HEREDITARY MULTIPLE EXOSTOSIS

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The earliest reference to a family with hereditary multiple exostosis was by Boyer in 1814. A more detailed description appeared in the Guy’s Hospital Reports in 1825. By the turn of the century all the important clinical features of the disease had been described (Bessel-Hagen 1891). Innumerable case reports have appeared since, and the pathology of the cartilage-capped exostoses has been described in detail (Jaffe 1943).

Nevertheless, the evolution of these curious lesions, their unusual distribution and the associated abnormalities of growth are still incompletely defined and largely unexplained. A statistical analysis of 1,124 cases collected from the literature by Stocks and Barrington in 1925 was marred by inaccuracies in most of the earlier papers upon which they drew. Yet in the absence of sufficient evidence to the contrary their conclusions have gradually become entrenched in the description of the disease.

The clinical, radiological, pathological and genetic characteristics have been re-examined in a study of this disease at the Royal National Orthopaedic Hospital and the Hospital for Sick Children, Great Ormond Street. The abnormalities of growth were described in a previous paper (Solomon 1961). The present paper deals with the development and distribution of the cartilage-capped exostoses and the complications associated with these lesions.

MATERIAL AND METHODS

All available records at the Hospital for Sick Children and the Royal National Orthopaedic Hospital were collected for study if indexed under any of the following headings: hereditary multiple exostosis, multiple exostosis, diaphysial aclasis, exostosis, osteochondroma, ecchondroma, enchondroma, chondroma, chondromatosis, dyschondroplasia, Ollier’s disease, chondrosarcoma. In most instances an examination of the case notes and the available radiographs was sufficient to separate those with true multiple exostosis (diaphysial aclasis) from the others. Doubtful cases were not included until they had been examined by the author.

Records of seventy-eight patients were found. Thirteen of these patients could not be traced; a further nine declined to attend for examination but completed questionnaires.

The remaining fifty-six patients formed the nucleus of this study. Four of them have died, one as a result of this disease; the other fifty-two were examined in detail, clinically and radiologically. In each instance earlier radiographs were available for comparison and often the lesions could be traced from their earliest appearance to the present time. When possible morbid anatomical and histological examinations of the lesions were also carried out.

Fig. 1
Radiograph of a sessile exostosis. End-on projection gives the appearance of vacuolation.
In addition to the patients forty relatives were examined clinically and radiologically and another forty-four were examined clinically but not radiologically. Seventy-six of these subjects, in whom anthropometric data were obtained, formed the basis of a previous study on bone growth in diaphysial aclasis (Solomon 1961).

**Differentiation from dyschondroplasia (Ollier’s disease; enchondromatosis)**—Most of the older case descriptions of multiple exostosis include examples of dyschondroplasia or multiple enchondromatosis. Indeed, Stocks and Barrington (1925) in their exhaustive review of the literature were led to conclude that “enchondromata may occur in some members of a family and exostoses in others, and any kind of admixture of the two disorders in families or cross-inheritance between them seems to be possible.”

Although there are superficial similarities, particularly in the deformities of the forearms and ankles, it is now well known that these are two separate and distinct conditions with different clinical, radiological and genetic features.

In multiple exostosis there is usually a family history; in dyschondroplasia never. The bony spurs or exostoses that do occur in dyschondroplasia are small and few in number; they are the least significant feature of a well recognised radiological pattern, which appears characteristically as a radiolucent streaking of the metaphysial region, due to retained columns of cartilage extending longitudinally from the epiphysial plate into the diaphysis. Central enchondromata are also common, particularly in the metacarpal bones and phalanges.

Figure 1 shows what has often been mistaken for an enchondroma in a case of multiple exostosis. This appearance is due to the radiographic projection giving an end-on view of a large sessile exostosis. Central, expanding chondromata do not occur in hereditary multiple exostosis.

**CLINICAL PRESENTATION**

The name hereditary multiple exostosis—preferred by some people for its descriptive simplicity, avoiding any reference to the basic pathology, as yet unknown, but hinted at in the term diaphysial aclasis—conveys at least two important features of the disease. It is a heritable disorder of the skeletal system; and its most striking manifestation are the numerous cartilage-capped exostoses which appear in different parts of the skeleton. The picture is completed by certain characteristic deformities resulting from deficient growth of the bones involved.

Over 80 per cent of the patients are discovered in the first decade of life, males and females being affected with equal frequency (Fig. 2). The skeletal distribution is shown in Figure 3. In fact the earliest lesions may be present at birth and are occasionally detected soon after by the searching hands of a parent herself afflicted by the disease. The mere presence of a bony lump, coupled with the knowledge that the disease is “in the family,” is sufficient reason for seeking medical advice in the vast majority of cases.

![Figure 2](image-url)

*Fig. 2*

Age and sex distribution of multiple exostosis in fifty-two index patients and twenty-four relatives examined radiologically. The histogram shows the age at which lesions were first discovered.
New mutants, on the other hand, are sometimes discovered accidentally in the course of investigations for some other illness. Occasionally it is one of the secondary deformities or the effect of pressure by an exostosis that first calls attention to the disease. Doubtless, too, some are affected so mildly as to remain unaware of their abnormality throughout life.

Lesions of the tibia and scapula are usually discovered first, simply because they are the most conspicuous sites in the child. A radiograph at this stage invariably shows early exostoses in many of the other bones as well. Soon these become palpable and visible as the picture of a generalised skeletal disorder unfolds. During the years of rapid growth the exostoses enlarge and pain in one or other of the lesions is common throughout this period.

The patient in an established case presents a characteristic appearance (Fig. 4). Numerous bony lumps may be seen and palpated as they jut into the soft tissues around the more actively growing parts of the endochondral skeleton. Juxta-epiphysial in origin, they are found typically at the ends of the tubular bones, the vertebral borders of the scapulae, the iliac crests and ribs.

Bone growth, too, is affected, giving rise to recognisable deformities in approximately three out of every four of these subjects (Table 1). The commonest are a shortness of stature, bowing of the radius with ulnar deviation of the wrist, subluxation of the humero-radial joint, valgus deformity of the knee and ankle, tibio-fibular synostosis and asymmetry of the pectoral and pelvic girdles. Of these, only the ankle deformity is usually bilateral and symmetrical; the others occur in haphazard association, but none ever in the absence of associated exostoses. The unmistakable radiological features are described below.

**Complications**—The natural course of the disease after the cessation of growth is unremarkable and there is little disturbance of health or functional activity in the usual case.

Despite the large number of exostoses it is surprising how seldom they interfere seriously with adjacent structures. In one case of this series a large pelvic exostosis caused urinary obstruction, renal failure and death: malposition of a pregnant uterus occurred in another. Intestinal obstruction and arterial aneurysms have been described, but were not encountered in the present series. An interstitial or subcutaneous bursa over an exostosis, pressure on a peripheral nerve or interference with tendons, though hardly ever serious, may call for removal of the exostosis. Fracture of a stalked exostosis occurred in two patients and necessitated removal of the loose fragments.

**Fig. 4**
The characteristic lesions in hereditary multiple exostosis.
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The most serious complication is malignant change in one or other of the cartilage-capped exostoses, which occurs in a significant percentage of cases. None of the fifty-six index patients in this series developed a chondrosarcoma, but one of the secondary cases did so and died after a hindquarter amputation. This subject is discussed in detail below.

The other three deaths were not attributable to this disease: one patient had a primary carcinoma of the lung, another died of cardiac failure and the third of bronchopneumonia.

TABLE I

INCIDENCE OF DEFORMITIES
(Fifty-two index patients and twenty-four affected relatives)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Multiple exostoses</th>
<th>Associated deformities</th>
<th>Percentage with deformities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>36</td>
<td>26</td>
<td>72</td>
</tr>
<tr>
<td>Female</td>
<td>40</td>
<td>30</td>
<td>75</td>
</tr>
<tr>
<td>Total</td>
<td>76</td>
<td>56</td>
<td>73.7</td>
</tr>
</tbody>
</table>

HEREDITY

Most of our knowledge about the hereditary characteristics of multiple exostosis is derived from Stocks and Barrington's (1925) report. From a statistical analysis of 1,124 recorded cases they concluded that the disease was inherited in 64 per cent of the cases, usually from an affected parent; that in one-quarter of the cases in which the mother transmitted the disease she remained unaffected herself; that the number of exostoses in individual cases increased from generation to generation; and that the sex ratio showed a 7:3 preponderance of males. Unfortunately many of the sources upon which this work was based included examples of dyschondroplasia. Furthermore, of the thirteen quoted instances of presumed transmission of the disease by an unaffected mother to her offspring, none was verified by physical and radiological examination.

Forty-six of the index patients in this present series were used in a study of the hereditary pattern of the disease, which is being reported in detail elsewhere. The ratio of males to females was 24:22. Sixty-three per cent of these patients had an affected parent. The disease appears to be determined by a single gene which always produces some detectable bony lesion in the heterozygote. No subject, either male or female, was found to have transmitted the disease without being affected.

The condition was inherited by about half the children of affected parents; there was no tendency for the lesions to increase in successive generations.

The type and distribution of the lesions showed no tendency to intrafamilial resemblance except in one family, where six of the affected members (two sisters each with two children) had exostoses predominantly on the bones of the hands, the more usual sites at the ends of the long bones being only mildly affected. It is possible that a different gene is concerned in this family. In the others only one mutant gene appears to be responsible.

EVOLUTION OF THE LESIONS IN MULTIPLE EXOSTOSIS

The characteristic lesion in multiple exostosis is not the pedunculated cortical exostosis (though this is often seen as well) but rather a diffuse, club-shaped thickening of the metaphysis, irregular in outline, heaped and cleft by innumerable bony excrescences or sessile exostoses. One or more of these may enlarge and project as a pedunculated mass, sometimes smooth in outline, sometimes cauliflower-like and of frightening dimension.
The initial deviation from normal bone growth is not related to any precipitating factor and may occur at any time from birth to the end of the growth period for that particular bone. Thus normal growth may precede and follow a starkly abnormal interlude; or new exostoses may be discovered at successive visits. However, in the present series there was no instance of a new exostosis arising after the cessation of growth.

The earliest lesion detectable radiologically is an asymmetrical or beaked overgrowth of the cortex immediately adjacent to the epiphysial plate (Fig. 5). Thereafter, as growth proceeds, one of two patterns may evolve. The juxtaepiphysial projection may be followed by normal growth, leaving an isolated exostosis jutting from the diaphysial shaft (Fig. 6); or the asymmetrical increase in width may be established as the new model for further growth, and new bone, irregularly heaped into a broad metaphysis, produces the characteristic club-shaped appearance of the bone end (Fig. 7). Which of these will result is totally unpredictable, nor is it certain that any potential site will develop an exostosis at all. The abnormality manifests itself as a completely isolated phenomenon each time it occurs, varying not only from patient to patient but from one bone to another in the same patient and even within the same bone at different times (Fig. 8).
Associated with the diaphysial thickening there is a retardation of bone growth which gives rise to a number of secondary deformities. Disproportionate shortening of the ulna causes bowing of the radius and ulnar deviation of the wrist or subluxation of the radio-humeral joint. Excessive shortening of the fibula causes valgus deformities of the ankle and knee. The other common deformities such as shortness of stature, asymmetry of the pectoral or pelvic girdle, coxa valga and shortness of the hands and feet are all accounted for by the same defect. The development of these abnormalities has been described in detail (Solomon 1961) and will not be discussed here.

As long ago as 1891 Bessel-Hagen suggested that the bone loses in longitudinal growth what it squanders in irregular transverse growth. It is still not known whether there is an exact relationship between these two phenomena. What is important is that both are part of a generalised abnormality of bone growth, and the exostoses are not neoplastic in the ordinary sense of the word.

In each exostosis is reproduced the structure of the bone from which it arises, an outer cortex and an inner marrow cavity continuous with the cavity of the bone. Thus the exostosis does not sit upon the underlying cortex, but arises in continuity with the parent bone.

Larger in size than the radiograph would suggest, the outer part of the mass consists of a cartilaginous covering or cap which surmounts the bony projection. Here the columnar arrangement of the cartilage cells of the epiphysial plate is crudely reproduced, and endochondral ossification proceeds slowly and irregularly until the epiphyses join (Fig. 9). After the cessation of growth most of the cartilage ossifies and the cap is thinned to a narrow lining which may be entirely absent in many places. Sometimes, however, it persists and may even increase in size.

Lorincz (1960) found that the urinary excretion of acid mucopolysaccharides (AMPS) was greatly increased in multiple exostosis and suggested that this was due to a disorder of connective tissue AMPS metabolism. This investigation has been repeated in eleven patients by the more accurate method of Di Ferrante and Rich (1956). The values in seven children between the ages of five and sixteen were all considerably above the mean but still within the normal range (the normal standards used were those of Teller, Burke, Rosevear and McKenzie (1962)). In the four adults the AMPS excretion was not raised at all. These findings may equally well be explained by the increased bulk of cartilage (which is the main source of AMPS) in children with multiple exostosis, and there is no need on the present evidence to postulate a metabolic abnormality.

Degeneration and calcification of the cartilaginous mass produces a characteristic radiological appearance which often causes unnecessary alarm (Fig. 10). The patchy density on the
Radiograph showing dense calcification of a cartilage-capped exostosis of the scapula. The uniform density of the tumour with its clear margins and the normal bone texture elsewhere suggest that this is a benign lesion.

Cross-section of the calcified exostosis shown in Figure 10. Confluent masses of calcified tissue have replaced the degenerating cartilage throughout the tumour.
radiograph may indicate a tumour of enormous size, suggesting the possibility of malignant change. However, unlike a chondrosarcoma, a benign exostosis shows a uniformity in the pattern, and the limits of the calcified area can be clearly and unequivocally traced at every point on the radiograph. Extensive areas of degenerative cartilage are replaced by the soft, chalky material, and calcium detritus may occur also in the bony part of the tumour (Fig. 11).

**MALIGNANT CHANGE**

The dividing line between "abnormal growth" and "neoplasia" in a cartilage-capped exostosis is hard to determine. Any increase in size after the normal period of growth always suggests the possibility of malignant change; but children, too, suffer this complication (Bennett and Berkheimer 1941) and here the point of departure from benign growth is imperceptible.

![Image 12](image12.png) ![Image 13](image13.png)

Figure 12—Radiograph showing a chondrosarcoma developing in a cartilage-capped exostosis. The variegated density, the poorly defined outline of the tumour and the signs of bone destruction strongly suggest malignancy.

Figure 13—Photomicrograph showing the features of a chondrosarcoma. Note the marked cellularity and the pleomorphism of the tissue.

Dahlin (1957), on the basis of 272 cases of osteochondroma of all types studied at the Mayo Clinic, estimated the incidence of chondrosarcoma in patients with multiple exostosis at more than 10 per cent. Jaffe (1958) believed it to be about 25 per cent. These figures are much higher than the experience of most clinicians suggests.

None of the fifty-six index patients in the present series developed this complication. Among the secondary cases only one example of a histologically proven chondrosarcoma was discovered. The patient, who had been aware of multiple bony swellings for as long as he could remember, injured his right thigh at the age of fifteen. Six months later a lump appeared at the site of injury and increased steadily in size over the next two years. At the age of eighteen a radiograph showed a large cartilaginous tumour at the proximal end of the femur. A clinical diagnosis of chondrosarcoma was made; amputation was advised but refused by the patient.
In spite of radiation therapy the tumour increased in size. Four years later there was radiological evidence of bone destruction with a more hazy outline to the calcified tumour, suggesting invasion of the soft tissues as well (Fig. 12). At the age of twenty-three he agreed to surgical treatment. A biopsy at this stage showed the histological features of a chondrosarcoma, but there was still no evidence of metastasis. The marked cellularity of the tumour, the presence of cells with plump nuclei and double nuclei, and the increased mitoses conformed to Jaffe’s (1958) criteria for diagnosing chondrosarcoma (Fig. 13). Disarticulation at the hip was followed by steady deterioration of his general condition and he died soon afterwards.

Several important features are illustrated by this case. Injury often precedes the alteration in the clinical pattern, though a causal relationship is difficult to prove. The commonest sites are in the bones around the hip: more than half of the patients reported in the literature developed the chondrosarcoma in the ilium or the proximal end of the femur. Characteristically the tumour increases slowly in size and metastases occur late; indeed the patient may die of some unrelated condition before the chondrosarcoma proves its malevolence.

Radiation therapy has no effect on this tumour; it should be removed completely as soon as the diagnosis is made.

**TABLE II**

**RELATIONSHIP BETWEEN RATE OF GROWTH AND FREQUENCY OF EXOSTOSES**

(Seventy-six cases)

<table>
<thead>
<tr>
<th>Bone</th>
<th>Proportionate contribution to bone length (expressed as a percentage of total elongation)</th>
<th>Number of cases affected</th>
<th>Percentage of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femur</td>
<td>Proximal end 20 80</td>
<td>58 71</td>
<td>77 94</td>
</tr>
<tr>
<td>Tibia</td>
<td>Proximal end 60 40</td>
<td>72 62</td>
<td>95 82</td>
</tr>
<tr>
<td>Fibula</td>
<td>Proximal end 55 45</td>
<td>69 55</td>
<td>91 72</td>
</tr>
<tr>
<td>Humerus</td>
<td>Proximal end 80 20</td>
<td>65 3</td>
<td>85 4</td>
</tr>
<tr>
<td>Radius</td>
<td>Proximal end 25 75</td>
<td>10 64</td>
<td>13 85</td>
</tr>
<tr>
<td>Ulna</td>
<td>Proximal end 15 85</td>
<td>7 52</td>
<td>9 68</td>
</tr>
</tbody>
</table>

The figures for the proportionate contribution of the proximal and distal ends to bone length are after Lacroix (1951).

**DISTRIBUTION OF LESIONS**

The lesions in multiple exostosis are strictly limited to bones developed in cartilage; the detailed distribution is characteristic (Fig. 3). The tubular bones, the iliac crests and the vertebral borders of the scapulae are the sites commonly involved; in the long bones the more actively growing ends are the most heavily affected of all (Table II). On the other hand exostoses are rare in the tarsal and carpal bones, the patellae, the vertebral bodies and the sternum. These peculiarities have not been satisfactorily explained.

Radiographic examination of representative sites at successive ages in forty-one subjects has shown that this selective distribution within the endochondral skeleton may be less a peculiarity of the disease than a reflection of certain differences in the normal process of growth at the different sites.

The distal end of the humerus and the proximal end of the ulna, where the epiphysical plates contribute very little to bone length, are no more frequently affected than a tarsal bone
such as the calcaneum which also develops an epiphysial plate and grows somewhat in length after the main body of the bone has been ossified (Fig. 3).

The other tarsal and carpal bones hardly ever show obvious lesions in routine radiographs.

However, when the development of these bones is traced by serial radiographs, abnormalities of growth can be demonstrated at some stage in at least half of the patients so examined. A typical case is illustrated in Figure 14. At five years two unusually dense ossification centres
appeared—one in the triquetrum and another in the scaphoid. Four years later these two bones had developed miniature exostoses and the distal pole of the triquetrum was clearly abnormal in size and shape. Thereafter, as growth proceeded, centrifugal ossification appeared to overtake these bony irregularities, gradually obscuring them in later radiographs. A single radiograph taken at the end of growth would probably show no sign at all of these early abnormalities.

Only the carpal bones have been studied in this way; it seems likely that the other small bones behave similarly. They have this in common, that growth and ossification proceed in all directions from the centre. Consequently there is no elongation of the bone, no marked transport of the growing end from its original position relative to the primary centre of ossification, no extensive modelling process—all characteristic features of growth in tubular bones. The presence or absence of an epiphysial plate as such is not important. The vertebral bodies, which develop two annular epiphysial centres at the upper and lower surfaces, are extremely rare sites of the disease.

On the basis of these observations it is suggested that all the bones developed in cartilage are equally exposed to the genetic disorder in hereditary multiple exostosis. The development of detectable lesions, however, is largely determined by the degree of elongation and modelling which the bone undergoes during growth; these factors are reflected in the characteristic differential incidence of lesions at the various sites.

PATHOGENESIS

Of the many theories that have been advanced to explain the basic defect of growth only three warrant serious attention—those of Müller (1914), Keith (1920) and Langenskiöld (1947). Müller (1914) observed, in the endochondral bones of a patient with multiple exostosis, small collections of cartilage cells arising from the proliferative layer of the periosteum. He suggested that these developed into exostoses and were due to a basic abnormality of the periosteum. Müller’s observations have been confirmed by others (Scherer 1928, Jaffe 1943), but there are serious objections to his theory of the pathogenesis of multiple exostosis. All the patients so far described as showing this phenomenon have been adults, and it is not known whether it occurs in areas that later develop exostoses. Furthermore, this could not account for the retardation of bone growth and the associated deformities which have been described.

Keith (1920) noted in radiographs of patients with multiple exostosis that the cortical density invariably stopped short at the metaphysial deformity (see Figs. 6 to 8). He believed that a sleeve of cortical bone extending to the epiphysial plate was required to confine the growing cartilage to its normal dimensions and prevent undue transverse growth; the absence of this cortical cuff would leave the epiphysial plate “exposed on the surface of the shaft and . . . free to give rise to irregular outgrowths or exostoses.” He suggested that this was the case in multiple exostosis and attributed the abnormality to a lack of coordination between endochondral ossification and longitudinal growth at the epiphysial plate and subperiosteal new bone formation at the periphery of the plate. The most vital aspect of Keith’s theory is that this is an abnormality of epiphysial growth and modelling, which could account for the associated skeletal deformities.

Although Keith did not present histological evidence for his theory, a “perichondrial ring of bone” has, in fact, been observed in animals surrounding the hypertrophic and calcified zones of the epiphysial plate (Lacroix 1951). The same structure can be demonstrated in human bones (Figs. 15 and 16). Histological and microradiographic examination of the distal end of the femur was carried out in fourteen specimens obtained at necropsy from previously healthy subjects who had died in accidents; their ages ranged from six months to eighteen years. The bony sheath is particularly well formed in those under five years; in the older subjects it may be altogether absent.
This perichondrial ring of bone is simply the most advanced edge of the periosteal new bone encountered farther down the diaphysial shaft and there is, as yet, no evidence that it is essential for normal orientation of epiphysial growth in humans. Certainly its absence in some of the older subjects studied had not led to the irregularity of growth which Keith suspected.

Nevertheless the lesions which have been described do suggest a dysfunction of whatever system normally coordinates the parameters of cartilaginous growth. It is generally held that growing cartilage increases its transverse diameter at right angles to the cell columns by appositional growth from the overlying perichondrium (Lacroix 1951, Weinmann and Sicher 1955, Ham 1957). Langenskiöld (1947), however, presented evidence that the opposite is the case: that the epiphysial cartilage normally expands by interstitial growth and that the outermost layer of cartilage cells is transformed into the proliferative layer of the peristeum. He explained the development of multiple exostosis as a persistence of the chondrogenic property of these most peripheral cells. Support for Langenskiöld's views has come from a more recent study of epiphysial growth with radioactive tritiated thymidine (Rigal 1961). This concept disposes of the main objection to Keith's theory and is not incompatible with Müller's observations.

**SUMMARY**

1. Hereditary multiple exostosis has been studied in fifty-six patients and their relatives. In most cases previous records were available and the progress of the disease could be traced over many years.
2. The characteristic lesions are described and the complications encountered in the present series of cases are noted. The disease is inherited in approximately two-thirds of the cases and invariably produces detectable lesions in the heterozygote.
3. The cartilage-capped exostoses are confined to the endochondral skeleton where their incidence is closely related to the growth potential of the sites involved.

4. An attempt has been made to explain the curious distribution of the exостoses, and the likely theories of the pathogenesis of the disease are discussed.

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