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Pathological Fractures of Long Bones in Nigerian Children and Adolescents

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Summary

Ebong, W. W. (**1977**). *Nigerian Journal of Paediatrics*, **5**(**1**) 16. **Pathological Fractures of Long Bones in Nigerian Children and Adolescents** Thirty cases of pathological fractures of long bones seen during a 12-month period in patients under twenty years of age were studied. Two-thirds of the subjects were patients with sickle cell disease and non-sicklers with severe nutritional rickets or chronic osteomyelitis. The femur was the commonest site of the fracture. The preponderance of pathological fractures in these three groups of patients is probably due to the high incidence of sickle cell disease, infections and malnutrition in the environment, as well as to delay in seeking medical care by patients.

THE commonest cause of pathological fracture of long bones in caucasian children and adolescents is simple bone cyst (Appley, 1968), while the commonest site of fracture in such cases is the humerus (Sharrad, 1971). The chance findings of pathological fractures involving the femoral shafts in some of our patients with sickle cell anaemia, and in others with severe nutritional rickets have prompted this prospective study of pathological fractures seen in the Orthopaedic Clinic, University College Hospital (UCH), Ibadan. This paper presents the pattern of the disease as seen during a one-year period in patients under twenty years of age.

Materials and Methods

A record of all cases of pathological fractures seen in the Orthopaedic Clinic between May 1st, 1974 and April 30th, 1975 was kept. Recorded information included age and sex of the patients, radiological features, underlying causes of the fractures, and response of the patients to treatment. The present analysis however concerns only patients under the age of 20 years.

Results

Thirty patients with pathological fractures of long bones in the age group under consideration were seen during the one-year period. They consisted of 16 males and 14 females giving a M:F ratio if 1.1 to 1. The ages of the patients ranged from 14 days to 19 years (mean 9.7 years).

Underlying pathology

In the Table is summarised the identifiable predisposing factors to pathological fractures in the 30 patients. It will be seen that in 20

patients (66.7 per cent) pathological fractures were associated with sickle cell disease, chronic osteomyelitis and severe nutritional rickets, the leading associated condition being sickle cell disease with nine (30 per cent) patients. Eight of the patients with sickle cell disease were homozygous S (HbS), while the ninth had haemoglobin SC. In five of the children with sickle cell disease, the pathological fractures were associated with bone infarcts and/or osteomyelitis, while the remaining four had steooporosis. Six of the pathological fractures (20 per cent) occurred in association with chronic osteomyelitis in non-sicklers, while five cases (16.7 per cent) occurred in association with severe nutritional rickets. The remaining 10 cases (33 per cent) were due to varying miscellaneous causes which included simple bone cysts (3), osteosarcoma (3), monostotic fibrous dysplasia (2), Burkitt's lymphoma (1) and osteogenesis imperfecta (1).

TABLE

Underlying Factors in Pathological Fractures of Long Bones in 30 Nigerian Children and Adolescents

Underlying Factor	Number of cases	Percentage
Sickle cell disease:	9	30
Hb S disease	8	
Hb S + C disease	1	
Chronic osteomyelitis (non-sicklers)	6	20
Nutritional rickets	5	16.7
Miscellaneous:	10	33.3
Simple bone cyst	3	
Osteosarcoma	3	
Monostotic fibrous dysplasia	2	
Burkitt's Lymphoma	1	
Osteogenesis imperfecta	1	
TOTAL	30	100

Analysis of the age and sex factors in relation to the pathological basis of the fractures revealed significant differences. The male to female ratios varied from 1:2 in patients with sickle cell disease to 2:1 and 3:2 in those with chronic osteomyelitis and nutritional rickets respectively. In the remaining 10 patients the number in each pathological group was too small for any meaningful analysis of the sex ratio. With respect to age patients with sickle cell disease ranged from 10 to 19 years (mean 13.4 years), while those with severe nutritional rickets ranged from 2 to 6 years (mean 4.4 years). Pathological fracture complicating chronic osteomyelitis in non-sicklers however occurred without preference to any particular age.

Sites of fractures

The femur was by far the commonest site of fracture, accounting for 23 (77 per cent) of the 30 cases. All the femoral fractures occurred in the shaft (Fig.1), except in one ten-year old girl in whom it occurred in the femoral neck (Fig.2). In eighteen patients, the femoral fractures were confined to one side. Both femoral shafts, one femoral shaft and both tibiae, and both femoral and tibiae, respectively, were fractured in 3 patients, all of whom had severe nutritional rickets. A fourteen-day old baby with osteogenesis imperfecta presented with fractures of both femoral shafts, as well as fractures of both tibiae, humeri and clavicles. In 6 patients fractures occurred in tibiae, while in one other patient, the humerus was the site of fracture. The later patient had chronic osteomyelitis of the humerus, but no haemoglobinopathy.

Radiological Features

Plain radiographs were diagnostic of the underlying pathology in 21 (70 per cent) of the cases. Diagnostic features included periosteal new bone formation in cases of osteomyelitis, and features of florid rickets such as deep growth discs and cupping of the metaphyses. However, in some cases of severe nutritional rickets with gross demineralisation, it was often difficult on radiological grounds alone to differentiate these from cases of sickle cell disease with severe osteoporosis.

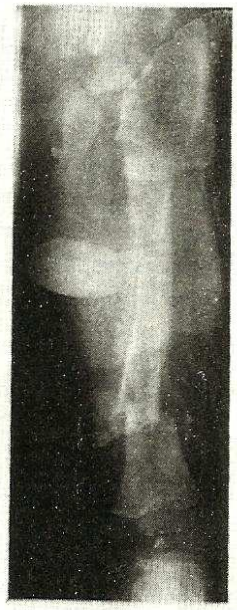

Fig. 1. Radiograph of a fracture of the distal third of the left femur in a patient with Hb S and salmonella osteomyelitis. Note the extensive diaphysical infarction, longitudinal intracortical fissures ("bone-in-bone" appearance) and excessive involucrum formation.

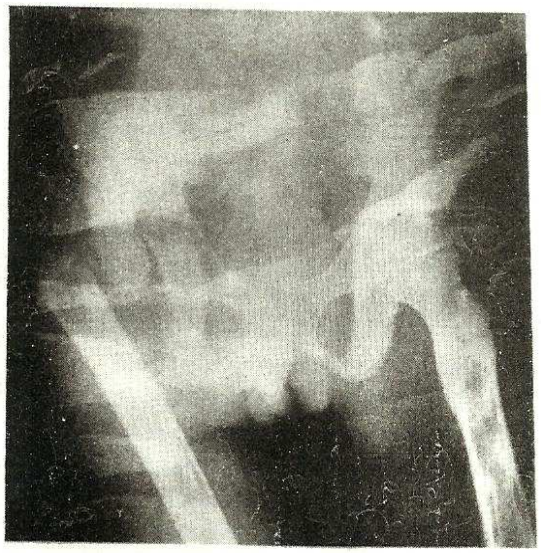

Fig. 2. Radiograph of a right femoral neck fracture in a patient with Hb S showing infarction of the right femoral neck and both femoral shafts.

Treatment and Outcome

Patients with pathological fractures complicating sickle cell disease were treated with plaster of Paris casts till the fractures united. They were all given folic acid (5 mg) daily and Daraprim (12.5-25 mg) weekly; blood transfussion was given to correct associated anaemia. Patients with sickle cell disease whose fractures were due to underlying osteomyelitis received chloramphenicol and penicillin as well. Most of these fractures united within the expected time for the particular bone for the age group. A few cases of delayed union were encountered, usually in patients whose fractures were due primarily to severe osteoporosis.

Patients with fractures due to severe nutritional rickets were immobilised in plaster of Paris casts and given Vitamin D 5,000 units orally daily, as well as calcium lactate, multivitamin supplements, Casilan, and a high protein diet. The fractures in most of the patients united within the expected time, but in one instance, it took up to five months for the child to start walking again.

Non-sicklers with pathological fractures complicating chronic osteomyelitis were also treated with plaster of Paris casts, where indicated, windows were left in the casts for dressing. Appropriate antibiotics were also given, but delayed union was common in this group.

Discussion

The occurrence of pathological fracture in sickle cell disease is well documented (Middlemiss, 1958; Diggs, 1967; Bohrer, 1971). Most authors (Middlemiss, 1958; Diggs, 1967) have reported that pathological fractures in this condition are commonly assosiated with osteoporosis but rarely with bone infarcts, because the pain caused by the infarct is thought to prevent the use of the affected limb. By contrast, 55.5 per cent of pathological fractures associated with sickle cell disease in the present

series were associated with bone infarct and/or osteomyelitis, while the remaining 44.5 per cent of cases had associated osteoporosis. Bohrer (1971), noting a high incidence of pathological fracture complicating bone infarct in sickle cell diseasea mong Nigerians, has suggested that this might be due to the fact that the patients were stoical. It should be noted however that the sickle cell gene occurs in 25 per cent and sickle cell disease in 3 per cent of our population (Effiong, 1975); the frequent association of pathological fracture with sickle cell disease in the present series may therefore be due to the high incidence of the sickle cell gene in the community.

The high incidence of pathological fracture in chronic osteoyelitis and nutritional rickets may also be explained by the high prevalence of these conditions in the environment; however, late presentation in hospital, and trivial injury following use of the limb are probably also contributory factors. Chronic osteomyelitis is very common in the tropics (British Medical Journal, 1967; De Voogt, 1967; Huckstep, 1968; Lancet, 1970). Most of these patients present late, and a few of them develop pathological fractures following minor trauma.

Florid nutritional rickets is also common in the tropics despite the abundant sunshine (Feldman, 1950; Lovett–Campbel, 1952; Wright, 1952; Walker, *et al.*, 1956; Antia, 1970) and this has been partly attributed to the purdah system which prevents children from receiving adequate sunshine (Antia, 1970). Malnutrition may also be an important factor in some cases. In the present series, for instance, three cases showed very thin bone cortices and ghost-like appearance of the bones, thus suggesting that besides demineralistion, underlying malnutrition may contribute to general bone wasting.

REFERENCES

Antia, A.U. (1970). Observations on nutritional rickets in childhood. West Afr. Med. J. **19,** 169-172.

Appley, A. G. (1968). A system of orthopaedics and fractures, 3r edition p 391. Butterworths, London.

Bohrer, S.P. (1971). Fracture complicating bone infarcts and, /or osteomyelitis in sickle-cell disease. Clin. Radiol., **22** 83-88.

British Medical Journal (1967). Changed character of osteomy elitis. Brit. Med. J., **3**. 255-256.

De Voogt, H. J. (1967). Osteomyelitis in the tropics. Trop. Geogr. Med., **19**, 352-356.

Diggs, L. W. (1967). Bone and joint lesions in sickle-cell disease. Clin. Orthop., **52**, 199-143.

Effiong, C. E. (1975). Sickle cell disease in childhood, in:- Isaacs-sodeye, A. (1975). Sickle cell disease, a hand book for the general clinician, 1st edition p 16. Claxton Press, Ibadan.

Feldman, N. (1950). Infantile rickets: its occurrence in non-europeans in Johannesburgh. S. Afric. Med. J. **24** 1053-1056.

Huckstep, R. L. (1968). The management of osteomyelitis in East Africa. E. Afr. Med. J., **45**, 429-462.

Lancet (1970). Chronic osteomyelitis. Lancet, *2*, 1235.

Lovett-Campbell, A. C. (1952). Clinical rickets in the tropics Trans. Roy. Soc. trop. Hyg., **46,** 107.

Middlemiss, J. H. (1958). Sickle cell anaemia. J. Fac. Radiol., **9,** 16-24.

Sharrard, W. J. W. (1971). Paediatric orthopaedics and fractures, 1st edition p 1060. Blackwell, London.

Walker, A. R. P., Falcke, H. C., Nestadt, A. and Cohen, H. (1956). Rickets in the tropics. J. Trop. Ped., **2**, 169-170.

Wright, E. J. (1952). Clinical rickets in the tropics. Trans. Roy. Soc. trop. Med. Hyg., **46,** 104-106.