



Auricular calcification in a patient with traumatic brain injury and adrenal insufficiency: clinico-radiographic correlation with 3D cinematic rendering

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Case description

A 47-year-old man with a history of traumatic brain injury (TBI) presented to the hospital with altered mental status. His initial brain injury occurred 3 years before when he was found at the bottom of a staircase and admitted to the hospital with Glasgow Coma Scale score of 6. He was found to have extensive bifrontal brain contusions overlying the anterior cranial fossa. Due to elevated intracranial pressure, he underwent urgent bicoronal craniectomy in addition to tracheostomy and gastrostomy placement. He subsequently developed chronic, refractory hyponatremia requiring daily sodium chloride tablets and new-onset orthostatic hypotension which improved with fludrocortisone. He had no history of documented hypercalcemia, abnormal thyroid function tests, elevated glucose, or hemoglobin A1C levels. Non-contrast head CT was performed to assess confusion and somnolence. Axial CT images revealed bilateral auricular cartilage calcification which had not been recognized on physical examination (Fig. 1a).

Discussion

Auricular calcification (AC), sometimes called “petrified auricle”, is a rare clinical entity which was first described in 1866 by the anatomist Bochdalek [1]. The pathogenesis of AC remains elusive. Reported cases have been attributed to trauma, thermal exposure/injury, and systemic inflammatory disorders (polychondritis, dermatomyositis, etc.), invoking bone morphogenetic signaling pathway disruption

as a mechanism. Interestingly, AC is also associated with endocrinopathies, including diabetes mellitus, hypothyroidism, hypopituitarism, and adrenal insufficiency [2]. Clinically, most patients are asymptomatic and AC is often detected incidentally.

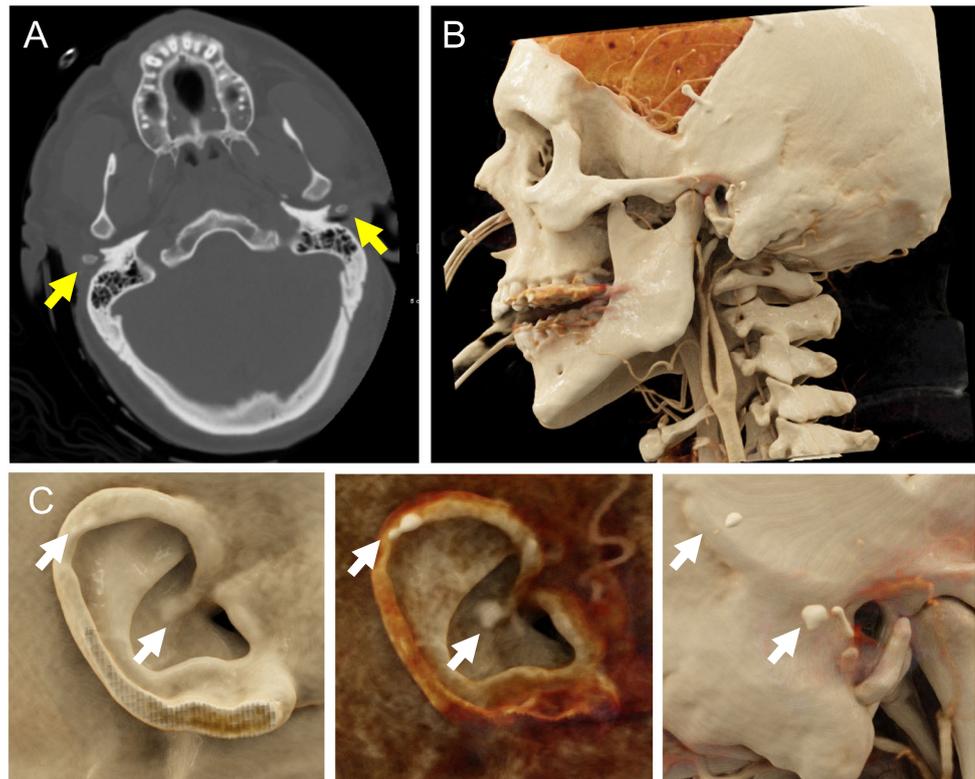
Neuroendocrine dysfunction due to anterior pituitary gland injury is common in TBI and may result in transient or permanent dysfunction [3, 4]. In a study of 50 patients with moderate-to-severe traumatic brain injury, Agha et al. found evidence of ACTH deficiency in 16% [5]. In this case, the patient’s post-traumatic hyponatremia (123–130 mmol/L) was initially suspicious for SIADH; however, serum osmolality levels remained normal (>275 mmol/L). Interestingly, arterial blood gas showed compensated respiratory alkalosis (pH 7.44, PCO₂ 28 mmHg) with low serum bicarbonate (range 20–21 mmol/L). Decaux et al. showed that this pattern of low total bicarbonate (TCO₂) and compensated respiratory alkalosis can differentiate hyponatremia related to ACTH deficiency from SIADH in which TCO₂ levels are typically normal [6]. Together, these clinical findings and auxiliary laboratory studies along with the finding of bilateral AC were felt to be consistent with post-traumatic secondary adrenal insufficiency.

Historically, plain radiography was used to identify dense calcification in the auricle when clinically suspected on physical examination. In this case, CT was diagnostic of AC, showing nearly symmetric, dense auricular calcification (Fig. 1a). CT data were post processed using 3D cinematic rendering (3DCR), a novel form of volume rendering which generates photorealistic images. 3DCR simulates nonuniform light ray distribution which approximates the behavior of natural light scatter [7]. The resultant images clarify surface textures and enhance depth perception, for example, showing the sequela of frontal lobe brain injury in this patient (Fig. 1b). Here, 3DCR was used to perform a “virtual” physical examination of the auricle. By manually adjusting the CT window/level settings, tissues of

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Fig. 1 Non-contrast head CT identifies bilateral auricular calcification (yellow arrows, **a**). 3D cinematic rendering of CT acquisition data highlights the post-surgical changes of bifrontal craniectomy **b**. **c** shows serial adjustment of CT window/level settings to evaluate different tissue layers of the auricle, revealing underlying calcification (white arrows)



differing radiographic density (Hounsfield Units) can be selectively emphasized or suppressed (Fig. 1c). This allows visualization of the skin, subcutaneous soft tissues, and ossified structures discreetly, contrasting the superficial appearance of the auricle with underlying foci of calcification.

This case highlights AC as an unusual manifestation of post-traumatic neuroendocrine injury and introduces a new radiographic technique (3DCR). 3DCR shows promise as a tool for visualizing complex anatomy and clinico-radiographic correlation of systemic manifestations of endocrine disease.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the patient's spouse, who serves as a legal health-care proxy, for publication of this case report and the accompanying images. The patient and the consenting health-care proxy were given access to this paper and images for approval prior to submission. A copy of the consent is available for review by the Editor-in-Chief of this journal on request.

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References

1. V. Bochdalek, Verknocherung der aurecula. *Prag. Vierteljahr.* **89**, 33–46 (1866)
2. W.A. High, M.J. Larson, M.P. Hoang, Idiopathic bilateral auricular ossificans: a case report and review of the literature. *Arch. Pathol. Lab. Med.* **128**, 1432–1434 (2004). [https://doi.org/10.1043/1543-2165\(2004\)128<1432:IBAOAC>2.0.CO;2](https://doi.org/10.1043/1543-2165(2004)128<1432:IBAOAC>2.0.CO;2)
3. M.K. Childers, J. Rupright, P.S. Jones, O. Merveille, Assessment of neuroendocrine dysfunction following traumatic brain injury. *Brain. Inj.* **12**, 517–523 (1998). <https://doi.org/10.1080/026990598122476>
4. L.A. Behan, J. Phillips, C.J. Thompson, A. Agha, Neuroendocrine disorders after traumatic brain injury. *J. Neurol., Neurosurg. & Psychiatry* **79**, 753–759 (2008). <https://doi.org/10.1136/jnnp.2007.132837>
5. A. Agha, B. Rogers, D. Mylotte et al. Neuroendocrine dysfunction in the acute phase of traumatic brain injury. *Clin. Endocrinol. (Oxf.)*. **60**, 584–591 (2004). <https://doi.org/10.1111/j.1365-2265.2004.02023.x>
6. G. Decaux, W. Musch, R. Penninckx, A. Soupart, Low plasma bicarbonate level in hyponatremia related to adrenocorticotropin deficiency. *J. Clin. Endocrinol. Metab.* **88**, 5255–5257 (2003). <https://doi.org/10.1210/jc.2003-030399>
7. M. Eid, C.N. De Cecco, J.W. Nance et al. Cinematic rendering in CT: a novel, lifelike 3D visualization technique. *AJR Am. J. Roentgenol.* **209**, 370–379 (2017). <https://doi.org/10.2214/AJR.17.17850>