Brief Reports

Renal Osteodystrophy with Multiple Osteolytic Lesions in the Skull

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Multiple osteopathic lesions in the skull are classically described in histiocytosis (1); the other uncommon causes include tubercular osteomyelitis, sarcoidosis, secondaries from neoplasms and multiple myeloma(1,2). We report the case of an 11-yr-old girl with failure to thrive, whose skull roentgenogram showed multiple lytic lesions.

Case Report

An 11-yr-old girl presented to Kalawati Saran Children's Hospital, New Delhi, with a history of failure to thrive for the last four years. Increasing pallor, poor appetite, generalized bone pains and inability to walk were present for the past one year. There was no history of fever, cough, urinary or bowel complaints.

On examination, she was a pale and emaciated child who resisted physical examination; her weight was 11.5 kg (less than 3rd percentile for age), and height was 107 cm (less than 3rd percentile for age). There was Grade III clubbing. There was no facial puffiness or pedal edema. The respiration was acidotic and blood pressure 120/90 mm Hg. She had generalized bone tenderness with frontal bossing, rachitic rosary, double malleoli and fixed flexion (30°) deformity of both hip joints. Examination of abdomen revealed a soft, nontender hepatomegaly and splenomegaly of 2.5 cm each. The respiratory, cardiovascular and nervous systems were essentially normal.

Investigations showed a hemoglobin level of 5.4 g/dl; normocytic normochromic anemia and ESR of 49 mm. Peripheral smear examination showed normal differential count with no abnormal cells. Urinary albumin was present in traces but there was no abnormality on microscopic examination. Urinary specific gravity ranged from 1004 to 1010. The blood levels of urea were 126 mg/dl, and serum creatinine 1.8 mg/dl, calcium 5.3 mg/dl (ionized 2.3 mg/dl), phosphate 4.7 mg/dl, alkaline phosphates 90 King Armstrong units/dl, sodium 141 mEq/L and potassium 4.6 mEq/L. Arterial blood gas analysis revealed metabolic acidosis with a negative base excess of 11.6. The glomerular filtration rate was 29 ml/min/1.73 m^2. A

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mid-molecule serum parathyroid hormone assay revealed serum levels of 160 ng/dl (normal up to 27 ng/dl).

Skeletal survey showed generalized osteoporosis, widening and fraying of metaphysis, Looser's zones with multiple fractures, bilateral slipped femoral epiphysis with avascular necrosis of head of femur and acrosteolysis of terminal phalanges; these features were suggestive of renal osteodystrophy. The skull roentgenogram showed multiple well-defined round to oval osteopathic areas (*Fig.*).

An ultrasound examination of the abdomen revealed bilateral contracted kidneys, with thin cortex, left hydroureter with hydropelvis. The micturating cystourethrogram was normal. The bone marrow was hypocellular with presence of giant cells (osteoclasts). A diethylene triamine penta acetic acid (99 mTcDTPA) nuclear scan revealed a small right kidney with markedly impaired glomerular functions, moderate impairment of function in the left kidney, left hudroureter and pelvicalyceal dilatation.

A diagnosis of chronic renal failure with renal osteodystrophy with secondary hyperparathyroidism was made. The patient was treated with oral vitamin D3 (calcirol), calcium gluconate (1 g/day) and alum gel (1 tsf with every meal). Acidosis was managed with oral sodabicarb (2mEq/kg/day). Proteins were restricted to 1.25 g/kg (essentially high biological value animal protein). A minimum caloric intake of 100 Cal/kg/day was ensured, contributed mainly by carbohydrates and fats. Foods rich in phosphates and potassium were also restricted.

On follow up, three months later, her weight was 13 kg and there was complete recovery from bone pains. Radiologically, bone density increased, the fractures healed completely and the osteopathic skull lesions also showed remarkable healing. Blood urea was 108 mg/dl and serum creatinine 1.6 mg/dl.

Discussion

Most patients of renal osteodystrophy

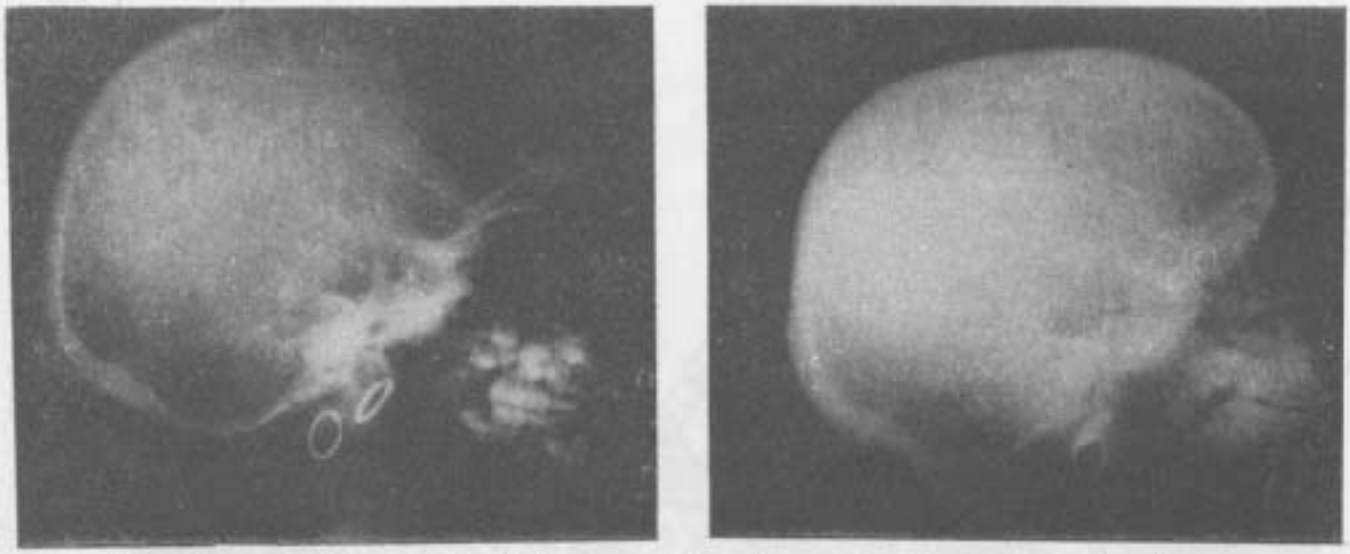

Fig. X-ray skull showing multiple osteopathic lesions and decreased bone density.

(ROD) exhibit generalized diminished bone density in the skull, which is of a spotty and non-homogeneous character. In the calvarium this spotty bone destruction may produce a granular mottled appearance often associated with small, focal, scattered radiolucencies or a ground glass appearance with poor definition of the Table(3). In the present case, the skull *X*-ray showed well defined multiple osteopathic lesions (*Fig.*). We could not come across any case with multiple such well defined lytic lesions in the skull due to ROD in the available literature; which prompted us to report this case.

Renal osteodystrophy or uremic bone disease was described as early as 1883 by Lucas and independently again in 1890 by Goodhart(4). It is still not established when in the course of uremia, secondary hyperparathyroidism sets in. Rodson *et al.*(5) found that ROD occurred once the glomerular filtration rates were less than 30 ml/min/1.73 m^2. It occurs in long standing CRF and is probably the reason it is not commonly encountered in children. Recent estimates of the clinical and radiological manifestations of bone disease in children with CRF ranges from 45 to 57%(6-8). It is estimated that at least 30% of the bone mineral must be lost before the loss can be detected by routine radiography(3).

The cause of chronic renal failure in this child appears to be bilateral vesicoureteric reflux (VUR) and consequent renal damage (reflux nephropathy), as the radionuclide scan showed pelvicalyceal dilatation and hydroureter on the left side. A normal micturating cystourethrogram was not unusual to find, because spontaneous disappearance of VUR is known to occur in over 80% of children with age(9).

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REFERENCES

1. Silverman Frederic N. The skull. *In:* Caffey's Pediatric *X*-ray Diagnosis, 8th Edn. Eds Silverman, Chicago, Year Book Medical Publishers, 1985, pp 71-83.
2. Katariya S, Thapa BR, Kumar L, Chand G. Tuberculous osteopathic lesions of the skull mimicking neuroblastoma. Indian J Pediatr 1988, 55: 149-152.
3. Shapiro R. Radiologic aspects of renal osteodystrophy. Radiol Clin North Am 1972, 10: 557-568.
4. Piel CF, Roof BS. Skeletal growth disturbances in renal diseases. *In:* Pediatric Nephrology. Eds. Rubin MI, Barrat MT, Baltimore, Williams and Wilkins, 1975, pp 740-741.
5. Hodson EM, Shaw PF, Dunstan CR, Rosenberg AR, Roy L. Growth retardation and renal osteodystrophy in children with chronic renal failure. J Pediatr 1983, 103: 735-740.
6. Potter DE, Wilson CJ, Ozonoff MB. Hypoparathyroid bone disease in children undergoing long term hemodialysis, treatment with Vitamin D. J Pediatr 1974, 85: 60-66.
7. Norman ME, Mazur AT, Borden S, *et al.* Early diagnosis of juvenile renal osteodystrophy. J Pediatr 1980, 97: 226-232.
8. Parfitt AM. Clinical and radiographic manifestations of renal osteodystrophy. *In:* Calcium Metabolism in Renal Failure and Nephrolithiasis, 1st edn. Ed. Dawin DS, New York, John Wiley and Sons, 1977, pp 145-239.

9. Edwards D, Normand ICS, Prescod N, Smellie JM. Disappearance of vesicoureteric reflux during long term prophylaxis of urinary tract infection in children. Br Med J 1977, 2: 285-288.

Pulmonary Aplasia: A CT Appearance

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The spectrum of pulmonary congenital anomalies due to developmental arrest of the lungs ranges from pulmonary agencies which signifies complete absence of the tracheo-bronchial tree, the pulmonary parenchyma and vasculature to pulmonary hypoplasia where the gross morphology of the lung is unremarkable but number and/or size of airways, alveoli and vessels is decreased. The spectrum is completed by pulmonary aplasia where a small rudimentary bronchus which ends in a blind pouch is present without any pulmonary parenchyma or vasculature(1). However, pulmonary agencies and aplasia have often been unfairly clubbed together because of clinical convenience and diagnostic difficulty(2). This arrangement overlooks the clinical significance of pulmonary aplasia where the blind bronchial pouch can often be the focus of repeated infections which adversely affects the prognosis of such patients(3). Recent advances in imaging have overcome the problems posed by unpleasant invasive techniques necessary for diagnosis(4). So far, probably less than a dozen reports of the two conditions have been published from the Indian subcontinent(5). The rarity of reports in Indian literature prompted the present description of a 10-year-old boy with aplasia of the left lung diagnosed with the help of CT thorax.

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Case Report

An asymtomatic 10-year-old boy was referred to our Institute for a flattened left hemithorax detected on routine school medical check-up. He was the first born of a non consanguinous marriage with uneventful antenatal and postnatal periods. He had normal milestones and had received complete immunization. There was no family history of congenital anomalies. Chest examination revealed a mediastinal shift to the left along with other features suggestive of left sided volume loss. On auscultation, normal vesicular breath sounds were audible on the right side but were reduced in intensity on the left side. Heart sounds, though normal in character, were distant.

A review of chest roentgenograms demonstrated an opaque left hemithorax with ipsilateral shift of mediastinum and associated herniation or right lung (*Fig. 1*). Fibrebronchoscopy visualised a small 1.5 cm long left main bronchus ending in a