat as a result of the closed jaws. At necropsy, it was not possible to force the upper and lower jaws apart even as much as 1 cm. Symmetric bony enlargements were palpated along both mandibles. The tympanic bullae in each of these dogs were also enlarged. The enlargements, however, were hidden by the long hair and whiskers that covered the head and could not be seen with palpation.

At necropsy, the heads of the dogs were sawed sagittally; half was macerated by boiling and the other half was decalcified
and prepared for histologic sectioning.

In the macerated halves, the new-bone formation was confined primarily to the mandibles and tympanic bullae. In four dogs, the frontal and parietal bones were thickened. This thickening had not noticeably disrupted the contour of the external surfaces (outer table) or the inner table of the calvarium. The diploe was filled by newly formed trabecular bone. The mandible and tympanic bullae were enlarged irregularly. The lateral, medial, and ventral surfaces of the body of the mandible were expanded bilaterally from the area of the middle mental foramen caudally, with the greatest change occurring in the region of the angular process. The mandible was not enlarged noticeably around the alveolar and interalveolar spaces.

New-bone formation filled and expanded the tympanic bullae. These cavities, which in small dogs are normally about 1.5 cm in diameter, expanded to 4-5 cm in diameter and were filled with new bone. In most instances each bulla fused with the nearby angular process of the mandible. This obstructed the movement of the mandible.

The new-bone surfaces were rough and irregular. After the skull was dried following maceration, the mineral crumbled away when gouged with a pointed instrument. No lesions were found in the oral cavity or regional lymph nodes.

Microscopically, the osseous lesions of CMO are the result of a complex and varied series of pathologic changes in selected areas of the mandible, the tympany bullae, to a lesser degree the bones of the calvarium, and on rare occasions, the long bones (Fig. 54-2, A).(14)

The changes are as follows:
1. Osteoclastic resorption of existing lamellar bone (Fig. 54-4).
2. Replacement of lamellar bone by a primitive coarse type bone that expands beyond the normal periosteal boundaries (Figs. 54-5, and 54-6).
3. Loss of normal bone marrow spaces between the coarse trabecular bone and replacement of the marrow by a highly vascular fibrous-type stroma (Figs. 54-5, 54-7, and 54-8).
4. Invasion of inflammatory cells at the periphery of the lymphocytes, plasma cells, and neutrophils. Connective tissue and muscle fibers are destroyed at the irregular boundaries of the newly proliferated bone.
5. Formation of new coarse trabecular bone with a "mosaic" pattern of irregular cement lines indicating the sporadic and rapid deposit and resorption of the abnormal bone (Figs. 54-7 and 54-9).

FIG. 54-4 Craniomandibular osteopathy. Histologic section of a portion of the mandible. The osteoclastic destruction of the lamellar bone is accompanied by a heavy invasion of inflammatory cells (neutrophils, lymphocytes, and plasma cells). (Riser WH, Parkes LJ, Shirer BS: Canine craniomandibular osteopathy. J Am Vet Radiol 80c 8:23, 1967)

FIG. 54-5 Craniomandibular osteopathy in a 7-month-old West Highland white terrier; there is marked new-bone formation in the region of the tympanic bullae and the parietal crest. Distinct layers of new bone may be seen in the diploe of the cranium. (Riser WH, Parkes LJ, Shirer By: Canine craniomandibular osteopathy. J Am vet Radiol 80c 8 23, 1967)

In some instances the pathologic changes in the mandible resemble certain forms of osteosarcoma, hyperparathyroidism, or callus formation.

The abnormal trabecular bone was coarse and dense. It occupied most of the space in an affected area. The size of the trabeculae was increased by the apposition of new layers of mineralized bone added to the existing surfaces in an unorderly manner. Thin basophilic seams (cement lines) marked the border where each new layer was formed. In some areas, the immature bone surrounded and encircled the older mature lamellar bone with its parallel bundles of collagen. In other areas, the lamellar bone had been destroyed and removed when the new woven immature bone was deposited as a replacement. The new bone with its low mineral content was held together by coarse fibers arranged in an uneven, interlacing fashion. The bone stained unevenly dark, with areas of patchy basophilia. Histologically, such construction gives the stained bone tissue a mosaic pattern as if the new bone were plastered upon the old. The cement lines separating the layers that had been constructed previously had undergone alternating phases of resorption and acceleration (Figs. 54-7 to 54-9).(14)

Occasionally, cartilage was present in areas of newbone formation, although it was not observed in every instance. Foci of new bone extended beyond the periosteum: the mandible and tympanic bullae often fused into a solid mass that restricted movement of the jaws. As endochondral growth ceased, abnormal bone formation slowed, regressed, and, in many instances, receded. Subsequently, the primitive bone was replaced by more mature bone of the lamellar type. Radiographically, at this time the bone borders appeared to become straight and regular as the edges were absorbed (Fig. 54-3). Histologically, the staining quality of the bone was improved as lamellar bone replaced the primitive bone. The number of inflammatory cells decreased markedly in the lesions that had been present for some time.(14)

ETIOLOGY
The etiology of CMO is unknown. The pathogenesis, however, seems reproducible. Osteoclastic resorption of mandibular lamellar bone occurs primarily, followed by production of woven bone. The woven bone is sufficiently proliferative to push beyond the normal periosteum. Similar new-bone production may fill the medullary cavity. According to Pool and Leighton, (11) "Irregular episodes of bone resorption and osteoblastic proliferation result in a characteristic histologic picture of new fibrous bone deposition separated by blue lines representing more mature bone." The end result may be maturation of the fibrous bone that remains permanently, although rarely the dog may show a spontaneous reversal of the disease that returns the bone to its normal condition.
TREATMENT
Treatment is symptomatic. Most animals can be made comfortable using aspirin or corticosteroids; however, treatment does not result in cure. Most animals stabilize with impaired mouth function but are capable of maintaining normal nutritional status. Surgical intervention to reduce bony mass or to increase temporomandibular joint range of motion has not resulted in improvement.(11)

REFERENCES

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