

"SYMMETRICAL OSTEOPOROSIS" IN A PALAEOANTHROPOLOGICAL MATERIAL

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(Received May 30, 1973)

Abstract

The cause of "symmetrical osteoporosis", hyperostosis spongiosa cranii or porous hyperostosis (grave No. 299 from the cemetery at Kiszombor) is the hyperplasia of the red bony marrow, the development of which can be explained by hemolytic anemia, namely Cooley's anemia. In thalassemia, the X-ray picture of the cranium shows "bristling skull bones" or "hair standing on end". Thalassemia can not be established beyond doubt as the etiology of the bone deformation mentioned, as it is not possible to achieve the family reconstruction for the clarification of the clinical picture. Bearing in mind the corresponding literature data, therefore, iron deficiency anemia can be considered, too. The skull in grave 299 indicates that "symmetrical osteoporosis" is an unsuitable expression, hyperostosis spongiosa cranii being more correct.

Introduction

A localised deformation of Pre-Columbian Indian crania, in which the diploic substance appeared enlarged in the external parietal layer, was named "symmetrical osteoporosis" by HRDLIČKA (1914). The process begins in the orbital roof or in the frontal squama. In an extreme case, the appearance of the porous tissue may even result in a "coral"-form. The frequency of this deformity is higher in the coastal region than in the mountainous districts.

Other authors too, e.g. WILLIAMS (1929), HOOTON (1930) and WAKEFIELD—DELLINGER—CAMP (1937), mentioned symmetrical osteoporosis — as it was then named — of the crania of historical times.

Materials and Methods

The bone destruction described by these authors is shown by the cranium of an individual (Inf. II) from grave No. 299 in the cemetery at Kiszombor dating from the Migration Period. A detailed anthropological and pathological description of the cranium is to be found in the book of BARTUCZ (1966). The main characteristics of its state are as follows:

1. In the parietal regions on both sides of the cranium, hyperplasia of the spongiosa with dendrite-shaped formations can be seen, encircling the area of the parietal tuber. That on the right is a little larger than that on the left (Fig. 1a, b, c).

2. In the upper medial parts of both orbitales tuberos protuberance is shown by the hyperplasia of the spongiosa, that in this case does not break through the external layer. According to NATHAN—HAAS (1966) this deformation is of trabecular type and corresponds to grade 6 (HENGEN 1972) (Fig. 2), established for the cribra orbitalia (WELCKER, 1888).

3. In the area above both mastoid processes, as well as in the alveolar and palatinal parts of the maxillary process and in the ala magna, small porosity can be observed.
4. Premature synostosis praecox.
3. The "hair on end" from (Fig. 3) is to be seen in the X-ray picture of the skull.

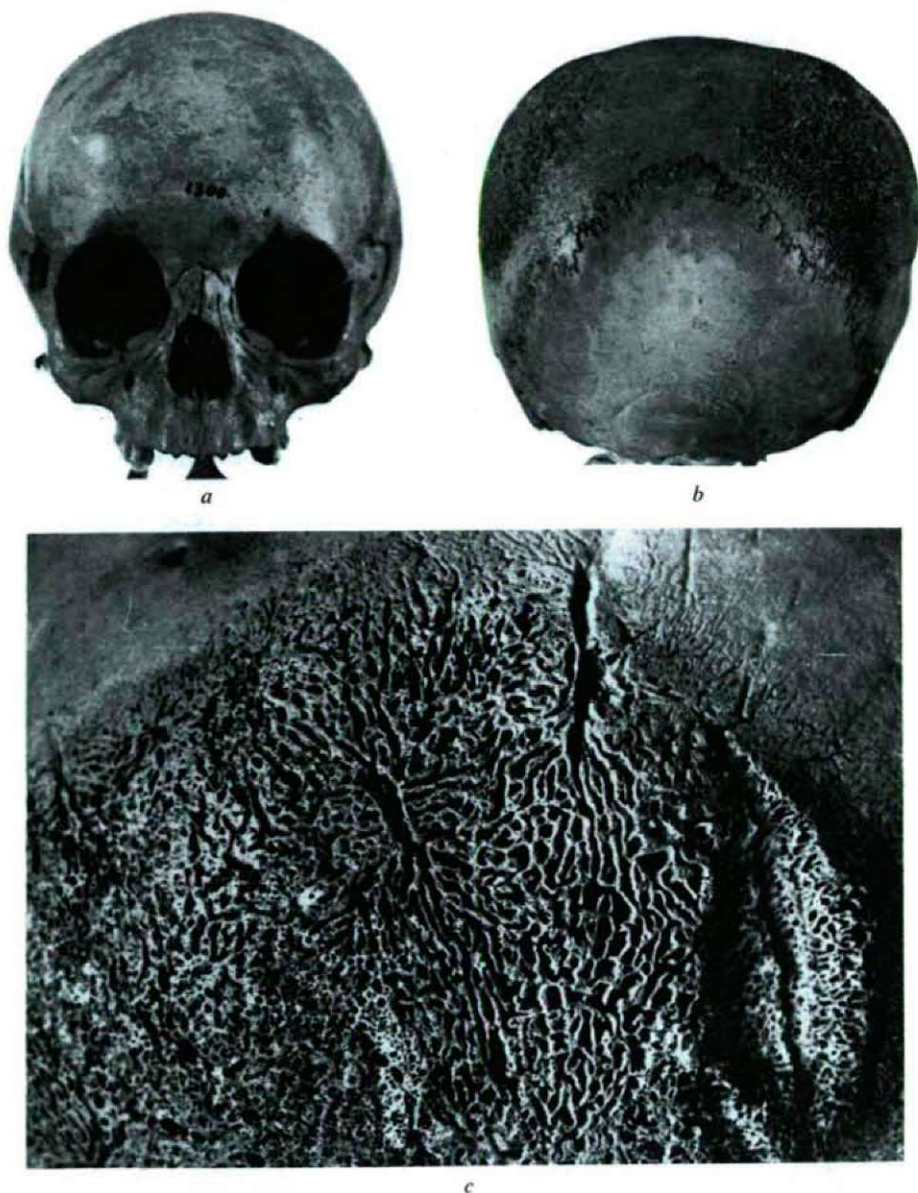


Fig. 1. Kiszombor, cemetery of the Migration Period grave 299 (1300).

- a) Ventral aspect of skull.
- b) Dorsal aspect of skull.
- c) Hyperostosis spongiosa cranii.

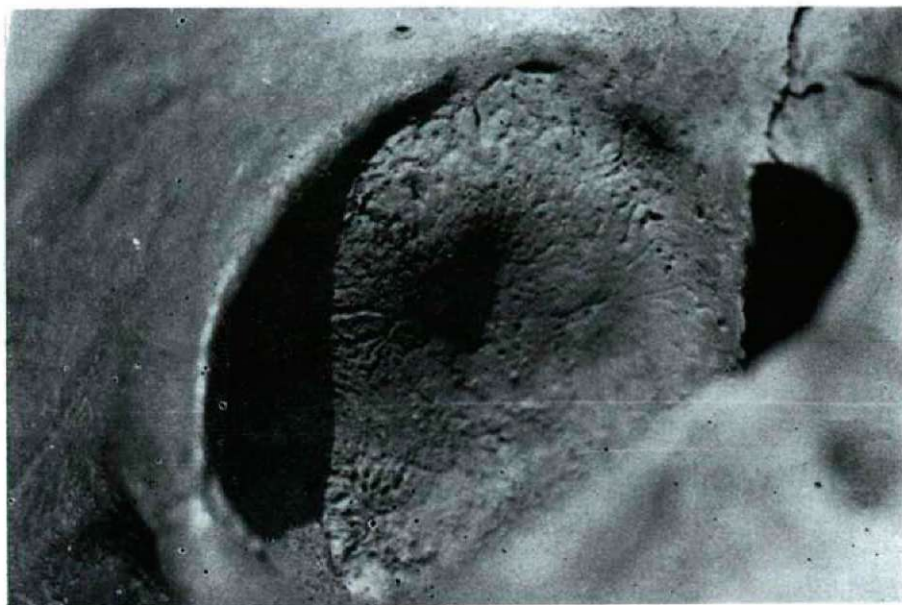


Fig. 2. Kiszombor, cemetery of the Migration Period grave 299 (1300).
Hyperostosis spongiosa orbitae (trabecular type).

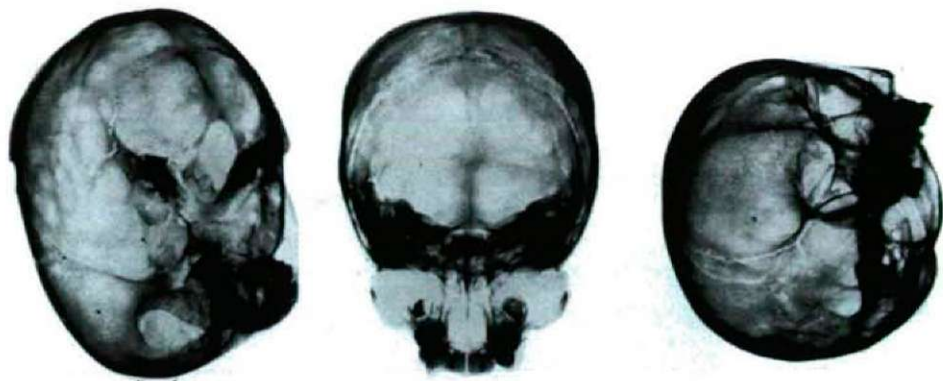


Fig. 3. Kiszombor, cemetery of the Migration Period grave 299 (1300). Cranial X-ray pictures: "hair on end".

Discussion

The cause of "symmetrical osteoporosis", hyperostosis spongiosa cranii (HAMPERL—WEISS, 1955; MOSELEY, 1965) or porous hyperostosis (ANGEL, 1964; 1966) is the hyperplasia of the medulla, the development of which can be explained by hemolytic anemia, namely Cooley's anemia. Red marrow is situated in the lacunae of the spongiosa of the osteophyte, whereby new erythrogenic foci develop roughly compensating the severe anemia of the organism (HAMPERL—WEISS, 1955). In thalassemia, the X-ray picture of the cranium, "bristling skull bones", produced by bone trabecules pressed together, is very characteristic (VOGT—DIAMOND, 1930; CAFFEY, 1957). This severe cranial deformation is characteristic mainly of thalassemia major (Cooley's anemia). Apart from the widening of the diploic substance of the bones, the lowered pneumatisation of Highmore's antrum, and the hypertrophy of the upper part of the maxilla are well-known (POWELL—WEENS—WENGER, 1965). The X-ray picture can be characterised by a bone destruction similar to thalassemia major in every kind of congenital hemolytic anemia, thus in th. intermedia, th. minor, th. hemoglobin-S, th. hemoglobin-E; in every sort of sickle cell anemia; and in spher- and elliptocytosis (MOSELEY, 1965).

In recent years, iron deficiency anemia turned out to give rise to the same skull defects as hemolytic anemia (MOSELEY, 1961; BURKO—MELLINS—WATSON, 1961; BRITTON—CANBY—KOHLE, 1960; SHAHIDI—DIAMOND, 1960; POWELL—WEENS—WENGER, 1965).

With regard to the X-ray picture of the cranium of a little girl suffering from severe anemia LIE (1958) states that the deformations are the same as those in Cooley's anemia. In this case, the anemia was the result of the multiplication of *Ancylostoma duodenale*.

The anemia-inducing effect of worms, particularly *Ancylostoma duodenale* and *Diphyllobothrium latum*, is well-known (HARANGHI, 1966).

Ancylostoma duodenale (Uncinaria) lives in the small intestine of the human, feeding on its mucous membrane and the blood of the vessels. One of its deleterious effects is thus to bring about loss of blood, while the penetration of its toxic dis-

charge into the blood causes serious complaints. *Diphyllbothrium latum* (wide tapeworm) is comparatively rare as a parasite of human (BREHM-revised by RAMMNER, 1960).

JELLIFFE and BLAKMAN (1962) give an account of the disease they named "Bahima". In this disease the X-ray picture of the cranium agrees with the well-known X-ray picture of hemolytic anemia, but the patients did not suffer from either thalassemia or sickle cell anemia or other hemolytic anemia. It was simply a question of iron deficiency anemia caused in the case of children by milk, their standing and main food.

In the establishment of the etiology of "symmetrical osteoporosis", the papers dealing with polycythemia must also be taken into consideration.

DYKSTRA—HALBERTSMA (1940) describe the thickening of the frontal region of the cranium in childhood.

CAFFEY (1961) reported on the connection with polycythemia in the case of cyanotic congenital heart diseases.

ASCENZI—MARINOZZI (1958) analyse the radiograms of the crania of patients suffering from this heart failure: a trabecular pattern with delimited fissures, and a parietal process, the bone marrow extending towards the periosteum.

The papers of MARIANI—BOSMAN (1962) and NICE—DAVIES—WOOD (1964) also report on bone deformations caused by the same disease.

The geographical localisation of the findspots of human skeletal remains originating from different historical ages and showing signs of porous hyperostosis exhibits a relation with the old-world occurrence of *Plasmodium falciparum* malariae, with the incidence of sickle cell anemia, and with iron deficiency anemia as one the results of protracted breast-feeding (MOSELEY, 1965).

From among the most important diseases characterised by erythroid hyperplasia in our case it is possible to exclude sickle cell anemia and its various forms, inherited spher- and elliptocytosis (MOSELEY, 1965); the case of polyglobulia developing with cyanotic congenital heart diseases; and polycythemia vera rubra, or Osler—Vaquez disease (HARANGHI, 1966); but thalassemia and hypochromic anemia may be involved.

Thalassemia is a syndrome consisting of heterogeneous, hereditary anomalies, manifested in homozygotes in the form of severe anemia, but in heterozygotes only in formal anomalies of the erythrocytes. The two main clinically important types of the syndrome are alpha and beta thalassemia, depending on whether the formation of the alpha or beta chain of the globin component of the hemoglobin is retarded. In a heterozygote state (thalassemia minor) the hypochromic anemia develops only slowly, while in homozygotes (thalassemia major) a marked hypochromic anemia with anisocytosis and poikilocytosis can be observed. There is, also a transition between thalassemia major and thalassemia minor of course: thalassemia intermedia. Less known are alpha thalassemia and the Hb LEPORE anomaly (MÁTYÁS, ref. 1973).

If some kind of thalassemia is considered, then in the case mentioned thalassemia major can come into question, as the roentgenogram of the cranium really corresponds to the bone deformity induced by this disease. At the same time, the localised osteoporotic area observed in the cranium of several individuals of the series (os frontale facies orbitalis) would possibly correspond to other anomalies of thalassemia or to their early signs (CAFFEY, 1937; 1951). In this case, grave 299

would be (German/Gepid) rather, than one from the Arpad-Age (CSALLÁNY, 1961) although it is described by TÖRÖK (1936) as a grave without accessories.

Naturally, it is not possible to establish thalassemia beyond doubt as the etiology of the bone deformation mentioned, as it is not possible to achieve the family reconstruction for the clarification of the clinical picture. Bearing in mind the corresponding literature data, therefore, iron deficiency anemia too can be considered as a cause of the patho-morphological picture of the cranium, possibly in connection with helminthiasis.

"Symmetrical osteoporosis", introduced by HRDLIČKA as an anthropo-pathological term, is suggested by the skull in grave 299 to be a fairly unsuitable expression for the indicated pathological process. In fact, the above-mentioned expression hyperostosis spongiosa cranii is more correct.

(The author's thanks are due to Dr. L. PÁLDY — Roentg. Clinic, Szeged — who X-rayed the bones.)

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