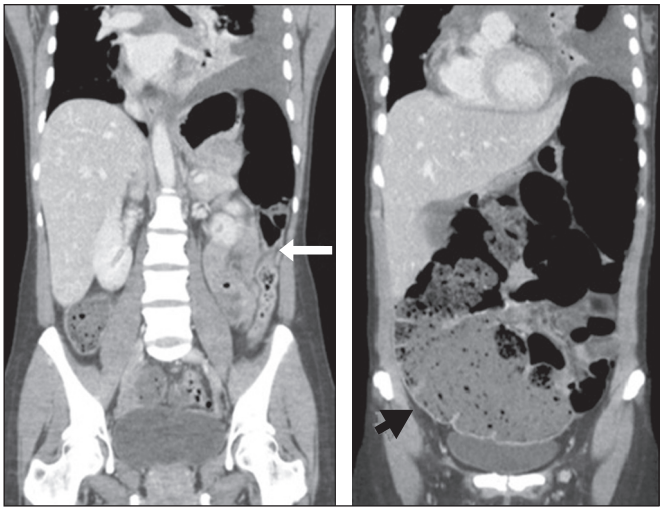

Figure 2. Coronal reconstruction of a CT scan, providing a better view of the transitional zone, where an abrupt transition to a normal caliber segment is observed, with no evident occlusive lesion (arrow). Note the marked dilation of the cecum, which measured 14 cm in diameter (arrowhead). Left pleural effusion can also be seen.

decompression and enema use were not considered because of the risk of cecal perforation. Therefore, the pseudo-obstruction was confirmed surgically. Thereafter, the patient was treated with gastric rest and her electrolyte levels were monitored.

Ogilvie's syndrome was named after William Heneage Ogilvie, who, in 1948, described a disorder of gastrointestinal motility, with dilation of the cecum and right colon in the absence of mechanical obstruction, that was autonomic in origin, with suppression of parasympathetic activity and activation of sympathetic activity[1].

The acute form of Ogilvie's syndrome arises from an autonomic imbalance, with a mismatch between parasympathetic and sympathetic activity, which are downregulated and upregulated, respectively. The distal colon is often atonic, whereas the proximal colon can still be functional[2]. Some chemotherapeutic agents have been implicated, such as those in the rituximab-cyclophosphamide-doxorubicin-vincristine-prednisone regimen, as have factors such as trauma, acute myocardial injury, electrolyte disturbances, hypothyroidism, renal failure, and neuropathy[2]. Lee et al.[3] observed that cancer patients developed Ogilvie's syndrome two to ten days after infusion of vincristine, the syndrome resolving after its discontinuation. Sandler et al.[4] found that patients treated with vincristine experienced abdominal pain and constipation within the first 4–72 hours after receiving the drug. Neutropenia and the use of antibiotic therapy have also been implicated in the development of the syndrome[3].

The symptoms of Ogilvie's syndrome include abdominal distension, abdominal pain, vomiting of fecal matter, and constipation[1,5]. Signs of peritonitis can indicate cecal perforation with pneumoperitoneum[6], especially when the distension is greater than 12 cm and lasts for more than six days. For evaluating diseases of the colon, CT has been shown to be the method of choice[7–11]. In Ogilvie's syndrome, CT is a useful for identifying the obstruction and determining the underlying cause[12], the main findings being dilation extending from the cecum to the transverse colon, with a transition zone in the splenic flexure, where the caliber of the adjoining loop is considerably smaller. The treatment involves the use of parasympathomimetic agents that increase colonic motility[13], endoscopic decompression or right hemicolectomy, the last being required in the presence of cecal ischemia or perforation.

Colonic pseudo-obstruction is associated with the use of chemotherapy. It is characterized by dilation of the loops of the colon and transitional zone. Attention should be paid to signs of perforation and the risk of death from cecal rupture.

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Pontine tegmental cap dysplasia accompanied by a duplicated internal auditory canal

Dear Editor,

A 48-year-old female with cognitive and auditory deficits presented for evaluation prior to cochlear implantation. Among her parents and four siblings, there was one brother with mental disability of unknown cause. Physical examination revealed ataxia. An electrophysiological study of hearing revealed the absence of waves from the cochlear nerve and of auditory brainstem pathways evoked by 95 dB nHL clicks and 500–1000 Hz tone bursts (also at an intensity of 95 dB nHL). There was also an absence of otoacoustic emissions in both ears, indicating profound sensorineural hearing loss. Computed tomography (CT) of the ears showed a narrow, duplicated internal auditory canal, one canal containing the facial nerve and the other containing

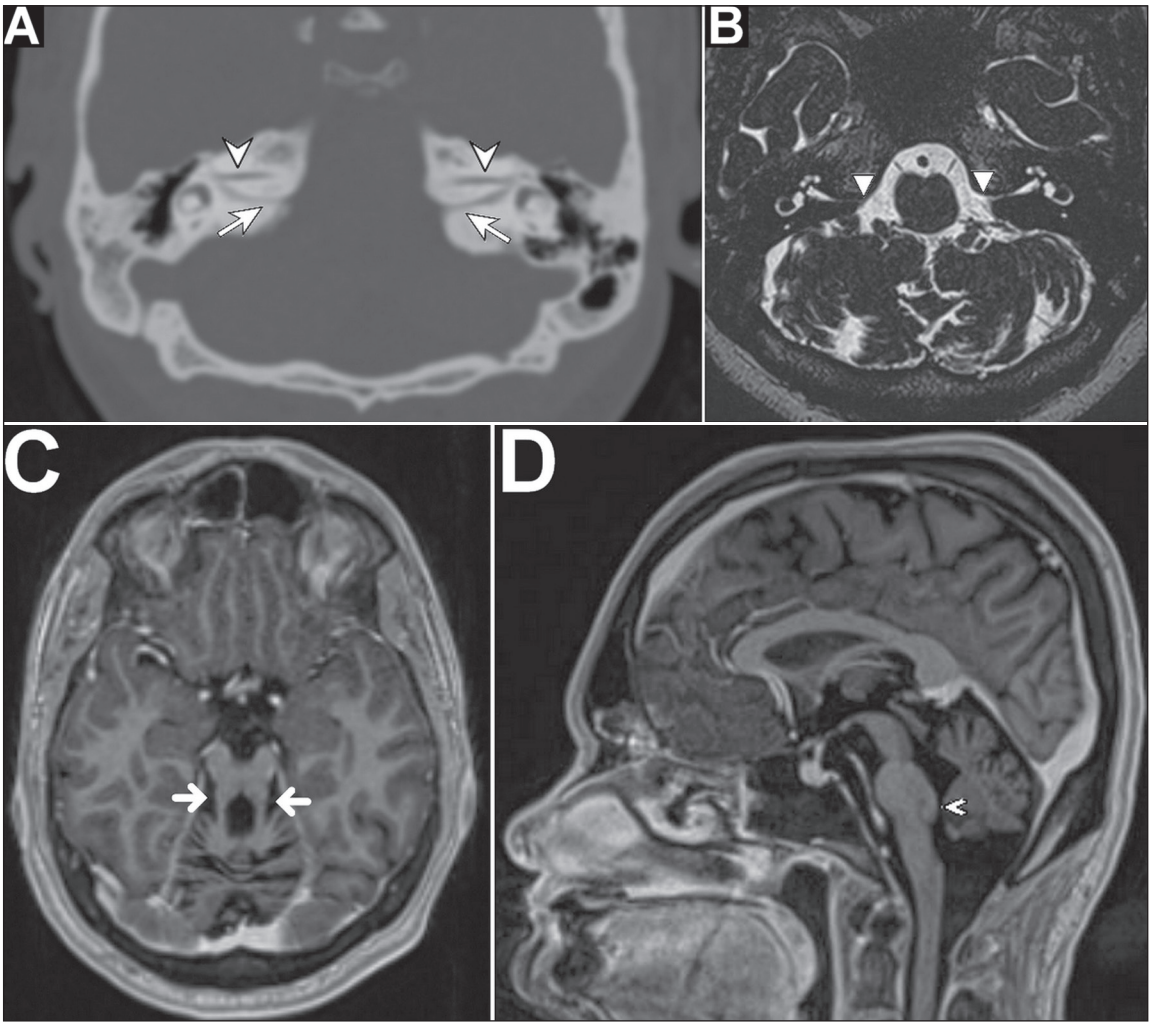

Figure 1. A: Oblique axial CT scan of the ears, with bone window settings, showing a narrow, duplicated internal auditory channel, one channel containing the facial nerve (arrowhead) and the other containing the vestibulocochlear nerve (arrow). **B:** Axial T2-weighted MRI scan of the brain and ears, revealing the absence of the eighth cranial nerve (arrowhead). **C:** Oblique axial T1-weighted, volumetric, intravenous contrast-enhanced MRI of the brain and ears, showing elongated, discretely lateralized superior cerebellar peduncles (arrows), similar in appearance to the molar tooth sign. **D:** Sagittal T1-weighted, volumetric, intravenous contrast-enhanced MRI of the brain and ears, showing cerebellar hypoplasia with a dysplastic aspect and with a reduction in the volume of the pons, especially in its ventral aspect, presenting a small prominence on the posterior surface projecting into the fourth ventricle (arrowhead).

the vestibulocochlear nerve (Figure 1A), together with a discrete reduction in the volume of the pons and cerebellum. In addition to the duplicated internal auditory canal, magnetic resonance imaging (MRI) of the brain and ears revealed the following: absence of the eighth cranial nerve (Figure 1B); elongated, discretely lateralized superior cerebellar peduncles, with an appearance similar to the molar tooth sign (Figure 1C); pons with a dysplastic aspect and a reduction in its volume, especially in the ventral region, presenting a small prominence, on the posterior surface, projecting into the fourth ventricle; and cerebellar hypoplasia, mainly in the vermis (Figure 1D). On the basis of those findings, the patient was diagnosed with pontine tegmental cap dysplasia (PTCD).

Cerebellar hypoplasia/hypogenesis can be seen in cases of metabolic disorder, exposure to teratogens, congenital infection or genetic disorders[1]. The molar tooth sign is observed in the axial plane of CT scans and, more clearly. of MRI scans at the junction between the rhombencephalon and mesencephalon, classically in the presence of cerebellar vermis hypoplasia/agenesis, deep interpeduncular fossa; Superior, poorly oriented, thickened and elongated superior cerebellar peduncles[2].

PTCD is a brainstem malformation[1,3,4], initially described in 2007 by Barth et al.[5]; to date, fewer than 50 cases have been reported[4]. The main signs and symptoms are auditory deficiency, in 92% of cases; cognitive deficit, in 76%; deglutition disorders, in 64%; facial paralysis, in 60%; abnormal eye movement, in 60%; trigeminal paresthesia, in 60%; ataxia, in 56%; hypotonia; cyclic vomiting syndrome; and various neurological disorders of the third to the eighth cranial nerves[4,6,7]. Other potential characteristics of PTCD include hypoplasia of the pons (notably in its ventral aspect); a mass of ectopic dorsal pontine fibers protruding into the fourth ventricle; hypoplasia/agenesis of the middle and inferior cerebellar peduncles; elongation of the superior cerebellar peduncles; cerebellar vermis hypoplasia/agenesis; absence or malformation of the inferior olivary nuclei; hypogenesis/absence of the third to eighth cranial nerves; costovertebral deformities; and cardiovascular anomalies[1,3–8]. PTCD can exhibit a feature similar to the molar tooth sign, although with lateralized, tapered superior cerebellar peduncles[1,4,6]. The differential diagnoses include pontocerebellar hypoplasia, as well as a number of syndromes[2,8,9]: Joubert; Dekaban-Arima; Senior-Loken; COACH; Váradi-Papp; Malta; and Moebius.

Although a duplicated internal auditory canal is extremely rare, it is found in at least 46% of all cases of PTCD. The two canals are often narrow (with a caliber of less than 2.0 mm) and accompanied by hypogenesis/agenesis of the eighth cranial nerve, which typically contraindicates cochlear implantation[4,8,10,11]. Some authors have reported differentiated cases in which the division is made by a bony septum, proposing that the term "partitioned" (rather than "duplicated") be used in such cases[11].

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