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Case Report

Biparietal osteodystrophy in an 86-year-old woman: An incidental finding following minor head trauma ☆

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ABSTRACT

Biparietal osteodystrophy (bilateral parietal bone thinning) is a rare but well-documented phenomenon characterized by symmetric thinning of the parietal bones of the skull. It predominantly affects older adults, particularly women. We report the case of an 86-year-old woman who was incidentally diagnosed with this condition after a minor head injury. She presented with a low-impact fall and underwent neuroimaging to exclude intracranial hemorrhage. Computed tomography (CT) of the head revealed bilateral concave thinning of the parietal bones with an intact inner table, and no acute fracture or hemorrhage. A comprehensive workup, including metabolic and endocrine tests, showed no secondary causes of bone loss. Neurosurgical evaluation confirmed that no intervention was required. The patient was managed conservatively and advised on fall prevention measures, given the increased risk of skull fracture and brain injury in the presence of severe calvarial thinning. This case highlights the importance of recognizing biparietal osteodystrophy on imaging and distinguishing it from more acute pathologies as well as the need for appropriate counseling regarding its clinical implications.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Introduction

Biparietal osteodystrophy, also known as bilateral parietal thinning, is an uncommon calvarial condition characterized by symmetrical thinning of the parietal bones [1]. Historically, it has been referred to by various names (eg, senile biparietal atrophy or *malum senile biparietale*) in older literature [2]. The condition was first recognized in the 18th century and has an estimated prevalence of 0.25%-0.80% in the general population [3,4]. It shows a predilection for elderly individuals, particularly women [5,6].

On imaging, biparietal osteodystrophy is defined by bilateral, oval or concave areas of thinning in the parietal bones, typically located between the parietal eminences and the sagittal suture [5]. The outer table and diploic space are thinned or absent in these regions, while the inner table of the skull is usually preserved [7]. These findings produce a smooth, symmetric depression in the skull vault on radiographs or computed tomography (CT), often discovered incidentally when imaging is performed for other reasons [5,7].

Clinically, most cases of biparietal osteodystrophy are asymptomatic and are discovered incidentally on imaging or autopsy. Some patients may notice palpable scalp depressions or present with nonspecific symptoms such as mild headaches or dizziness [8]. The principal clinical importance of recognizing this condition lies in its potential to predispose patients to serious injury after head trauma. The thinned areas of the skull have reduced mechanical strength, rendering them vulnerable to fractures or inward buckling from relatively minor impacts. Epidural hematoma and life-threatening intracranial injuries resulting from trivial head trauma in patients with unrecognized parietal thinning have been reported. Thus, identifying this entity is crucial, and appropriate precautions can be advised. In the following case, we describe an elderly woman in whom biparietal osteodystrophy was detected incidentally on imaging after a fall, and discuss the implications for management.

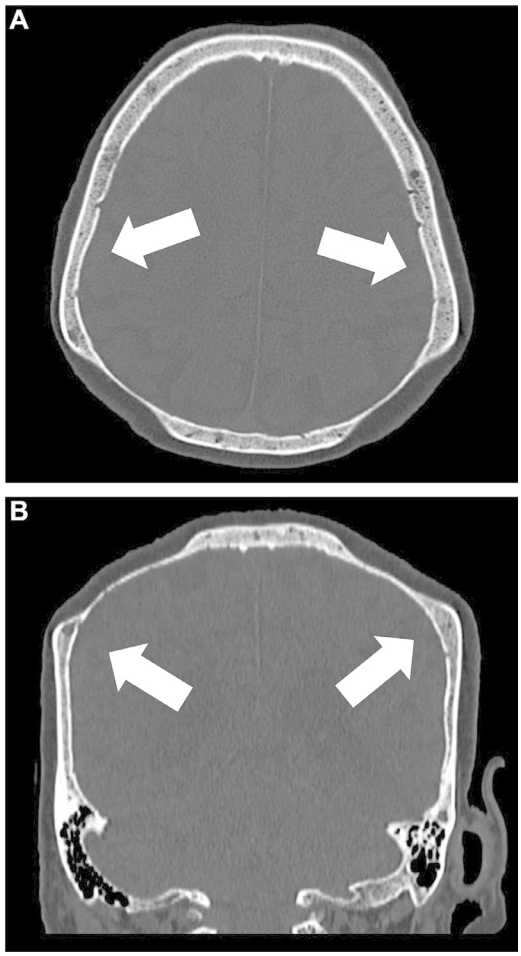

Fig. 1 – Axial (A) and coronal (B) CT head images in bone window, demonstrating symmetric thinning of both parietal bones (white arrows). The affected areas appear as bilateral concave depressions with loss of the outer table and diploic space, while the inner table remains intact. The contour of the skull vault is smoothly indented at these sites, and no fracture lines are seen.

Case presentation

An 86-year-old female presented to the emergency department with a sudden unwitnessed fall at home. The patient sustained a superficial laceration on her left forehead and minor abrasions on her cheek and nose. She did not lose consciousness and was brought in primarily for evaluation of possible head injuries. Her medical history was notable only for well-controlled bronchial asthma managed with an inhaler, and she had no history of osteoporosis or long-term steroid use. On examination, she was alert, with a Glasgow Coma Scale score of 15. Her vital signs were within normal limits (blood pressure, 135/88 mmHg, pulse 74/min, afebrile), and she had no focal neurological deficits. The New Orleans Criteria (NOC) was +3 (age >60 years, headache presentation, and visible trauma above the clavicle). Apart from the external facial injuries, physical and neurological examinations were unremarkable. Given the patient's advanced age and head trauma above the clavicle, a non-contrast CT scan of the brain was performed to rule out intracranial hemorrhage or skull fracture.

CT imaging of the head revealed an unexpected calvarial abnormality. There were bilateral symmetric areas of thinning involving the parietal bones. These appeared as shallow concave depressions in the parietal regions on axial and coronal images (Fig. 1). The thinned regions spanned the area between the sagittal suture and parietal eminence on both sides. The outer table and diploic space of the parietal bones were markedly attenuated in these regions, while the inner table remained intact, resulting in smooth inward bowing of the skull vault. No overlying scalp swelling or defect was present on the soft-tissue windows of the CT (Fig. 2), confirming that the depressions were due to bone thinning rather than an external injury. There was no acute cranial fracture. Apart from the calvarial findings, head CT showed evidence of chronic small-vessel ischemic changes and age-related cerebral atrophy, but

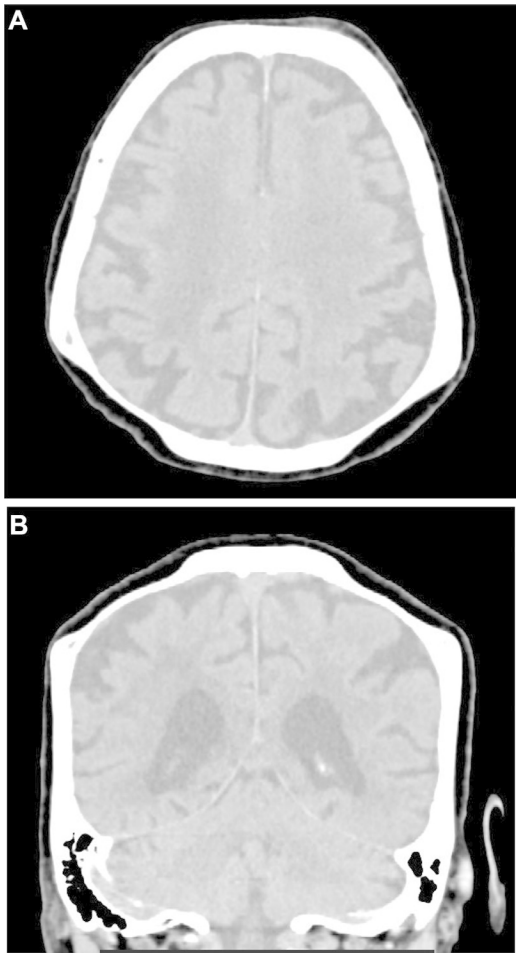

Fig. 2 – Axial (A) and coronal (B) CT head images in soft-tissue window, showing the overlying scalp and soft tissues. The scalp is intact with no external defect or hematoma, confirming that the depressions are confined to the bony calvarium. There is no evidence of soft tissue mass or swelling over the thinned parietal regions.

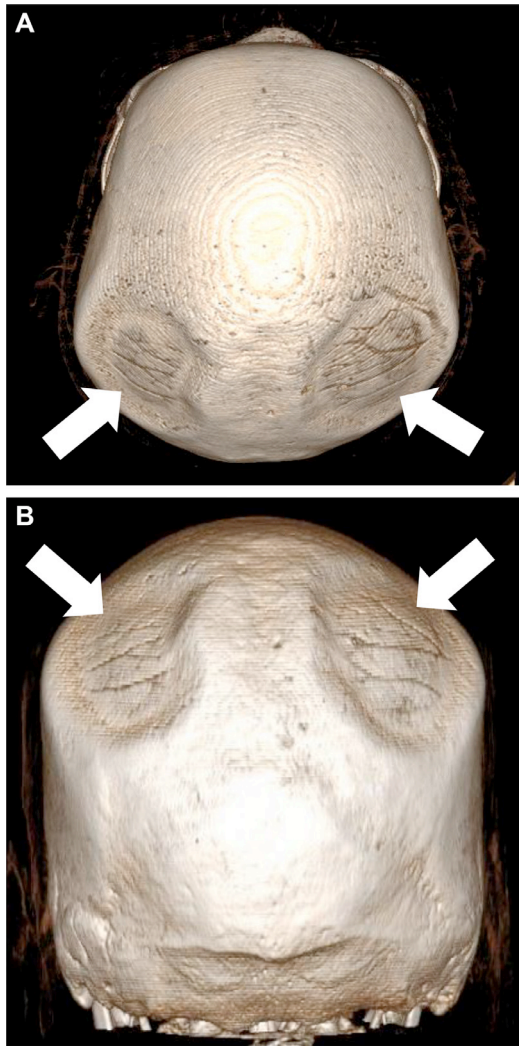

Fig. 3 – Three-dimensional volume-rendered CT reconstructions of the skull from a superior (A) and a posterior (B) perspective. These images clearly depict the bilateral parietal bone concavities (white arrows) on the vertex of the skull. The defects are symmetric and located between the sagittal suture (midline) and the parietal eminences laterally. The smooth margins and absence of any full-thickness perforation are evident on these reconstructions, consistent with biparietal osteodystrophy.

no acute intracranial hemorrhage or focal brain lesion. Three-dimensional reconstructed images of the skull were generated for better visualization of the bony anatomy (Fig. 3), which clearly demonstrated the bilateral parietal bone concavities and smooth margins. These imaging features were consistent with those of biparietal osteodystrophy.

The patient was co-managed by emergency and neurosurgery teams. A neurosurgical consultation confirmed that bilateral parietal bone thinning was an incidental benign finding, and no surgical intervention was required. She was admitted for observation due to head trauma but remained neurologically stable with no signs of delayed hemorrhage or other complications during a 24-hour observation period. A multidisciplinary discussion with a neuroradiologist confirmed the diagnosis of biparietal osteodystrophy and excluded other potential causes of focal calvarial thinning (such as lytic lesions or metastases). Additional laboratory investigations were performed to evaluate any underlying metabolic or endocrine abnormalities that could contribute to the bone loss. These tests included serum calcium and vitamin D levels, thyroid function tests, parathyroid hormone levels, and a panel of tumor markers. All results were within the normal limits. No reversible or secondary cause of parietal thinning was identified, supporting the idiopathic (age-related) etiology in this case.

The patient was discharged home with instructions and precautions related to her condition. In particular, she and her family were educated about her increased susceptibility to head injuries due to thinning of her skull. Preventive strategies were emphasized: she was advised to avoid high-risk activities that could lead to falls or head trauma, use assistive devices for ambulation as needed, and make safety modifications in her home (such as removing tripping hazards and

installing bathroom grab bars). A follow-up with her primary care physician was arranged to further assess the risk of falls and bone health. It was recommended that the patient undergo bone density evaluation and receive appropriate management for osteoporosis if indicated. At the time of discharge, she had no neurological deficits and no routine neurosurgical follow-up was deemed necessary. The plan was for clinical observation; further imaging would be considered only if the patient developed new symptoms or had additional head injuries in the future.

Discussion

This case illustrates a prototypical presentation of biparietal osteodystrophy in an elderly patient discovered incidentally on imaging after minor head trauma. Biparietal osteodystrophy is a rare entity, but radiologists and clinicians should recognize to avoid misdiagnosing it as an ominous pathology. In our patient, the bilateral and symmetric nature of the parietal bone depressions, along with the preservation of the inner table and lack of destructive bone margins, were key imaging clues that this was an instance of benign age-related parietal thinning. The absence of scalp injury or hematoma overlying the depressions further supports that these were chronic changes rather than acute fractures. Identifying these features is important because other conditions, such as extensive lytic skull lesions or post-traumatic bone defects, could potentially appear similar on scans, but would require very different management.

Bilateral parietal thinning has been documented in the medical literature for over two centuries [3], yet its exact cause remains uncertain. Earlier reports, including archaeological findings, noted this phenomenon in ancient skulls and gave rise to various descriptive terms (eg, “senile parietal atrophy”) [4]. By the late-20th century, clinical researchers like Cederlund et al. [3] conducted a large radiographic survey and found parietal thinning in about 2.4% of skull X-rays, supporting that it is uncommon but not exceedingly rare in the elderly. More than 90% of their cases exhibited a “flat” type of thinning (smooth shallow depressions) rather than a deeper “grooved” type [3]. Our patient’s imaging likewise demonstrates the classic flat-type thinning, with a gentle concave contour and no focal bony defect.

Multiple hypotheses have been proposed to explain why biparietal osteodystrophy occurs, but none is definitive. Earlier authors debated whether it represented a congenital calvarial dysplasia or an acquired process of bone loss [2,4]. One theory suggests that it may result from chronic mechanical factors: the parietal regions lack direct muscle attachments (unlike the temporal or occipital bones that have muscles inserted); thus, the presence of only the galeal aponeurosis over the parietals might lead to reduced biomechanical stress on those bones, predisposing them to localized osteoporosis and thinning over time [2,4]. Contemporary evidence favors an acquired, age-related degenerative process (involutional osteoporosis of the skull) rather than a congenital variant [9].

Systemic factors are also considered. Given that most patients are postmenopausal women, an analogy to postmenopausal osteoporosis has been drawn–age-related hormonal changes could contribute to generalized bone loss, including in the skull [3,6]. Histopathological examination in prior cases demonstrated an absence of osteoclast activity in the affected bone, suggesting that decreased bone formation (as seen in senile osteoporosis) rather than active bone destruction underlies the thinning [3]. A recent clinical study of 43 patients with bilateral parietal thinning found that increasing age was the only significant independent risk factor for progression of the bone loss [9]. In that series, the authors observed an average decrease of ~0.66 mm in parietal bone thickness per year of aging, confirming that the condition tends to slowly progress over time [9]. Interestingly, that study also noted an association between hypertension and greater defect size, while other comorbid factors (eg, osteoporosis, steroid use) were not statistically significant [9]. Overall, biparietal osteodystrophy can be viewed as an idiopathic or age-related calvarial osteopenia localized to the parietal bones.

Other reported associations include long-term corticosteroid use, chronic diseases (such as diabetes mellitus or hyperparathyroidism), inflammation, or even rare bone disorders such as Gorham-Stout disease [10–12]. However, in many cases (such as ours), no specific causative condition is identified, and thinning is considered idiopathic. The recent study by Sanati-Mehrizy et al. [1] provides strong evidence that age itself is the driving factor, showing a correlation between advancing age and both the degree and progression of parietal thinning. In our patient, after excluding other pathologies, we likewise conclude that her biparietal osteodystrophy was an age-related involutional change.

From a clinical perspective, the most significant concern in biparietal osteodystrophy is the increased risk of intracranial injury from trauma. The structural integrity of the skull is compromised in thinned areas, which can fracture more easily upon impact. Even without a fracture, thinner bones may transmit force to the brain more readily. Several case reports have highlighted these dangers. Yılmaz et al. [11] described a 78-year-old woman with known biparietal thinning who suffered a trivial head bump yet developed a linear parietal bone fracture and an epidural hematoma as a result. Mann et al. [13] reported an autopsy case of a 65-year-old man who died after a minor fall, and postmortem examination revealed marked bilateral parietal thinning and fatal contre-coup brain contusions underlying the thinned areas of the skull. These reports, along with earlier observations in the literature, [6] make it clear that what might ordinarily be a harmless bump on the head can cause a life-threatening hemorrhage in patients with this condition. Fortunately, our patient did not suffer a fracture or bleeding from her fall, but her case reinforces the principle that clinicians should counsel such individuals to minimize head injury risk. For this reason, in our management, we placed heavy emphasis on fall prevention strategies.

The management of biparietal osteodystrophy is generally conservative. There is no specific medical treatment to reverse established bone thinning. Management centers for monitoring and protecting patients from trauma. Some authors have recommended follow-up imaging (eg, periodic CT scans) to assess if the thinning is progressing, especially in

relatively younger patients [9]. In cases where the bone becomes extremely thin to the point of near-transparency, or if there are signs of developing skull perforation, prophylactic surgical intervention can be considered. There are few reports of cranioplasty performed in patients with very advanced biparietal osteodystrophy to reinforce the skull and protect the brain [14]. This is generally reserved for extraordinary cases, such as those with actual defects or imminent risk of brain herniation or cerebrospinal fluid leak due to bone loss. In the vast majority of cases, including the present one, treatment consists of observation and addressing modifiable risk factors. Our patient's workup did not reveal osteoporosis, but if it had, standard osteoporosis therapy (calcium/vitamin D supplementation, bisphosphonates, etc.) could be justified in hopes of improving the overall bone density. Close coordination between radiologists, neurologists, neurosurgeons, and primary care providers is helpful to ensure that the diagnosis is understood and that the patient receives appropriate advice. Recognizing this entity is also important in forensic and radiologic contexts to avoid misinterpreting depressions as evidence of trauma or skull defects on post-mortem scans.

Conclusion

Biparietal osteodystrophy is a rare diagnosis characterized by symmetrical thinning of the parietal bones and is typically identified in older patients. It is usually an incidental finding with no direct symptoms; however, it has important implications. Recognizing the characteristic imaging appearance of this entity is crucial for distinguishing it from other destructive skull lesions or acute traumatic injuries. Although no specific treatment is required for bone thinning, patients with this condition should be educated about their increased vulnerability to head trauma. Preventive strategies, such as fall prevention and protective measures, are advisable to reduce the risk of serious intracranial injuries. This case underscores the importance of considering biparietal osteodystrophy in the differential diagnosis of calvarial findings and for proactively managing patients once the condition is identified.

Research data for this article

Data not available/The data that has been used is confidential.

Patient consent

Written informed consent was obtained from the patient for the publication of this case report.

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