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Case Report

"Ping-pong" fracture: An exclusive entity of neonates and infants? A case analysis and literature review

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ABSTRACT

Background: "Ping-pong" fractures are a type of depressed fracture in which there is no rupture of the inner or outer table of the skull. It is produced by incomplete bone mineralization. Its appearance is frequent during neonatal and infant ages and is extremely rare outside of these age periods. The objective of this article is to present the case of a 16-year-old patient who presented a "ping-pong" fracture after a traumatic brain injury (TBI) and discuss the underlying physiopathogenesis of these types of fractures.

Case Description: A 16-year-old patient presented to the emergency department with a TBI, referring headaches and nausea. Non-contrast brain computed tomography displayed a left parietal "ping-pong" fracture. Laboratory tests showed hypocalcemia, subsequently diagnosing hypoparathyroidism. The patient remained under observation for 48 h. He was managed conservatively and started on calcium carbonate and vitamin D supplements with a favorable evolution. Hospital discharge was granted with TBI discharge instructions and warning signs.

Conclusion: The age of presentation of our case was atypical, according to the reported literature. When faced with a "ping-pong" fracture outside of an early age, underlying bone pathologies must be ruled out, which could potentially generate incomplete bone mineralization of the skull.

Keywords: Age, Bone mineralization, Depressed, Fracture, "Ping-pong"

INTRODUCTION

"Ping-pong" fractures are a type of depressed fracture, in which there is no rupture of the inner or outer table of the skull.[12,21] They are classified as congenital and acquired. The former occurs in utero, while acquired types develop during delivery or due to trauma at an early age.[8] Treatment can be conservative or surgical.[13,22] Less invasive treatments with suction reduction systems have also been described.[9,10]

Rahimi *et al.* described that Albucasis (Arabic surgeon who lived in the Middle Ages from 936 to 1013 AD) defined "ping-pong" fracture as follows: It is a fracture due to a fall or blow from a stone or similar, causing a dent in the surface of the bone, this occurs mainly in heads whose bones are soft, such as those of babies.[16] Like Albucasis, most authors acknowledge that this

type of fracture occurs during early ages (neonatal and infant period), due to incomplete bone mineralization of the skull. Its appearance outside of these age periods is extremely rare.[1,2,5,12,21,22]

There is abundant information in the literature regarding the appearance of these fractures during neonatal and infant periods, but little is known about their appearance at later ages. The objective of this article is to present the case of a 16-year-old patient who presented a "ping-pong" fracture after a traumatic brain injury (TBI) and discuss the underlying physiopathogenesis of these types of fractures.

CLINICAL CASE

A 16-year-old patient with no relevant history presented a TBI without loss of consciousness. The patient reported headaches, nausea, and dizziness. On physical examination, the patient was alert, oriented in all spheres, with reactive intermediate isochoric pupils, mobilizing 4 extremities on command, without sensory-motor deficits or gait disturbances.

A brain computed tomography (CT) without contrast was performed, displaying a "ping-pong" fracture with a diameter of 30.5 mm and depression of 9.2 mm. No intraparenchymal lesions were identified. Brain CT bone window and 3D reconstruction showed the fracture in the left parietal bone [Figures 1 and 2].

Laboratory tests were performed and showed low values of ionic and total serum calcium (4.8 mg/dL and 7.8 mg/dL, respectively). With this result, the serum parathyroid hormone (PTH) levels were analyzed, which showed a low value as well (6 mg/dL), diagnosing hypocalcemia and hypoparathyroidism. Consultation with the endocrinology service was made and the patient started on calcium carbonate and vitamin D supplements, remaining under observation in the general ward for 48 h with a favorable evolution. Due to the absence of neurological deficits, intraparenchymal lesions, large cosmetic defects, and/or symptomatic progression, conservative management was performed. Hospital discharge was granted with follow-up instructions and TBI warning signs. The patient did not attend the last control (6 months after the trauma), so the evolution of the depressed "ping pong" fracture is unknown. In the previous control (1 month after the trauma), there were no complications.

DISCUSSION

Bone mineralization begins during the embryonic phase of human development, but most of this process occurs in the third trimester of gestation. Osteoblasts control organic bone matrix production and subsequent calcium and phosphate deposition, generating progressive expansion of bone volume through an increase in trabecular thickness.[4] Mineral availability also influences osteoblastic and osteoclastic function and activity.[6] The peak of bone mineralization is generally reached between the ages of 18 and 25.[7,18] Pathological conditions that affect this process are related to abnormalities during the intrauterine and neonatal

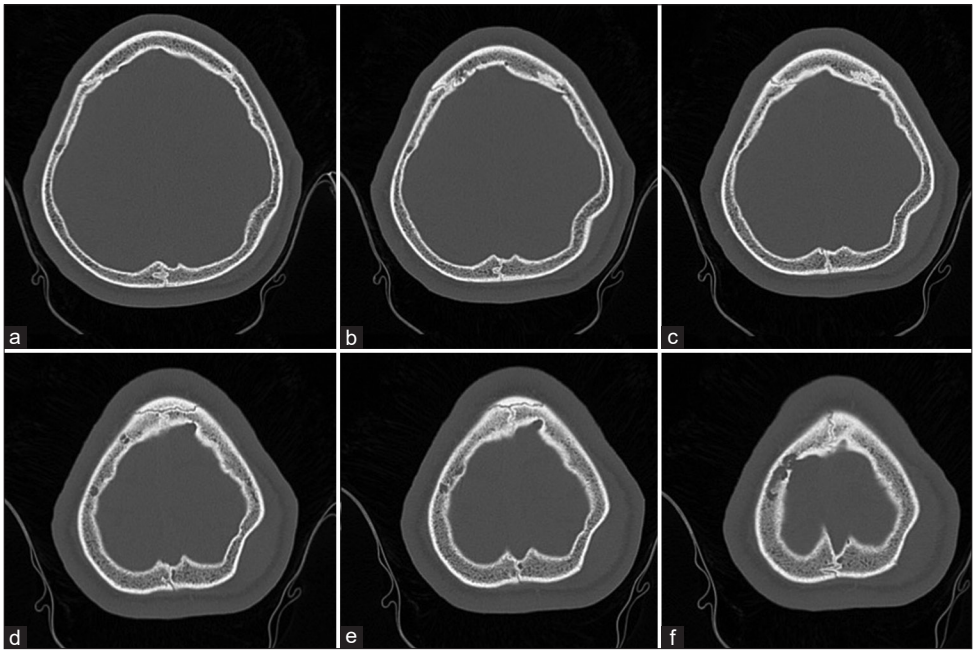

Figure 1: (a-f) Computed tomography scan bone window where we observe a depressed "ping-pong" fracture in the left parietal bone.

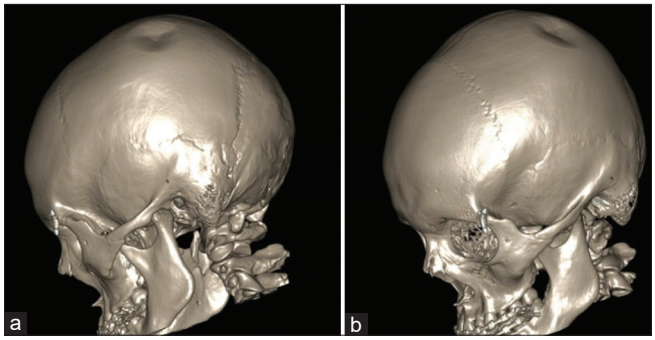

Figure 2: (a and b) 3D bone reconstruction where we observe a depressed "ping-pong" fracture in the left parietal bone.

period as well as nutritional, endocrinological, and/or autoimmune pathologies, among others. In this way, loss of bone mineralization and therefore deterioration of the microarchitecture of bone tissue generates a laxer tissue with greater susceptibility to bone damage.[7,11,14,18,20]

There are two types of depressed fractures, those with rupture of the inner and outer table of the skull and those without rupture of them. These last ones are called "ping-pong" fractures.[16] According to the literature, "ping-pong" fractures occur during early ages (neonatal and infant period) due to incomplete bone mineralization of the skull.[2,5,21,22] Prakash *et al*. presented one of the largest series of depressed skull fractures, declaring that the most frequent age of onset is between 16 and 30 years. However, in this series, more than 90% of the patients did not present "ping-pong" fractures since they presented rupture of the inner and/or outer table of the skull.[15] The only clearly documented report of a ping-pong fracture outside of the neonatal and infant period was described by De Paul Djientcheu *et al*., who reported a case of a ping pong fracture in a 17-year-old patient.[3] The authors did not mention the patient's medical history in their publication.

Rubin *et al*. conducted a study to measure the characteristics of bone tissue in patients with hypoparathyroidism before and after the administration of recombinant human PTH (rh PTH). The authors concluded that while rh PTH may have short-term beneficial effects on cancellous bone microarchitecture, further studies are required to explore whether the treatment of hypoparathyroidism with rh PTH affects skeletal strength and fracture incidence.[19] As part of the study, Young's modulus (described by Rho *et al*.[17]) was also measured in bone tissue. Unfortunately, due to limited hospital resources, we were unable to administer rh PTH to our patient.

A possible explanation for the appearance of "ping-pong" fractures outside the neonatal and infant period is undiagnosed nutritional, endocrinological, and/or autoimmune disorders (among others), which could potentially generate incomplete mineralization of the skull.[7,11,14,18,20] We believe that this could be an explanation of what happened in our patient, who was diagnosed with hypoparathyroidism.

CONCLUSION

We have presented the case of a 16-year-old patient who presented a "ping-pong" fracture after a TBI. The age of presentation was atypical, according to the reported literature. When faced with a "ping-pong" fracture outside of an early age, underlying bone pathologies (nutritional, endocrinological, and/or autoimmune, among others) must be ruled out, which could potentially generate incomplete bone mineralization of the skull. However, we believe that more cases must be studied to accurately reveal the physiopathogenesis of these fractures outside their predominant age range.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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