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SHORT REPORT

Rhinomaxillary Changes in a New-Caledonian Cranium: Palaeopathological Differential Diagnosis[1]

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Introduction

A human cranium, housed in the anthropological collection of the Museum National d'Histoire Naturelle of Paris (Institut de Paléontologie Humaine), exhibits rhinomaxillary pathological changes. These mainly erosive, bilateral and symmetrical facial lesions allow us to discuss their aetiology geographically, historically and in the context of their general health. This well-preserved cranium (labelled n° 1969-71-3) was brought back from Pins island, New-Caledonia, during the second half of the 19th century by a French Naval physician, Dr. Ponty. According to available information, this skull probably pre-dates 1850 and the French colonization of the New-Caledonian area. Its morphological characteristics suggest that it belongs to an older adult male. No post-cranial bones are associated with it.

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Description of the lesions

The gross examination of the cranium shows lesions mainly in the rhinomaxillary area (Figure 1). The most impressive osteological feature is an empty nasal cavity. On the CT scan (Figure 2), the frontal plane displays the loss of the inferior and superior turbinates as well as a partial atrophy and a coarse pitting of the intermediate turbinate. The paranasal sinus walls appear intact, without any increase or decrease in their thickness. In the midsagittal plane, the perpendicular plate of the ethmoid and the two anterior thirds of the vomer in the nasal septum are atrophied. Both lateral margins of the nasal aperture are thick, smooth and resorbed; the nasal bones are atrophied and collapsed (Figure 1a). A short anterior nasal spine is characteristic of the Pacific populations (Hamy, 1869; Le Double, 1906) but, in this case, there is clearly a partial absorption of the anterior portion of the anterior nasal spine exposing the spongy bone underneath and a loss of its intranasal part. The alveolar process of the maxilla displays a partial resorption affecting the anterior walls of the central incisors whereas prosthion, between the incisors, is intact. A clear alveolar recession,

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Figure 1. Cranium from Pins island (New-Caledonia): (a) facial view showing rhinomaxillary lesions; (b) basal view showing the palatal perforation.

around 3 mm in height, and an extensive pitting are observed on all surfaces of this bone.

A triangular lesion, 9 mm by 10 mm, perforates the nasal cavity into the oral cavity. This perforation with irregular anterior margins occurs in the medial part of the bone. It is located in the posterior half of the palate. Anteriorly, there is an area of pitting mid-line (Figure 1b). The mid-palatine suture is nearly closed and the incisive foramen is reduced to a residual perforation. Note that such an anatomical variation has been previously mentioned by Le Double (1906). In addition, the anterior teeth are more worn than the posterior teeth and there are numerous carious lesions. A resorption of the mandibular alveolar bone and the presence of pitting on this bone indicate probable periodontal disease. Finally, an advanced bilateral temporomandibular joint arthritis is observed in the glenoid fossae and on the mandibular condyles. The other parts of the face, the base and the cranial vault are free of pathological modifications.

Discussion

The partial recession of the anterior walls of the central incisors and the rhinomaxillary lesions are probably related to two distinct syndromes. The first, also associated with the mandibular changes, is most probably related to periodontal disease. The other set of facial modifications seems to be due to an unicist pathological process which is destructive, acute or chronic.

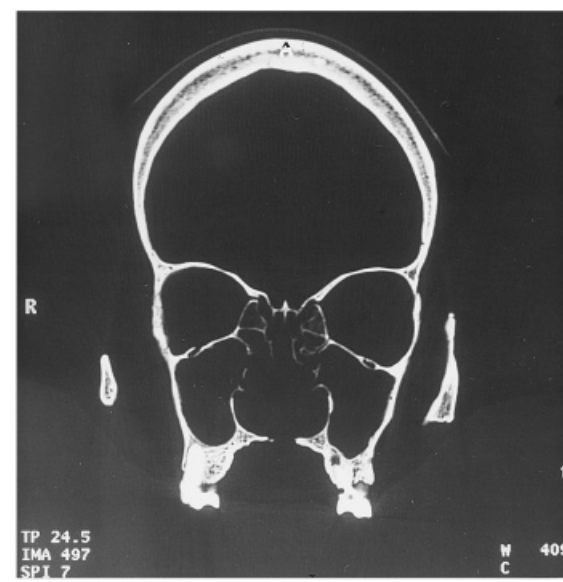

Figure 2. Cranium from Pins island (New-Caledonia): frontal scan showing loss of the intranasal structures and the palatal perforation.

The erosive process might have destroyed the whole nasal septum. Mid-facially, it spread up and down, resulting in a palatal perforation. The loss of the intranasal structures has been produced by the collapse and retraction of the nasals and the margins of the nasal aperture. Such a pathological description suggests that these facial modifications are not the result of trauma but most probably are due to a disease.

Aetiological considerations

The following discussion considers the symmetrical versus asymmetrical aspect of the lesions and the mid-line location; the existence of possible associated lesions in the face and in the vault; the existence of possible absorptive and proliferative bone reactions; the lesions of the nasals; the lesions of the paranasal sinus and the presence and location of the palatine perforation. The examination of these criteria allows us to discuss the different possible diagnoses starting from the less to the most probable one.

A congenital disorder such as cleft palate seems unlikely in this case since nasal cavity bone destruction is not associated with this kind of disorder. The recession of the nasal septum associated with atrophy of the sinus walls, which may result in a saddle-nose, characterizes Wegener's disease. However, a palatal perforation does not occur in this pathology. On the other hand, the mid-line granulomatosis of Stewart causes central destruction of the face with perforation of the nasal septum of the palate and sinus cavities. Such a description does not fit with the Pins island cranium pathological portrait, leading us to reject these two diagnoses.

In Noma (*cancrum oris*), if the lesions start most often in the lateral part of the alveolar process resulting in dental malposition, they also may occur sometimes mid-facially (Reynaud, 1980; Gentilini and Duflo, 1986). The normal dental position, the symmetry and the median location of the lesions, as well as the nose involvement, make this diagnosis very unlikely in the Pins island case.

Among the mycoses: aspergillosis and mucormycosis give a mid-facial lesional pattern which can be discussed as a differential diagnosis. Aspergillosis preferentially reaches the upper part of the face including the paranasal sinuses and the orbits (Hamel, 1973; Ortner & Putschar, 1985). Mucormycosis may produce necrosis of the turbinates and the nasal septum, and a palatal perforation may occur (Dellamonica *et al.*, 1978; Ortner & Putschar, 1985). Nevertheless, the strict unilaterality of such lesions results in the elimination of these fungal diseases in the present case.

Lytic lesions of the paranasal sinuses, the zygomatics, the mastoid processes and the petrous pyramid, inflammatory changes in the vascular foramina and modification of their number have been noticed in actinomycosis which rarely attacks the bones (1–15%) (Gentilini & Duflo, 1986) and, when it does, preferentially the lower part of the face and the mandible (Ortner & Putschar, 1985). Such pathological features do not seem consistent with the description of the Pins island cranium.

A palatal perforation and lesions of the nasals have been formerly described in tuberculosis (Meng *et al.*, 1942). However, the facial location of this disease is very rare and the characteristic pathological combination observed on this skull make such a diagnosis unlikely.

Leishmaniasis may produce lesions in the nasal cavity and in the palate. The loss of the anterior part of the nasal septum and of the inferior turbinates resulting in an empty nasal cavity have been mentioned (Sirol *et al.*, 1978). The Pins island cranium shows some of these features. Destruction of the inferior turbinates, the nasal bones proper and the maxilla occurs in rhinoscleroma (Badrawy, 1966a,b). The resulting lesions are not specific, so this is also a possibility.

Lepromatous or near lepromatous leprosy may produce facial lesions similar to those observed on the Pins skull which, in addition, exhibits no cranial vault lesions. Nevertheless, the lack of prosthion resorption associated with the anterior nasal spine atrophy and the destruction of the intranasal structures associated with the lack of inflammatory changes in the middle of the nasal surface are inconsistent with the specific criteria of the *facies leprosa* described by Møller-Christensen (1978). The nasal aperture

margins are totally anomalous while Andersen & Manchester (1992), in their description of the *'rhinomaxillary syndrome in leprosy'*, mention that the remodelling of the lateral margins of the nasal aperture *'appears to involve the inferior third to half of the aperture'*. The location of the palatal perforation, in the posterior third of the bone, does not correspond to that expected in leprosy (Manchester, 1995). In addition, the lack of prosthion recession which is an early gross change in leprosy seems inconsistent with the existence of the palatal perforation and the intranasal structure loss which usually occur in the advanced stages of this disease (Hackett, 1975). On the other hand, the presence of atrophied and collapsed nasals do not appear to be a leprosy distinctive criterion. In palaeopathological cases of leprosy, the nasals rarely seem involved (Andersen & Manchester, 1992; Manchester, 1995). For example, out of the 39 cases presented by Møller-Christensen (1978), only one shows nasal lesions. However, radiological studies report frequent resorption of the nasals in living victims (Ennouri *et al.*, 1991).

The facial changes observed on this skull are also similar to the naso-palatine lesions occurring in the tertiary stage of treponematosis. They fit especially with the description of *facies gangosa* (Delahaye *et al.*, 1968; Hackett, 1975, 1976; Ennouri *et al.*, 1991). The location of the palatal perforation, the ethmoid and vomer necrosis and the combination of pathological features are consistent with this hypothetical diagnosis. On the other hand, Fournier's figures of 1906, cited by Ortner & Putschar (1985) exhibit a higher frequency of the naso-palatine lesions than the cranial vault changes. So, the lack of cranial vault lesions is not a decisive criterion to dismiss the treponematosis diagnosis. Nevertheless, the absence of paranasal lesions seems a negative criterion, since a thickening of the wall of the sphenoidal and maxillary sinuses has been observed in a medieval case of probable syphilis (Mafart *et al.*, submitted).

Therefore on the basis of the osteological criteria, several conditions can be considered; among them rhinoscleroma, leishmaniasis, leprosy and treponematosis seem the most probable.

Health status in New-Caledonia around 1850

Since 1850, numerous cases of facial destruction have been described, especially by the French Naval physicians, at the beginning of the French colonization of New-Caledonia (Vinson, 1858; de Rochas, 1860, 1862; Bourgarel, 1860–1863). Schirmer (1986) records the presence of: *phtisie, scrofula, ulcer, elephantiasis* . . . in New-Caledonia. Leprosy was mentioned for the first time in 1865 on the Grande Terre, and in 1878 on Pins island (Grall, 1894). The existence of tuberculosis and treponemal infection was also recognized at this time. On the other hand, yaws could have been introduced in Pins island around 1830 from Tonga (Guiart, 1963; Gentilini & Duflo, 1986). However, the lesions of the soft tissues of the face, especially those of the rhinomaxillary region, are not very characteristic and diagnostic errors between rhinoscleroma, mycosis, treponemal infection, leprosy, leishmaniosis and oronasal tumour are possible (Hackett & Loewenthal, 1961). The first observers could have been victims of this confusion, even though the environmental context is in favour of the presence of infectious agents. Consequently, it is legitimate to be cautious with the interpretation of the historical data.

Conclusion

The destructive lesions of the rhinomaxillary region are generally not very specific and the possible causes are numerous. It is presumptuous to hope to reach a firm diagnosis for this isolated cranium, even though the geographical and historical contexts are well-known. From an osteological point of view, frequency analyses of lesions in a large population sample, with frequencies of possible associated lesions, could improve the aetiological discussion. Regarding this isolated cranium from Pins island, four infectious causes are the most probable: rhinoscleroma, tuberculosis, leprosy and treponemal infection. Available historical and health data tend to support these possibilities.

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