Radiology Case Reports 18 (2023) 3560–3564

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journal homepage: www.elsevier.com/locate/radcr

Case Report

Seizure caused by intraparenchymal hemorrhage from migration of mandibular dental wire through foramen ovale in a child: A case report ☆

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ARTICLE INFO

Article history:
Received 9 January 2023
Revised 9 July 2023
Accepted 14 July 2023

Keywords:
Foramen ovale
Trigeminal nerve
Mandibular dental wire
Intraparenchymal hemorrhage
Seizure

ABSTRACT

The foramen ovale is a structure that allows for the extracranial passage of multiple significant intracranial structures, most notably the mandibular branch of the trigeminal nerve (CN V_3). Here we report the case of a 12-year-old male who presented to the emergency department (ED) with a two-day history of nausea and emesis and a one-day history of altered mental status. Prior to presentation, he started speaking only Spanish, which was unusual because he primarily speaks English. He was also showing signs of absence seizures. Computed Tomography (CT) showed his orthodontic wire had migrated and was entering his skull through the foramen ovale, terminating within the inferior temporal lobe. Associated with the wire was an intraparenchymal hemorrhage. Imaging indicated the sparing of the internal carotid artery and its major branches, suggesting the hemorrhage was likely venous in nature. The wire was then safely removed with no complications. Same day and follow-up neurologic exams all demonstrated no deficit in CN V_3 or any of the other surrounding structures. To our knowledge, this is the first case described in the literature in which a foreign object penetrated the skull floor through the foramen ovale.

Introduction

The foramen ovale is a structure formed from neural crest-derived mesenchyme located on the infratemporal surface of the greater wing of the sphenoid bone [1,2] It allows for the passage of multiple significant structures between the infratemporal fossa and the intracranial space. One such structure is the mandibular branch of the trigeminal nerve (CN V_3), which supplies sensation to the lower face, jaw, and anterior tongue, as well as motor input to the muscles of mastication [3]. Other structures include the accessory meningeal artery,

☆ Competing Interests: There are no declarations of interest to disclose for this case report.

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https://doi.org/10.1016/j.radcr.2023.07.026

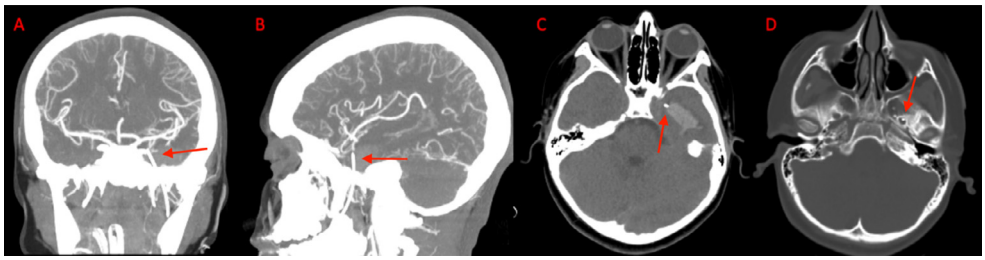

Fig. 1 – (A & B) Coronal and Sagittal view of the CT angiogram demonstrating the wire penetrating through the skull floor. (C) Axial view of CT demonstrating the wire in the temporal lobe and the associated intraparenchymal hemorrhage. (D) Axial Bone Window CT demonstrating the wire entering through Foramen Ovale.

which supplies the trigeminal ganglion; the lesser petrosal nerve, which supplies the otic ganglion; and an emissary vein, which allows for cranial-cerebral anastomosis [4–6].

The external opening of the foramen ovale is close to many other significant structures. It is posterolateral to the foramen rotundum, which contains the maxillary branch of the trigeminal nerve (CN V_2), responsible for sensory innervation to the middle third of the face [7]. Posterolateral to the foramen ovale is the foramen spinosum, which contains the middle meningeal artery that supplies the posterior dura mater [1]. Posteromedial to the foramen ovale is the carotid canal, which contains the internal carotid artery, sympathetic nerve plexus, and internal carotid venous plexus [8]. Here we describe the case of an orthodontic wire traversing the left foramen ovale with termination in the inferior left temporal lobe.

Case presentation

A 12-year-old male presented to the emergency department (ED) with a two-day history of nausea and emesis and a one-day history of altered mental status. Per the parents' report, the patient had his braces replaced one month prior. Two weeks after the replacement, he complained about jaw pain, and his family noted that they could no longer visualize the wire. They went to an outside urgent care, which prescribed antibiotics for what they thought was a gland infection. The day prior to his presentation, the patient started to speak only in Spanish, which is unusual for him since English is his primary language. The following morning, his parents brought him in for suspected seizures, which fit the description of absence seizures. The patient was initially taken to an outside hospital (OSH), where a computed tomography (CT) scan showed his orthodontic wire had migrated into his left temporal lobe, causing an associated intraparenchymal hemorrhage. He was given vancomycin, sedated with midazolam, and intubated at the OSH before being transferred.

Upon arrival at our ED, an X-ray was obtained, which confirmed a metallic foreign body entering his skull. He was started on metronidazole and ceftriaxone due to his high risk of infection, was further sedated with a bolus of propofol and a fentanyl drip, and was sent for a CT head and neck angiography. Imaging confirmed the orthodontic wire entering his skull via the foramen ovale and terminating within the temporal lobe (Fig. 1). Associated with the wire was an intraparenchymal hemorrhage measuring 2.4 × 1.6 cm with early signs of vasogenic edema. Imaging also indicated the sparing of the major vessels within the area. The wire was safely removed with no complications.

After the removal of the wire, a repeat CT showed complete removal with no new hematoma or worsening of the original hematoma. The patient's sedation was weaned, and he was extubated. A full neurologic exam completed upon his awakening showed all cranial nerves were intact. He endorsed some dizziness and blurry vision; however, no diplopia was noted. Follow-up magnetic resonance imaging (MRI) on hospital day two showed a stable intraparenchymal hematoma with vasogenic edema and no evidence of infection (Fig. 2). On hospital day four, the patient's pain was well controlled with acetaminophen, and he denied any nausea or vomiting. He was discharged at this time with a walker to assist with ambulation.

The patient presented to the clinic two weeks later due to parental concern for focal and absence seizures, but no tonic-clonic seizures. He also had episodes of uncontrollable laughter, consistent with gelastic seizures. MRI obtained at this point showed appropriate evolution of the hematoma and no signs of infection. His neurologic exam was negative, and he no longer used the walker for assistance. He was referred to neurology for seizure management and was maintained on 600 mg of levetiracetam twice daily for a total of 1200 mg daily. The following month, he followed up with his family care physician. On physical exam, the patient demonstrated an intact left CN V_3 with preserved sensory and motor function. CN V_2 was also intact. A 2-month follow-up MRI showed a collapsed porencephalic defect at the original site of the trauma (Fig. 3). His most recent follow-up was with neurology 3.5 months after his initial presentation. He is being managed with 300 mg of oxcarbazepine twice daily and reports only one seizure in the preceding month.

Discussion

During embryological development, the skull base is formed by endochondral ossification, whereas the remainder of the cranial vault and facial bones undergo intramembranous ossification [2]. The developing skull base divides into two main

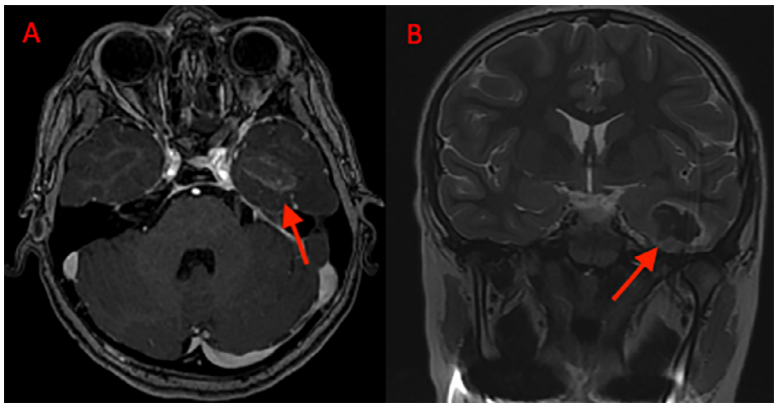

Fig. 2 – (A) Axial MRI with contrast demonstrating residual hematoma within the temporal lobe two days after removal. (B) Coronal MRI without contrast also demonstrating temporal lobe hematoma.

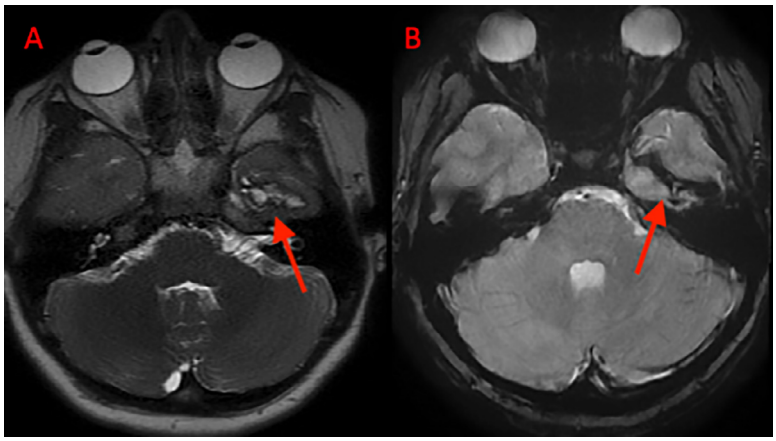

Fig. 3 – (A) An axial T2 SSFSE and (B) an axial T2 GRE MRI at the 2 months follow-up demonstrating the collapsed porencephalic defect in the left temporal lobe.

cartilage types during development. The prechordal chondrocranium, anterior to the rostral notochord boundary, contains neural crest-derived mesenchyme. The chordal chondrocranium, composed of paraxial mesoderm, is located on the posterior side of the boundary [2,9]. Prior to condensation of the mesenchyme, blood vessels, and cranial nerves develop, ensuring proper formation and location of skull bone foramina [2].

The foramen ovale originates from the posterior wing of the sphenoid bone and opens into the infratemporal fossa. Its location is surrounded by many important structures and the foramina through which they travel [10,1]. Of the multiple structures traveling through the foramen ovale, the most notable is the mandibular branch of cranial nerve five- the trigeminal nerve (CN V_3). The mandibular nerve is the largest of the three branches and is a mixed nerve. It is responsible for afferent sensory innervation from the temporal region and lower third of the face, as well as motor innervation to the muscles of mastication. CN V_3 also provides sensory innervation to the anterior two-thirds of the tongue but does not include taste [11,12]. Damage to CN V_3 can lead to disruption and loss of sensation in the aforementioned areas and difficulty with chewing. Another complication that is common following trigeminal nerve injury is trigeminal neuralgia; trauma is the most common identifiable cause. Trigeminal neuralgia can occur in any of the three branches and is characterized by sudden, excruciating pain that is short and can profoundly affect the quality of one's life [10,13,14]. The patient presented here had no sensory loss on physical exam and did not report any weakness of his jaw muscles or symptoms associated with trigeminal neuralgia. Therefore, we concluded that CN V_3 did not sustain any significant damage from the dental wire and may have even been untouched.

CN V_3 is not the only constituent of the foramen ovale as the foramen is also traversed by emissary veins, the accessory middle meningeal artery, and the lesser petrosal nerve [4–6]. The emissary vein of the foramen ovale connects the cavernous sinus and the pterygoid plexus, allowing for anastomosis and a route for venous blood ultimately into the internal jugular vein. Damage to the emissary vein can lead to an epidural or subdural hematoma [5]. Similarly, the accessory middle meningeal artery passes through the foramen ovale. The accessory middle meningeal artery is the intracranial branch from the ascending intracranial ramus

of the middle meningeal artery. Damage to this artery could also lead to bleeding and an epidural hematoma. It is a small artery that is absent in roughly 18% of people [4]. It is unlikely either of these vessels was damaged due to the lack of blood in the subdural and epidural space. Finally, the lesser petrosal nerve also travels through the foramen ovale to the otic ganglion. The lesser petrosal nerve is mainly a branch of the glossopharyngeal nerve but receives contributions from the vagus and facial nerves. It carries preganglionic parasympathetic fibers to the parotid gland [6]. There was also no evident deficit of the lesser petrosal nerve.

Apart from having many significant constituents, the intracranial portion of the foramen ovale is surrounded by many noteworthy structures. It is bordered anteromedially by the foramen rotundum, medially by the cavernous sinus, posteromedially by the carotid canal, and posterolaterally by the foramen spinosum [10]. These foramina are home to multiple important structures responsible for blood supply and facial sensation. The carotid canal is located around 10 mm from the foramen ovale and contains the internal carotid artery, which is responsible for anterior intracranial blood circulation [10,1,15,8]. Intracranial penetrating trauma to the internal carotid artery is a rare complication; however, extracranial trauma to the internal carotid artery occurs in approximately 5%-6% of penetrating neck trauma. External trauma carries a mortality rate of around 100% without surgical intervention. The mortality rate decreases to 6%-33% in those who survive the surgery. Penetrating intracranial internal carotid artery trauma most commonly results in a traumatic intracranial aneurysm. The rupture of this aneurysm has a mortality rate of approximately 50%. Profound neurologic deficits in survivors often persist after rupture [16]. Had the orthodontic wire traveled as little as 10 mm in the posteromedial direction, the internal carotid artery could have sustained damage, likely leading to detrimental effects.

Similar to the carotid canal, the foramen rotundum is close to the foramen ovale, laying 9 mm in the anteromedial direction. The vital structure traversing this foramen is the maxillary branch of the trigeminal nerve (CN V_2). CN V_2 and CN V_3 are close together as they emerge from the Gasserion, or trigeminal, ganglion and travel to their respective foramina. CN V_2 provides sensation from the middle third of the face, which includes the area inferior to the eyes to the superior lip. Damage to this nerve would have led to a deficit of sensation in this distribution; however, the patient did not experience sensory changes in this area [3].

Finally, only 3 mm away is the foramen spinosum through which the middle meningeal artery travels, a terminal branch of the maxillary artery. The middle meningeal artery provides blood supply to much of the dura mater. It is a common culprit of epidural hematomas following trauma. After entering the skull through the foramen spinosum, the middle meningeal artery is tightly adherent to the skull [17]. This kept it clear of the orthodontic wire as it coursed upwards through the foramen ovale.

Due to the curve of the orthodontic wire, it took a superiorly angled course, sparing the major vessels and nerves within the area. Similarly, the curve kept the wire towards the anterior aspect of the foramen ovale (Fig. 1D). The anterior location, coupled with the superior trajectory, likely spared the constituents of the foramen ovale, most notably CN V_3. We suspect the wire entered the foramen ovale over a multiple-day span and was continually pushed in through chewing and the use of the patient's jaws until it terminated in the temporal lobe.

Conclusion

The patient described in this case report did not sustain measurable damage to any of the structures within or surrounding the foramen ovale despite his orthodontic wire traversing the foramen. Injury to the carotid artery in this case likely would have been detrimental; however, this artery and its major branches were spared. Furthermore, as indicated by his normal follow-up neurologic examination, he did not sustain measurable damage to CN V_3 or the nearby CN V_2. To our knowledge, this is the first case described in the literature in which a foreign object penetrated the skull floor through the foramen ovale.

Patient consent

I certify that the patient presented here gave written consent for the publication of this case report and the images entailed within it.

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