Case Report

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Middle Ethmoid Artery Epistaxis: A Case of Post-Traumatic Severe Bleeding

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Abstract:

Post-traumatic epistaxis is a common sequela of head trauma, typically arising from injury to the nasal mucosa or disruption of vascular structures, including the anterior-posterior ethmoid and sphenopalatine arteries. The ethmoid arterial system exhibits considerable anatomical variation, with middle ethmoid arteries identified in approximately 30-36% of cases. This report presents a rare instance of post-traumatic epistaxis originating from the middle ethmoid arteries in a pediatric patient with congenital thrombocytopenia, confirmed through computed tomography imaging and intraoperative assessment. The case emphasizes diagnostic and therapeutic challenges, highlighting the role of endoscopic surgical intervention in achieving hemostasis. Understanding the anatomical complexity of the ethmoid arterial system is crucial for accurate diagnosis and optimal management in similar cases.

Keywords: epistaxis, middle ethmoid artery, trauma

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Introduction

Post-traumatic epistaxis is frequently encountered in emergency settings following head injuries, with the most common sources of bleeding being the anterior ethmoidal artery, posterior ethmoidal artery, and sphenopalatine artery[1-3]. However, the ethmoid arterial system exhibits significant anatomical variability, and in some cases, additional branches, such as the middle ethmoid arteries (MEAs), may contribute to sinonasal vascular supply. Reports indicate atypical presentations of the MEAs in approximately 30-36% of cases[4-6]. The involvement of MEAs in post-traumatic epistaxis is rare, posing unique diagnostic and therapeutic challenges. A thorough understanding of the anatomical course and variability of the ethmoid arteries is essential for managing cases where conventional approaches fail to achieve hemostasis. This report highlights an unusual presentation of persistent post-traumatic epistaxis originating from the MEAs in a pediatric patient with congenital thrombocytopenia. The case emphasizes the significance of endoscopic surgical intervention in achieving hemostasis when conservative measures prove ineffective.

Case report

A 6-year-old female patient with a known history of congenital thrombocytopenia presented to the emergency department with persistent epistaxis following a fall. The patient sustained a head injury after tripping and striking her forehead on the ground. Initial assessment revealed moderate nasal bleeding that was unresponsive to standard first-line management, including nasal packing and topical hemostatic agents. On physical examination, the patient was hemodynamically stable, but active bleeding from the left nasal cavity was noted. Anterior rhinoscopy and nasal endoscopy identified continuous bleeding from the middle turbinate and superior ethmoidal recess (SER). Given her underlying thrombocytopenia, further hematologic evaluation was conducted, revealing a platelet count of 50,000/μL. A computed tomography (CT) scan of the paranasal sinuses was performed to evaluate for fractures or hematomas. The imaging ruled out facial bone fractures but demonstrated opacification of the left ethmoid sinus, suggesting ongoing hemorrhage. The source of bleeding was traced to an atypical arterial structure consistent with the anatomical course of the middle ethmoid arteries (MEAs), confirming their involvement (Figure 1). Due to the persistence of epistaxis, despite conservative measures, surgical intervention was warranted.

The patient subsequently underwent endoscopic sinus surgery (ESS) under general anesthesia. The procedure included the following steps:

1. Uncinectomy and ethmoidectomy: initial exposure was achieved via uncinectomy and anterior/posterior ethmoidectomy, facilitating access to the ethmoid sinus and its arterial supply.

2. Identification of bleeding source: intraoperative assessment confirmed active bleeding from a small arterial branch coursing through the superior ethmoidal recess, consistent with the anatomical location of the MEAs (Figure 2).

3. Hemostatic techniques: hemostasis was achieved using electrocautery, supplemented with hemostatic agents, including Surgicel and Gelfoam. Additionally, a platelet transfusion was administered to correct thrombocytopenia, increasing the platelet count to 100,000/μL.

Postoperatively, nasal packing was undertaken to support clot formation, and the patient was monitored in the hospital for 48 hours. No further bleeding episodes were observed, and the patient was subsequently discharged with instructions for a follow-up evaluation. At the subsequent six-month follow-up, the patient remained free of recurrent epistaxis and experienced no complications, confirming the effectiveness of the intervention and overall management plan.

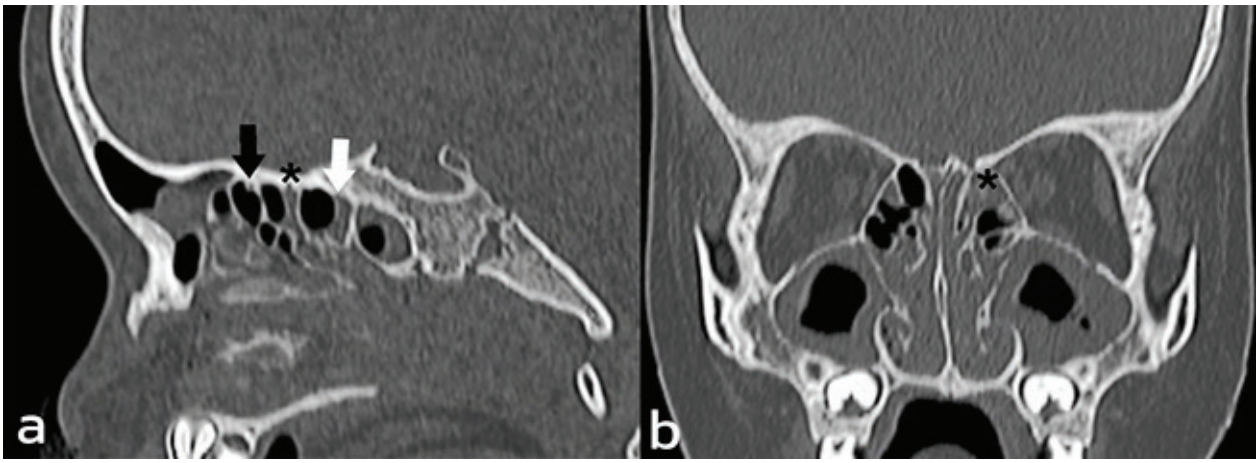

Figure 1 The sagittal (a) and coronal (b) sections of a computed tomography image of the paranasal sinuses illustrate the location of bleeding from the middle ethmoid arteries (indicated by an asterisk). The bleeding is located between the anterior ethmoid artery (indicated by a black arrowhead) and the posterior ethmoid artery (also indicated by a black arrowhead).

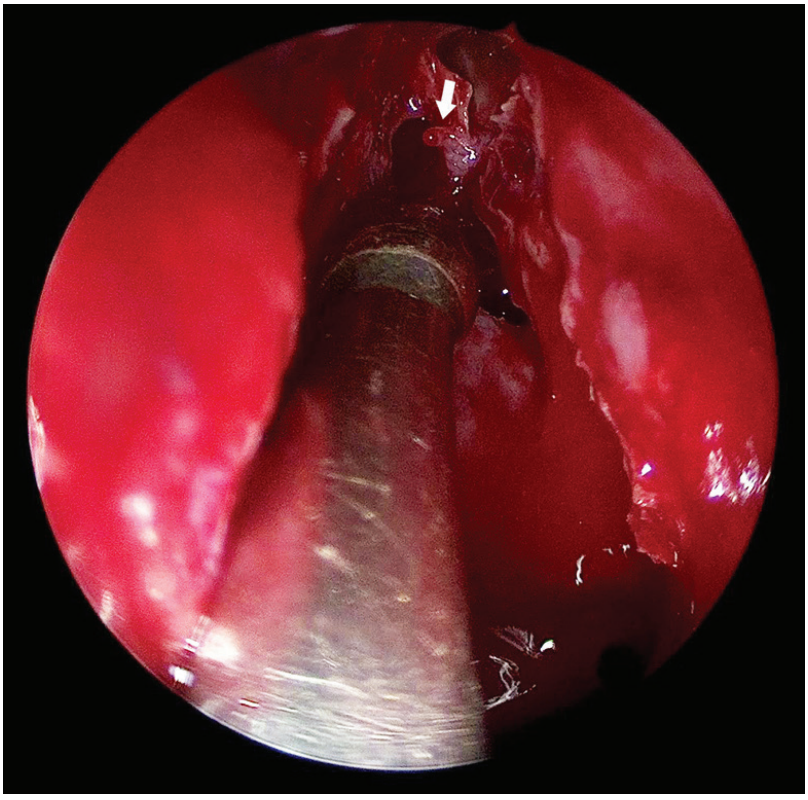

Figure 2 Illustrates the location of bleeding from the middle ethmoid arteries (indicated by a white arrowhead).

Discussion

Head trauma frequently results in nasal bleeding due to the disruption of the extensive vascular network within the sinonasal region. Blunt-force trauma can induce compression or shearing injuries, leading to vascular rupture. Post-traumatic epistaxis, characterized by bleeding from the nasal passages, commonly occurs following head trauma and typically results from lacerations of the nasal mucosa or tears in the ethmoidal or sphenopalatine arteries. This bleeding may present with an acute or delayed onset, often due to pseudoaneurysm formation[7-9].

The extent and severity of nasal bleeding depend on the force and direction of impact, which can directly affect the ethmoidal arteries. While post-traumatic epistaxis most frequently originates from the anterior ethmoidal artery (AEA) or the sphenopalatine artery, involvement of the MEAs remains rare. The presence of MEAs introduces additional complexity in epistaxis management, as these arteries are not consistently depicted in standard anatomical descriptions. These arteries play a variable role in sinonasal blood supply, with their presence and significance differing among individuals, yet their clinical significance remains underexplored. Variability in their origin, course, and anastomotic connections renders their involvement in epistaxis unpredictable and challenging to diagnose.

The MEAs are an uncommon arterial variation that arises between the AEA and the posterior ethmoid artery (PEA). In our case, the MEAs were identified intraoperatively based on their anatomical course within the superior ethmoidal recess, located between the AEA and PEA. Additionally, preoperative computed tomography (CT) imaging provided further confirmation, showing a distinct arterial structure consistent with the expected course of the MEAs[10].

Diagnosing MEAs-related epistaxis requires a high index of suspicion, particularly in cases where hemorrhage persists despite conventional interventions. Computed tomography angiography (CTA) or digital subtraction angiography (DSA) can aid in identifying unusual arterial contributors. However, CT imaging may provide sufficient diagnostic clues when bleeding patterns align with the anatomical course of the MEAs[11].

The management of post-traumatic epistaxis typically follows a stepwise approach:

Conservative measures: initial treatment includes nasal compression, administration of vasoconstrictive agents (e.g., oxymetazoline), application of topical hemostatic agents, and nasal packing.

Endoscopic interventions: if conservative measures fail, endoscopic cauterization or vessel ligation may be warranted. Endoscopic sinus surgery (ESS) allows precise localization and cauterization of the bleeding artery, thereby minimizing morbidity[12,13].

Embolization: in cases of severe or recurrent hemorrhage, angiographic embolization may be considered, particularly if pseudoaneurysm formation is suspected. However, embolization carries risks such as stroke or ischemia and is generally reserved for refractory cases[14].

In this case, the decision to proceed with ESS was guided by persistent hemorrhage despite conservative management, anatomical localization of the MEAs as the bleeding source, and the necessity for targeted hemostasis. The combined use of bipolar cautery was primarily used for hemostasis, as it provides more controlled coagulation while minimizing thermal spread to adjacent structures. This technique involves direct bipolar coagulation of the bleeding point, followed by the application of hemostatic agents (Surgicel and Gelfoam) to reinforce hemostasis.

The findings in this case align with those of Felippu et al.[2], who reported successful endoscopic management of post-traumatic epistaxis involving the ethmoidal arteries. Comparable studies emphasize that ESS offers several advantages over external ligation or embolization, including reduced morbidity, shorter hospital stay, faster recovery

time, and direct visualization of the bleeding source[15]. This case emphasizes the pivotal role of endoscopic techniques in managing complex epistaxis cases and highlights the necessity of understanding anatomical variations in sinonasal vascular supply in order to optimize patient outcomes.

Conclusion

Post-traumatic epistaxis involving the MEAs is a rare but significant clinical entity that requires careful evaluation and intervention. When initial management fails, endoscopic sinus surgery provides a minimally invasive yet highly effective approach for achieving hemostasis. This case emphasizes the importance of recognizing ethmoidal artery variations and integrating surgical expertise with imaging modalities for optimal patient outcomes. Future studies and case reports can further elucidate the role of MEAs in sinonasal bleeding, improving diagnostic and therapeutic strategies in this domain.

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Conflict of interest

None of the authors has any conflict of interest to declare, and no author maintains any proprietary interest in the products or concepts addressed in this article.

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