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# A case report of an incidental finding of biparietal osteodystrophy in a patient with traumatic cerebellar haematoma

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**Keywords**
biparietal osteodystrophy, cerebellar hematoma, computed tomography scan, head injury, parietal thinning

## ABSTRACT

Biparietal osteodystrophy refers to thinning of the parietal skull bone. It is a rare entity, commonly discovered incidentally on imaging of the skull for other reasons or at autopsy. Following head trauma, fracture of the thinned-out parietal bone, with or without underlying hematoma, is expected due to its reduced protective effect, especially with direct impact. In this case report, we present an elderly woman who sustained a cerebellar hematoma and frontal lobe contusion with intact parietal bone following head injury, with an incidental finding of symmetrical thinning of the parietal bones on cranial computed tomography scan.

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## INTRODUCTION

Biparietal osteodystrophy (BPOD), a symmetrical thinning of both sides of the parietal bones of the skull, is an extremely rare condition, with an incidence 0.4-0.5%.[1,2] In the past 25 years of existence of neurosurgical service at our facility, this is the first case of BPOD we have seen. Likewise, following a diligent literature search, there is a dearth of report on BPOD in the sub-Saharan Africa. However, this condition shows no geographical or racial predilection.[3] The scarce report of biparietal osteodystrophy could either be due to its rarity or the fact that it is mostly asymptomatic and only discovered incidentally on cranial imaging or it could be both factors at play. In this report, we present an elderly woman with an incidental finding of biparietal osteodystrophy on cranial computed tomography (CT) scan carried out following head injury.



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## CASE PRESENTATION

Patient is a 74 -year- old right-handed woman who presented to our facility with altered sensorium four days after she was involved a pedestrian-motorcycle road traffic crash. She sustained isolated head injury and had no other symptoms. Patient received an initial care at a peripheral hospital prior to referral to our facility on account of delay in regaining consciousness. She was both hypertensive and diabetic, controlled on medications. She had no preceding history of bone or joint pains neither was she on steroid use or any other medications apart from her antihypertensive and oral hypoglycemic drugs.

Physical examination revealed an elderly woman with a Glasgow coma score (GCS) of 11 [Eye Opening (EO)-3; Best Verbal Response (BVR) -3; Best Motor Response (BMR) -5] with blood pressure of 150/80mmHg and pulse rate of 88 beats per minute. Her Pupils were 3mm and reacted to light bilaterally (both direct and consensual). She moved all limbs equally; tone and deep tendon reflexes were normal globally and had no palpable scalp depression. Patient had a cranial computed tomography (CT) scan which showed right cerebellar hematoma, right frontal lobe contusion and symmetrical thinning of the parietal bones **(**figure 1**)**.

She was managed non-operatively because there was no evidence of fourth ventricular or brainstem compression nor hydrocephalus. She made gradual and progressive improvement, with her GCS improving to 14 (with BVR of 4) at 5$^{th}$ day on admission. Laboratory investigation showed normal serum calcium; 2.5mmol/litre (normal range: 2.2-2.7mmol/litre), normal serum phosphate; 0.89mmol/litre (normal range: 0.6-1.4mmol/litre), normal alkaline phosphate (ALP); 172.7 IU/litre (normal range: 64-306 IU/litre) and normal serum 25-hydroxylcholecalciferol (25-OH D3); 87.91ng/ml (normal range: 30-100ng/ml). The renal function test was also within the normal limit.

However, on the 8$^{th}$ day of admission (12 days post traumatic event), she developed fever with coarse crepitations on chest auscultation and her GCS dropped to 9. She was co-managed with chest physician and endocrinologist for chest infection and diabetes. A repeat brain CT scan showed resolving cerebellar hematoma and frontal lobe contusion and there was no hydrocephalus (figure 2). Unfortunately, she succumbed to sepsis on the 11$^{th}$ day of hospital admission (15 days after head trauma).

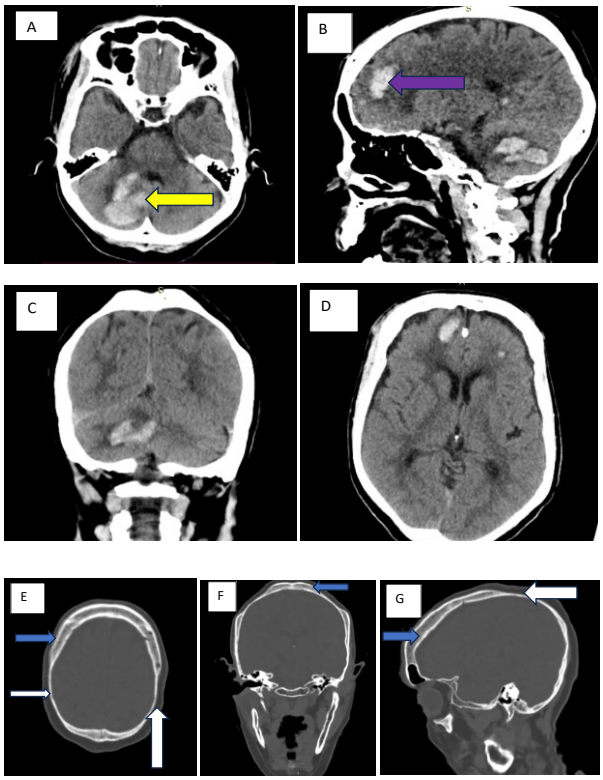


**Figure 1.** (Cranial computed tomographic images in brain windows (A-D), showing hyperdense collections (HU=62) consistent with right cerebellar hematoma (yellow arrow) and right frontal lobe contusion (purple arrow) and in bone windows (E-G) which show bilateral and symmetrical thinning of the parietal bones (white arrows) with preservation of other bones (blue arrows) of the skull.

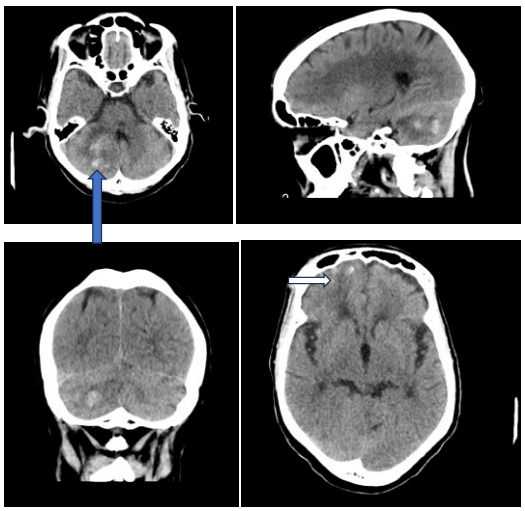

**Figure 2.** Repeat brain CT scan of the patient 9 days after the initial scan, shows resolving cerebellar hematoma (blue arrow)

and almost completely resolved frontal lobe contusion (white arrow).

## Discussion `

Though a rare condition, biparietal osteodystrophy (BPOD) has been with us since antiquity, with the first case documented in the 18th century by Sandifort (1783).3,4 This condition is commoner among women after the age of 60 years but when it occurs in younger age group, it is commoner in males.3–5 This epidemiological finding correlates with the presented patient, an elderly woman in her eight decade of life.

Initially, BPOD was thought to be a normal anatomical variant and that it could have been present in the patient since childhood, however recent studies have labelled it a pathological entity.3,5 Although, the underlying aetiology is not known, there are theories that have been postulated to explain its development. These theories include developmen¬tal defect of the calvarium/ congenital dysplasia of the diploe, senile osteoporosis, external mechanical pressure on the scalp/calvarium from carrying of load, trauma and inflammatory conditions of the scalp/skull.4,6,7 Some other systemic conditions have also been linked with symmetrical thinning of the parietal skull bone, such as hyperparathyroidism, diabetes and long-term steroid use, but none of these proposed mechanisms could adequately explain the pathology.8,9

The congenital theory was refuted after years of archaeological, pathological and radiological studies, that the condition is an acquired and progressive in nature rather than a developmental dysplasia.3,5,6 Earlier on in the discovery of the biparietal osteodystrophy, mechanical theory from chronic scalp/skull trauma was implicated because the archaeologists attributed the frequency of the lesion among the Egyptians to the pressure of the weight of the wig worn by the Pharaonic courtiers. However, even today, millions of people habitually carry heavy loads on their heads, without developing this anomaly, leading to the dismissal of the mechanical theory.3 Another theory that gained popularity was senile osteoporosis due to the fact that this condition is mostly discovered in the elderly women following loss of female sex hormones.3,4,6 However, some cases have been found among children which cannot explain the senescence theory.10,11 Even though the patient reported by Verma et al had osteoporosis,12 the index patient did not report any history suggestive of osteoporosis and serum calcium, phosphate, alkaline phosphatase and vitamin D were all normal.

The localization of the lesion to the parietal bones and not involving other bones also queries the aetiological factor of systemic conditions, at best these conditions could be viewed as association rather than aetiology. Given that the underlying aetiology of biparietal thinning remains to be fully elucidated, what is however certain in the pathogenesis is that histological analysis demonstrated an erosion of the outer table of the skull, remodeling and subsequent loss of the diploe, leaving a thin often less than 1mm inner table.3,11

The thinning of the parietal bone can render an individual more susceptible to skull fracture and intracranial hematomas following head injury, particularly if the site of impact involves the thin parietal bone.7,11 However, in the majority of cases, BPOD is found incidentally as documented in the index report. Other reports show that many cases were diagnosed at autopsy and on archaeological and anatomical dry bones specimens.3,7,11,13 It can also manifest as a scalp depression with or without pain.1,12 In the setting of trauma, patient could present with parietal skull fracture with or without underlying hematoma due to the reduced protective effect of the thinned-out skull.

Muhammet et al reported a linear parietal skull fracture with underlying epidural hematoma in a 78-year-old woman with incidental finding of biparietal osteodystrophy.14 It was quite surprising that there was no skull fracture nor brain contusion or hematoma in the parietal region of the index patient despite the history of trauma to the head. One would have expected contusion or hematoma in the parietal area, instead there were frontal lobe contusion and cerebellar hematoma despite the thickness of the occipital bone and thick muscular buttressing protective layer overlying the cerebellum. The probable explanation for this is that the patient might have had coup and contrecoup injury to the back and front of the head, sparing the parietal region and hence sustaining cerebellar hematoma and frontal lobe contusion.

Biparietal osteodystrophy is mostly asymptomatic, hence in most cases requires no treatment. However, some authors suggested

cranioplasty for cosmetic reason and also to protect the brain from atmospheric pressure effect and injury during head trauma.4,9 However, in the setting of trauma with associated intracranial hematoma, there may be need for surgical evacuation of the hematoma if indicated. Opportunity can also be taken to reconstruct the thinned-out parietal bone by performing a cranioplasty at the same sitting for future protection of the brain.

Just like the index patient whose hematoma/contusion were managed conservatively, the patient reported by Muhammet et al though had epidural hematoma was also managed non-operatively as the hematoma was minimal in volume and there were no indications for surgical evacuation.14 The fact that the index patient improved neurologically initially and the repeat neuroimaging at deterioration showing significant resolution of hematoma and contusion without hydrocephalus, indicated that the cause of death was most likely not related to the traumatic hematoma/contusion but sepsis.

## Conclusion

This report presents a rare case of biparietal osteodystrophy, the first in our 25 years of neurosurgical service. The rarity of this condition may not be unconnected with its asymptomatic nature in most cases, with majority being found incidentally, like reported in this case. Our report agrees with previous reports that the condition is commoner in women after the age of 60 years.

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