METAPHYSEAL CHONDRODYSPLASIA IN A TIBETIAN MASTIFF DOG

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Metaphyseal chondrodysplasia, a relatively rare skeletal abnormality was diagnosed in a 16 week old male Tibetian Mastiff dog. Clinically, the dog showed bowing of fore limbs, palmagrade posture and severe lameness. Radiographically, a large irregular triangular radiolucent zone was evident at the distal metaphysis of radius and ulna, which extended deep into the distal third of diaphysis. Plasma biochemical changes included hypercalcaemia, hyperphosphataemia and hypovitaminosis-D3. Treatment was given with vitamin D3 and A. Clinically moderate improvement was noticed after two and half months of treatment. After one year, radiograph showed complete resolution of triangular cartilagenous lesion present in the distal third of diaphysis of radius and ulna.

Key words: Dog, Metaphyseal chondrodysplasia, Tibetan Mastiff.

Introduction
Metaphyseal chondrodysplasia is a group of conditions in which abnormalities of the metaphysis predominate, with essentially normal epiphysis, skull and trunk (Jezyk, 1985). Though the exact etiology is unknown, it is said to be a diet related disorder seen in large breed dogs and associated with metaphyseal dysplasia leading to retention of endochondral cartilage (Stogdale, 1979). The present case report describes metaphyseal chondrodysplasia in a young Tibetian mastiff dog, which was probably due to imbalance in Ca: P ratio and / or vitamin D3 deficiency.

Case history and Clinical examination
A four months old male Tibetian Mastiff dog weighing 12 kg was presented to polyclinic with the history of bilateral bowing of fore limbs and difficulty in walking for about two months. The dog was fed on diet containing 250 ml milk three times a day and daliya (grounded wheat) twice a day. There was no history of meat, bone meal and egg feeding. The dog was vaccinated against canine distemper, infectious canine hepatitis and leptospira diseases and dewormed against gastrointestinal parasites. Clinical examination revealed severe lameness, palmagrade posture, cranial deviation of both carpal joints, severely enlarged distal radius - ulna metaphysis, pain on palpation of the enlarged metaphysis and hind limb weakness with cow hock posture.

Diagnosis and Treatment
Orthogonal radiographic views of the affected forelimbs (radius-ulna) revealed the presence of a triangular irregular radiolucent zone in the distal metaphysis, which extended deep into the distal diaphysis of radius-ulna (Fig.1A&B). Thickened physes, enlarged distal metaphyseal border, cranial bowing of the distal metaphysis and thinning of cortices of diaphysis were also noted. Haemoglobin, packed cell volume and differential leukocyte count were within the normal range. Plasma levels of different biochemical and hormone parameters estimated are given in table no. 1. On the basis of the radiographic findings and biochemical findings, the case was diagnosed as metaphyseal chondrodysplasia.

Treatment consisted of injection of vitamin-A (Arovit; Piramal Health Care) 2 ml (100,000 i.u.) intramuscularly and vitamin - D3 (Arachitol; Duphar) 1 ml (3, 00,000 I.U. or 7.5 mg) intramuscularly at weekly interval for three months. Anti-inflammatory drug, Nimesulide (Nise; Dr. Reddy Labs) 50 mg orally was advised twice a day for the initial 3 days to reduce the pain. The owner was also advised to feed a balanced diet containing adequate milk, egg and meat with bone daily and restrict the movement of dog. After 15 days of the treatment, there was no improvement clinically, but improvement was noticed in biochemical parameters (table no. 1). After two and a half months of treatment, moderate improvement in the condition of the dog was seen in terms of reduction in lameness and improvement in posture. After one year, marked improvement was noticed with only a slight cranial bowing in the radii of both forelimbs. Radiographic examination revealed resolution of the large triangular cartilagenous lesion present in the distal metaphysis of...
radius-ulna, with a slight cranial bowing of distal third of the radial diaphysis (Fig. 2A&B).

Fig. 1: Radiograph at 4 months shows the presence of a large triangular irregular radiolucent zone at the distal metaphysis of radius and ulna (arrows) along with flaring and broadening of the distal metaphyseal border; (A) Cranio-caudal view, and (B) Medio-lateral view.

Fig. 2: Radiograph taken at one year of age shows complete resolution of cartilagenous lesion (arrow) with the normal distal metaphysis and epiphysis; (A) Cranio-caudal view, and (B) Medio-lateral view.

Table-1: Plasma biochemical and hormone analysis on day 0 and day 15

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day 0</th>
<th>Day 15</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium (mmol/L)</td>
<td>3.78</td>
<td>2.92</td>
<td>2.25-2.83 (2.55±0.15)*</td>
</tr>
<tr>
<td>Phosphorus (mmol/L)</td>
<td>3.93</td>
<td>2.78</td>
<td>0.84-2.00 (1.39±0.29)*</td>
</tr>
<tr>
<td>Ca : P</td>
<td>0.96</td>
<td>1.04</td>
<td>1.2-2.00 *</td>
</tr>
<tr>
<td>ALP (U/L)</td>
<td>206.82</td>
<td>95.693</td>
<td>20 – 156 (66±36)*</td>
</tr>
<tr>
<td>Total Protein (g/L)</td>
<td>62.50</td>
<td>72.00</td>
<td>54-71 (61±5.2)*</td>
</tr>
<tr>
<td>iPTH (pg/mL)</td>
<td>7.716</td>
<td>1.26</td>
<td>1.27-2.54 (2.04±0.50) †</td>
</tr>
<tr>
<td>Calcitonin (pg/mL)</td>
<td>8.308</td>
<td>5.23</td>
<td>8.71-38.41 (19.12±8.46) †</td>
</tr>
<tr>
<td>Osteocalcin (ng/mL)</td>
<td>1.086</td>
<td>0.75</td>
<td>1.52-98.25 (43.25±25.00) †</td>
</tr>
<tr>
<td>Vitamin-D₃ (nmol/L)</td>
<td>90.31</td>
<td>214.54</td>
<td>160-330 (235.65±31.96) †</td>
</tr>
</tbody>
</table>

Ca- Calcium, P- Phosphorus, ALP- Alkaline phosphatase, iPTH- Intact parathyroid hormone
* Kaneko and others (1997), # Bennett (1976)
† Plasma value recorded in normal growing dogs (Kushwaha, 2003)

Discussion

Metaphyseal chondrodysplasia is a syndrome in which disorders predominate in the metaphysis (Jezyk, 1985). The radiographic finding in the present case was the presence of a large triangular radiolucent zone in the distal metaphysis with an irregular border extending deep into the metaphysis. This might have occurred as a result of improper mineralization of the cartilage in the metaphyseal region. Similar finding was also observed by Jezyk (1985) in a young viszla dog. Johnson et al. (1988) reported that the metaphyseal chondrodysplasia and copper deficiency (Baxter and Van Wyk, 1953) diseases resemble to that of rickets. Bingel and Sande (1994) have reported chondrodysplasia in five disproportionate short limbed, short trunkecd (dwarf) Great Pyreness pups.

In the present case, the clinical signs reported were similar to that of rickets (Bennett, 1976; Malik et al., 1997; Sharma, 2002) and might be due to the failure of mineralization at the physeal zone and metaphyseal area resulting in thickening of physis by a zone of proliferating cartilagenous cells that deforms and bow.
because it can not support the body weight (Rosol and Capen, 1997).

The levels of plasma calcium and phosphorus were higher and their ratio was less (0.96: 1) than the reference value (1.2:1 - 2: 1; Bennett, 1976). This disturbed Ca : P ratio might be one of the cause of failure of mineralization of the osteoid matrix, as the disturbed Ca : P ratio tend to reduce the bioavailability of calcium. Unlike man, the dog is dependent on dietary sources of vitamin D, since cutaneous synthesis of the vitamin is inadequate, due to the low concentration of the vitamin D precursor 7-de-hydrocholesterol in the skin of carnivores (How et al., 1996; Hazewinkel et al., 1988). In the present case, a decreased level of vitamin D3 was found in the plasma of the affected dog, which might be due to the diet deficient in vitamin D3. The present condition was probably due to the hypercalcaemia, hyperphosphataemia (low Ca : P ratio) and low level of vitamin D3 in the plasma. However, Pitt (1981) reported that metaphyseal chondrodysplasia and copper deficiency diseases have no abnormalities in calcium, phosphorus or vitamin D metabolism.

Hypercalcaemia may result from an imbalance of calcium released from bone, calcium excretion by the kidney and / or calcium absorption from the intestinal tract (Rosol and Capen, 1992). Pathogenic mechanisms of hypercalcaemia include excessive secretion of parathyroid hormone (PTH), excessive absorption of calcium from the intestine due to the increased vitamin D, excessive bone resorption, reduced renal excretion of calcium and increased total serum calcium due to increased protein bound and/or complexed calcium with normal ionized calcium levels (Rosol and Capen, 1997). The excessive dietary phosphorus with vitamin- D and A deficiency causes hypocalcaemia even if there is adequate calcium in the diet (Miller, 1969). In the present case, the hypercalcaemia seems to have occurred due to either high plasma iPTH or reduced excretion of the calcium by kidney, whereas hyperphosphataemia might be due to the excessive intake of phosphorus present in the daliya i.e. grounded wheat (Miller, 1969).

Parathyroid hormone is the principal hormone involved in the regulation of blood calcium in mammals. An important action of PTH on bone is to mobilize calcium from skeletal reserves into extracellular fluids (Canalis et al., 1994). Calcitonin hormone’s main action is to inhibit bone resorption from blockade of osteoblastic osteolysis. However, in the present case, the increased level of PTH and decreased level of calcitonin along with hypercalcaemia could not be explained.

After treatment the vitamin D3 level in plasma became normal (214.54 nmol/L), whereas Ca : P ratio approached towards low normal range ( 1.04 : 1) in 15 days. The total protein increased from 6.25 g/dl to 7.2 g/dl. From the present case report, it can be concluded that metaphyseal chondrodysplasia may be associated with hypercalcaemia, hyperphosphataemia, and hypovitaminosis-D, which responded satisfactorily to vitamin-D3 and A therapy.

References
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