CASE REPORT

A case report of hyperostosis frontalis interna identified in a gross anatomy cadaver laboratory

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Abstract

Hyperostosis frontalis interna (HFI) is an uncommon condition characterized by excess bone growth with multiple nodules mostly on the inner table of the frontal bone.

We report a case of HFI in 85-year-old female identified during a routine dissection in the Faculty of Dental Sciences, University of Peradeniya. The inner table of the calvarium of the frontal bone was covered with large, irregular nodular bony thickening without internal damage to the brain.

Although there's a significant prevalence of HFI among general population, there are very limited research studies carried out in Sri Lankan population. Therefore, the adequate awareness about HFI, among medical professionals will help in more accurate interpretation of radiographs.

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Introduction

Hyperostosis frontalis interna (HFI), first described by Santorini and Morgagni in 1765, involves a remodeling of the endocranial plate into a cancellous bone structure (1). The condition is typically an incidental finding during skull X-rays or CT scans. In cadaveric dissections, it may present as visible bony overgrowth on the inner surface of the frontal bone. Here, we report a case of HFI in an 85-year-old female discovered during a routine dissection at Peradeniya University.

Case Presentation

An 85-year-old female cadaver dissected for a routine dissection class for the undergraduates of Faculty of Dental sciences, University of Peradeniya. The opening of the calvaria was done according to the dissection guidelines of the Department Manual by the academic staff of the Anatomy division. The consent of cadaver doners were obtained during registration for the body donation programme using the cadaver in teaching and research purposes.

The internal surface of the calvaria, presented with significant, irregular bony thickening extending over frontal bone; about 2cm on left side and 1.5cm on right (Figure 1A & 1B). No abnormalities were noted on the parietal or occipital bones. The temporal ridges were prominent on both sides (Figure 1C & 1D), and some areas of the parietal bone were markedly thin (Figure 1E). Suture lines were indistinct externally (Figure 1F). The brain showed no internal damage upon removal, and the gyri and sulci appeared intact.

According to the death certificate, this woman had died from cardio-respiratory arrest following subdural hemorrhage and cerebral herniation from an accidental fall.

Discussion

HFI is often seen with a prevalence of 16.4% among postmenopausal women aligning with our presentation. While HFI is typically asymptomatic, severe cases can lead to seizures, cognitive and mood disorders, Schizophrenia, impairment of memory due to brain compression and reduced blood flow (2).

The exact cause of HFI remains uncertain, though it is believed to involve both endocrine and metabolic factors. Leptin, a hormone that regulates satiety and metabolism, has been proposed as a potential contributor to HFI development due to its effects on other hormonal pathways. Additionally, increased leptin levels resulting from modern lifestyles may be contributing to a rise in HFI cases. Estrogen, which promotes angiogenesis, is also thought to play a role in HFI, as shown by the increased vascularization of affected bones (2). However, some cases, such as the report of 7-year-old boy with normal hormone levels, presented by Sue and Szakacs suggests that genetic or congenital factors may also be involved (3). Also in our case, there was no evidence of endocrinological disorders, aligning with typical presentations of HFI in postmenopausal females.

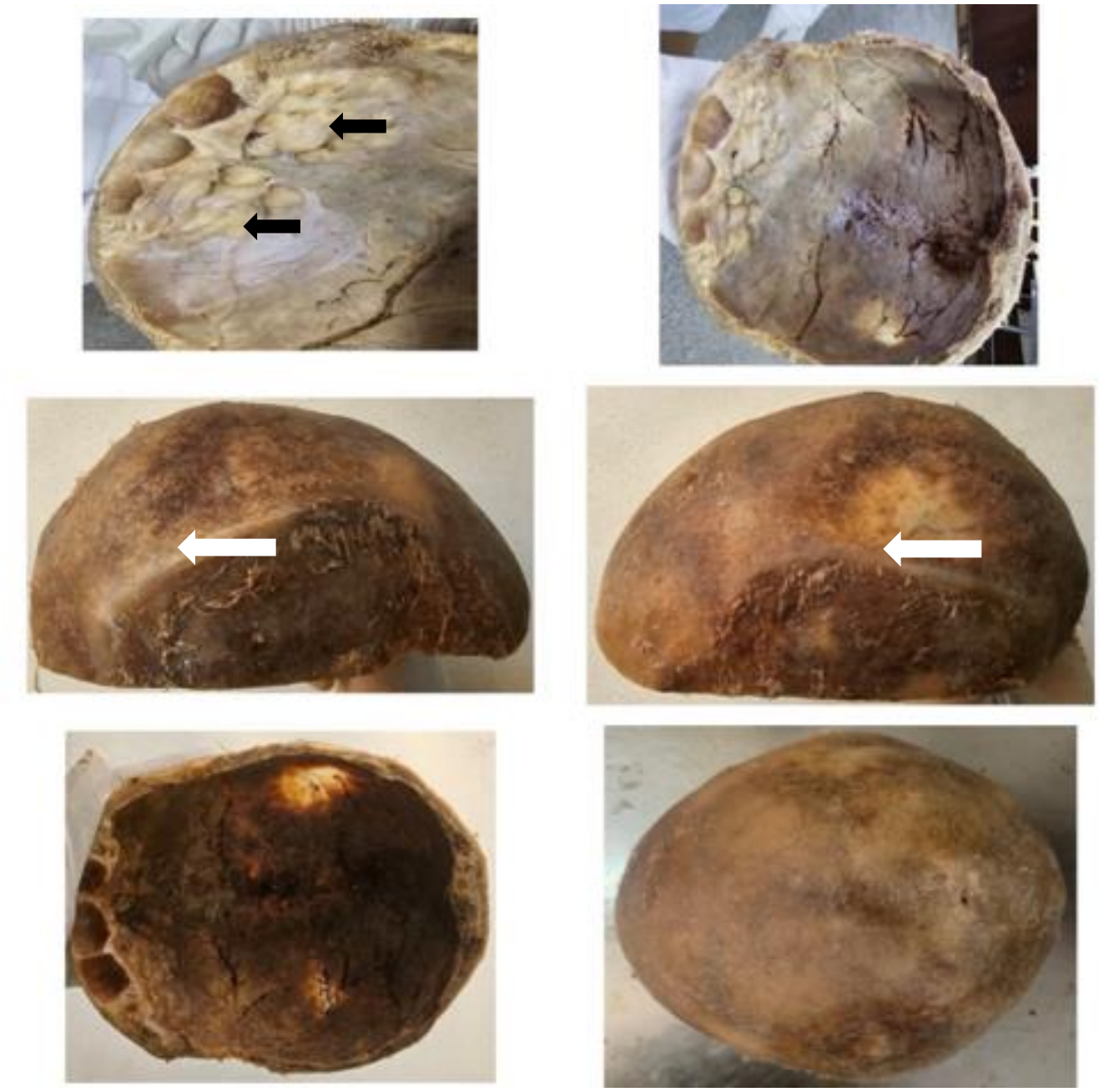

Figure 1: (A-B) The internal view of the calvaria. The black arrows indicate the bony thickenings. (C-D) The External view of the calvaria. The white arrows show prominent temporal ridges of the right and left side. (E) The internal view of the calvaria showing the thin parietal bones. (F) Superior view of calvaria showing fused suture lines.

The extent of bone overgrowth in frontal area of endocranium in HFI, has been classified as A, B, C, D and E according to "Hershkovitz's morphological and histopathological findings". Type A is less than 1 cm beyond normal bone growth. Type B, C and D are bone overgrowth involving 25, 50 and more than 50 percentages respectively. Severe HFI with soft tissue expansion involving dura classifies as E (4). In our case presentation, the overgrowth of bone falls under type D classification (overgrowth >50%) without any obvious brain tissue damage.

Though the cause of death was mentioned as subdural hemorrhage (SDH) and cerebral herniation according to the death certificate, we couldn't observe such findings during examination of the cadaver. The relationship between the two is not clearly established. It is possible that hormonal-driven angiogenesis in HFI could increase the risk of hemorrhages (5). Also, the CT findings can misinterpret HFI as hemorrhages and metastasis (6). Therefore, there is a chance of misinterpretation of radiological findings.

The examination of brain in this cadaver, showed no atrophy, and the sulci and gyri were intact. Suture lines were fused possibly due to intracranial pressure, aging, or increased bone deposition associated with HFI. The parietal bones were thinner than expected, which may be attributed to the varying embryological origins of different skull bones and age-related changes in bone metabolism. No other bony abnormalities were found in this case.

Although there's a significant prevalence of HFI among general population, there are very limited research studies carried out in Sri Lankan population. The CT findings can misinterpret HFI as bone metastases, or hemorrhages. Therefore, the adequate awareness about HFI, gold-standard diagnostic methods, and radiological findings in HFI among medical professionals will reduce such false positive diagnoses.

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