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Case report

Multidisciplinary management of a traumatic posterior meningeal artery pseudoaneurysm: A case report and review of the literature

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ABSTRACT

Background: Meningeal arterial injuries represent <1% of all blunt traumatic brain injuries (TBIs). Middle meningeal artery (MMA) lesions comprise the majority. However, there is little clinical data on posterior meningeal artery (PMA) injuries.

Case report: A 69-year-old man was brought to our trauma center after sustaining a fall inside a warehouse. He was GCS (Glasgow Coma Scale) 3 on arrival. Non-contrast CT (computed tomography) brain showed subarachnoid hemorrhage with diffuse cerebral edema and a basilar skull fracture. The patient subsequently underwent emergency ventriculostomy. Immediately after the procedure, further imaging with CTA (computed tomography angiography) head identified a hyperintense posterior cranial fossa lesion, prompting cerebral angiography with identification and embolization of a traumatic PMA pseudoaneurysm. The patient improved and was discharged to a long-term acute care facility. At 3 months post-discharge, the patient was eating, talking with family, and working aggressively with physical therapy.

Discussion: This case represents a functional neurologic outcome from a rare subset of TBI. Early CTA head imaging is not supported by limited literature, but allowed for expedient identification and definitive management of this PMA pseudoaneurysm. In the critical care setting, hyperosmolar therapy, CSF (cerebrospinal fluid) drainage, prompt enteral nutritional support, and early tracheostomy all represent evolving evidence-based strategies to optimize care for severe TBI.

Conclusions: The initial evaluation and management of severe TBI can be nuanced. Future research may refine indications for CTA head to the diagnostic evaluation of patients with both severe TBI and skull fractures.

1. Background

Traumatic meningeal pseudoaneurysms comprise <1% of all intracranial aneurysms. The most common mechanism of injury causing a meningeal artery pseudoaneurysm involves blunt lateral head trauma and classically associated with an epidural hematoma. There is an association with depressed skull fractures at the level of the pterion, a complex of five suture lines on the temporal skull behind which the middle meningeal artery (MMA) courses. By contrast, posterior meningeal artery (PMA) injuries are more clinically obscure. The literature contains relatively few documented PMA pseudoaneurysms, and those cases seem to point towards an association with higher energy mechanisms and increased severity of traumatic brain injury (TBI) [1]. This paper presents a case of severe TBI after a fall associated with a traumatic PMA pseudoaneurysm. The case discussion emphasizes the importance of multidisciplinary management with collaborative efforts between neurosurgery, neurointerventional radiology, and trauma/surgical critical care. This report aligns with the 2020 Surgical Case Report (SCARE) guidelines [2].

2. Case report

A 69-year-old man was brought to our trauma center after sustaining a fall from six feet onto a concrete floor. He was a Glasgow Coma Score (GCS) 3 with dilated, sluggish, yet reactive pupils on arrival. He had obvious blunt head trauma, including battle signs and bilateral proptosis on examination. He was promptly intubated, started on infusions of normal saline, 3% hypertonic saline, and 1 g loading doses of both tranexamic acid and levetiracetam given the clinical picture of severe TBI. He was administered DDAVP and platelets based on a history of

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home clopidogrel use. Emergent non-contrasted CT brain demonstrated a TBI, a basilar skull fracture, a diffuse subarachnoid hemorrhages, associated cerebral edema, but no midline shift, or uncal or falcine herniation (Fig. 1). Neurosurgery placed an emergent external ventricular drain. The initial pressures were below 20 mm Hg. Based on clinical suspicion, neurosurgery requested immediate CT angiography (CTA) of the head after ventriculostomy, which revealed a hyperintense focus of posterior cranial fossa blood products (Fig. 2). Neurointerventional radiology subsequently performed cerebral angiography and identified a trauma pseudoaneurysm with extravasation of the PMA, near the fracture (Fig. 3). Following this, neurointerventional successfully embolized the pseudoaneurysm with an ethylene vinyl alcohol copolymer.

Critical care was conducted in the trauma ICU for the remainder of the patient's hospitalization. On ICU day 3 the patient underwent tracheostomy, percutaneous endoscopic gastrostomy tube placement, and inferior vena cava filter insertion. Follow up CT examination a week later revealed no further pseudoaneurysm. His external ventricular drain was removed. He made slow progress and was discharged to a neurologic long-term acute care hospital (LTACH) on hospital day 30 with a neuro exam of GCS 9 T.

The patient made remarkable progress with neuro-rehabilitation after discharge. Follow-up at 3 months post-discharge showed that the patient is having verbal conversations with family members, eating, and drinking.

3. Discussion

We present a case of severe neurotrauma with a pattern of injury that is almost uniformly fatal or unsalvageable. Traumatic MMA pseudoaneurysms are very uncommon, with less than fifty cases reported in the English literature. Traumatic PMA pseudoaneurysms remain even rarer. This may be due to the fact that patients with these injuries incur a high mortality rate early in hospitalization. Another aspect unique to these cases is that the location of many PMA pseudoaneurysms are not easily accessible to conventional approaches for open neurosurgical intervention. Treatment of these uncommon and usually fatal injuries demands a collaborative effort with neurointerventional radiology. In our case, this proved successful in stopping hemorrhage, thereby temporizing cerebral edema and subsequent herniation.

PMA pseudoaneurysms are associated with posterior cranial fossa epidural hematomae (PFEDH). This phenomenon is most often because depressed skull fractures can tear the dura mater and the PMA. In these cases, subdural and subarachnoid hematomae can form from an occult epidural bleed, either from venous sinuses or meningeal arteries. In a review of 89 cases of PFEDH only three were identified in which an associated meningeal artery pseudoaneurysm [3].

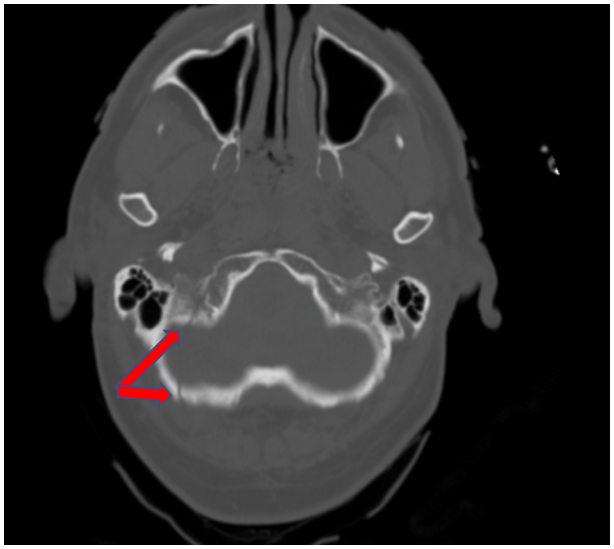

Fig. 1. Initial non-contrasted CT brain, showing the basilar skull fracture.

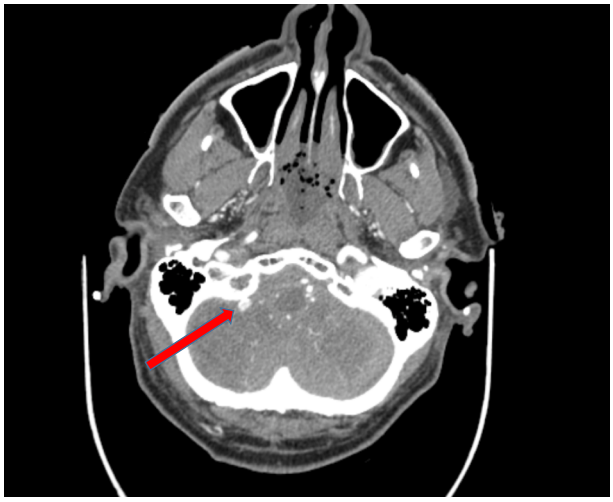

Fig. 2. CT Angiography of the head, demonstrating subarachnoid blood products with a hyperintense lesion in the cranial fossa, reviewed by neurosurgery, neuro-interventional radiology, and characterized as a likely traumatic posterior meningeal pseudoaneurysm.

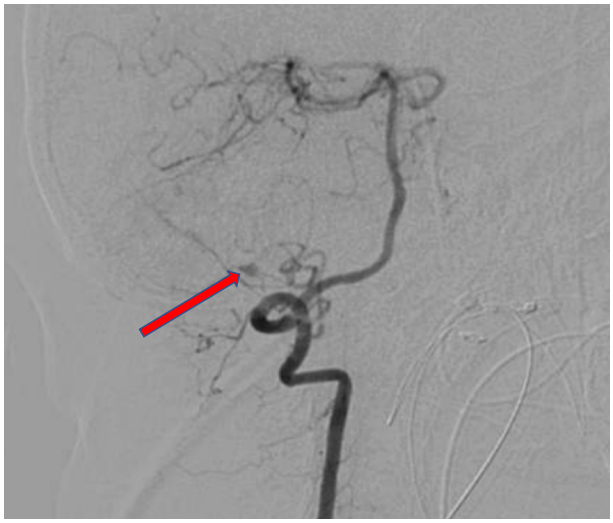

Fig. 3. Diagnostic angiography of the posterior meningeal artery showing the traumatic pseudoaneurysm with active extravasation, which was subsequently embolized.

Cerebral angiography and angioembolization has grown in popularity for diagnosis and intervention of meningeal arterial injuries [4,5]. Moreover, some meningeal artery branches, especially those in the posterior fossa, tend to be harder to access by means of traditional open neurosurgical vessel ligation. In our particular case, the lesion was low in the posterior fossa along the basilar skull and more feasible to treat with endovascular techniques.

Several factors are necessary in order to maximize the opportunity for patients with these injuries to have a functional recovery. Early recognition of severe TBI in the emergency department or trauma bay is crucial. Prompt reversal of coagulopathy and hyperosmolar therapy helps slow or temporize cerebral edema and impending herniation. Index CT imaging is usually focused on non-contrasted brain scans. For patients with severe TBI coupled with skull fractures, although no sufficient evidence is available, we still believe there is a role for obtaining

CTA head imaging. CTA head imaging also allows neurosurgical and neurointerventional consultants to collaborate simultaneously. This is helpful because for patients like the present case, a combination of neurosurgical procedures and endovascular neuro-interventional therapies may be necessary. The sequence of interventions can be tailored to each individual case. Intensive monitoring and control of intracranial hypertension, either through ventriculostomy or cranial decompression, is equally important as angioembolization for these injuries. In our case, by having prompt CTA imaging, neuro-interventional radiology had the opportunity to review the case and plan their intervention early during the initial resuscitative phase.

At the same time, available literature on the utility of CTA head imaging on arrival for patients with TBI has not yet found much benefit. A 2015 case-control study of 600 blunt TBI patients included a group of 132 patients who had CTA head imaging performed on arrival as a supplement to the typical non-contrasted CT brain. They concluded that CTA head results did not change patient management or disposition outcomes [6]. The authors called for abandoning the practice of obtaining CTA head imaging on arrival. While the practice of uniformly obtaining CTA head imaging may be not recommended, there may be a role for arrival CTA in select patients. In this present case report, however, early CTA head made our team realize that there was an identifiable injury which could be treated. If CTA was delayed or forwent altogether, the patient would have likely been treated expectantly in the trauma ICU after ventriculostomy and incorrectly considered to have an unsalvageable TBI. For these reasons, the trauma literature may benefit from further studies which investigate which patients should undergo immediate CTA head.

Clinical and radiographic parameters could be defined in order to refine an indication for immediate or early CTA head imaging. The decision for early CTA head imaging in this case was informed by the presence of skull fractures in the vicinity of vessels belonging to the posterior cerebral circulation. For future research, surgeons can search for methods to identify CTA head candidates based on immediate review of the initial non-contrasted CT brain from the CT control room. Patients with a neurologic assessment of GCS <6 accompanied by skull fractures, or subarachnoid blood products emanating along vascular distributions, may represent a more refined group for which CTA head on arrival is indicated. Identifying patients who may benefit from CTA during the initial evaluation also helps for practical reasons, meaning the patient only requires one trip to the CT scanner. This overall reduces interruptions in the resuscitation phase and allows for higher quality critical care.

Aside from definitive endovascular embolization of the traumatic PMA pseudoaneurysm, many surgical critical care principles were employed to optimize the patient's chances of functional recovery in this case. Hyperosmolar therapy, seizure prophylaxis, tranexamic acid, and early tracheostomy were all strategies used that can be tailored to each patient with severe TBI early during their hospitalization. The Brain Trauma Foundation (BTF), American College of Surgeons Trauma Quality Improvement Program (ACS-TQIP), and World Society for Emergency Surgery have all provided evidence-based practice recommendations for severe TBI from 2015 to 2020 (Tables 1 and 2) [7–9]. All three sets of recommendations are first-grade quality, however each document emphasizes different aspects of TBI care. For example, the latest WSES consensus focuses on providing the highest quality care to patients with severe TBI and polytrauma, but its scope is limited to the first 24 h of care [9].

Hyperosmolar therapy aims to alleviate or prevent cerebral edema, thereby reducing the risk or cumulative impact of cerebral herniation. Hypertonic saline (HTS) is commonly employed in 3% form, although other concentrations up to 23.4% are available. Mannitol is another effective hyperosmolar agent, but can cause hypotension based on its profound osmotic diuretic effect when undergoing renal clearance. Mannitol functions optimally for intracranial hypertension when given as a bolus of 1 g/kg, whereas HTS products are generally delivered as IV drip infusions in consideration of the perceived risk for central pontine myelinolysis. Many studies up to the level of large metanalyses have demonstrated no significant difference in severe TBI outcomes based on choice of hyperosmolar agent [10,11]. Some interesting case-control studies have found that 3% HTS can be superior to mannitol when given as a bolus, different from its typical administration in trauma [12,13]. Along these lines, the Continuous hyperosmolar therapy for traumatic Brain-Injured patients (COBI) trial in France is currently investigating the impact of 48 h continuous hyperosmolar therapy on 6 month TBI outcomes [14], as there has already been some evidence that this strategy may be more effective than intermittent bolus therapy. Nonetheless, perhaps the most important aspect in the trauma bay is how quickly the patient can begin receiving hyperosmolar therapy after arrival. At our center, we begin infusing 3% HTS at 30 mL/h from arrival for patients who are GCS 3 with dilated pupils as they are being transported to CT scanner. Somewhat paradoxically, supplying pre-hospital emergency responders with hyperosmolar therapy protocols for unresponsive patients with severe head trauma has not led to improved clinical outcomes when compared to standard pre-hospital normal saline therapy [15].

Tranexamic acid (TXA) is an inhibitor of plasminogen which has been shown to reduce hemorrhagic sequelae in trauma. The 2019

Table 1
Comparative Summary of Critical Care Practices for Severe Traumatic Brain Injury.

Comparative summary of critical care practices for severe traumatic brain injury		
	ACS-TQIP 2015 [7]	BTF 2016 [8]
Hyperosmolar therapy	Mannitol 0.25–1 g/kg every 6 h ***or*** 250 mL 3% HTS over 30 min ***or*** 30 mL 23.4% HTS[a]	Mannitol is effective to control ↑ICP except in cases of SBP <90 mm Hg[b]
CSF drainage	Intermittent EVD drainage is advised unless an ICP monitor is in place. Nonetheless, when draining CSF continuously, measured ICP measurements are not accurate	Continuous EVD drainage calibrated at level of midbrain is more effective than intermittent drainage; CSF drainage for patients GCS < 6 can be helpful to lower ICP during first 12 h after injury
Seizure prophylaxis	Not covered in ACS-TQIP 2015 Best Practices for TBI management	Phenytoin or levetiracetam therapy for 7 days[c]
Early tracheostomy	Tracheostomy performed for stable patients with severe TBI at <8 days post-injury	Recommended for reducing ventilator days for stable patients
Barbiturate therapy	Barbiturate coma therapy may be used for patients with malignant intracranial HTN who have not responded to aggressive treatment measures	High dose barbiturates are indicated for intracranial HTN refractory to medical/surgical therapies when patients are hemodynamically stable
Nutrition	Enteral nutrition should begin early, ideally 24–48 h post-injury	Enteral nutrition should begin by day 5 post-injury
Chemical DVT prophylaxis	Low and moderate risk patients can usually have chemical DVT prophylaxis at 24 h and 72 h post-injury, respectively; high risk patients may require IVCF placement[d]	Can be considered when the ICH is considered stable and benefit outweighs risk of ICH expansion

Abbreviations
BTF: Brain Trauma Foundation; ACS-TQIP: American College of Surgeons Trauma Quality Improvement Program; HTS: hypertonic saline; ICP: intracranial pressure; CSF: cerebrospinal fluid; IVCF: inferior vena cava filter
Notes
[a] Hypertonic saline therapy should be monitored with serum sodium and osmolality measurements every 6 h.
[b] BTF 2016 does not mention hypertonic saline therapy.
[c] Evidence is based on studies with phenytoin, not levetiracetam.
[d] At our institution, we follow the modified Berne-Norwood criteria for designating low, moderate, and high-risk TBI.

Table 2
Selected monitoring parameter goals for sever traumatic brain injury.

Selected monitoring parameter goals for severe traumatic brain injury	
ACS-TQIP 2015 [7]	WSES 2020 [8]
General monitoring goals in critical care for managing patients with TBI	Goals intended for the first 24 h of care for patients with severe TBI
PaO_2 > 100 mm Hg	PaO_2 60–100 is acceptable during emergency interventions
$PaCO_2$ 35–45 mm Hg	$PaCO_2$ 35–40 is acceptable during emergency interventions
SBP >100 mm Hg	SBP >100 mm Hg *or* MAP >80 mm Hg
ICP 20–25	No value given, but heavy emphasis placed on early intracranial pressure monitoring
CPP >60 mm Hg	CPP > 60 mm Hg
INR < 1.4	INR < 1.5; TEG or ROTEM is recommended to guide volume resuscitation
Platelets > 75,000	Platelets > 50,000 for emergent operations, higher for neurosurgery
Hemoglobin > 7 g/dL	Hgb > 7 g/dL with consideration of transfusion at higher Hgb values for "at risk" populations. MTP should be 1:1:1 (RBCs: Plasma:Platelets)
Body temperature 36–38 °C	–
Glucose 80–180 mg/dL	–
Serum sodium 135–145	–
pH 7.35–7.45	–
SpO_2 > 95%	–

Abbreviations
ACS-TQIP = American College of Surgeons Trauma Quality Improvement Program, WSES = World Society for Emergency Surgery, SBP = systolic blood pressure, MAP = mean arterial pressure, ICP = intracranial pressure, CPP = cerebral perfusion pressure, INR = international normalized ratio.

CRASH-3 trial implicated reduced mortality for patients who receive TXA within 3 h of closed head injury. Current studies suggest that TXA should be given immediately upon arrival as 1 g IV loading dose administered over 10 min, followed by a 1 g IV drip infused over 8 h [16]. We follow this pattern of management for all TBI at our center.

Early tracheostomy is another management strategy which has become popular in recent literature. The timeline for "early" has been defined as ≤8 days after injury in observational studies conducted with data from ACS-TQIP database (Table 1) [17]. Current evidence, while varying between studies, associates early tracheostomy for severe TBI with benefits including reduced ICU length-of-stay, reduced hospital length-of-stay, reduced ventilator days, decreased incidence of pneumonia, and improved neurological outcome [18,19]. While early tracheostomy may be desirable in severe TBI, it should not take precedence over the initial period of resuscitation. The primary goal of treating TBI is preventing secondary insult to the brain tissue, so tracheostomy should also be avoided in patients early in their hospital course who continue to have intracranial hypertension, hemodynamic lability, or high oxygenation requirements (generally FiO2 > 50% and PEEP > 10 cm H2O) [7].

4. Conclusions

This report presented a rare case of traumatic PMA pseudoaneurysm in which the patient had a functional and meaningful neurological outcome. Initial management to achieve definitive control of the vascular injury was an Multidisciplinary effort between trauma, neurosurgery, and neurointerventional radiology. CTA head imaging shortly after arrival was tremendously useful in identifying the lesion. Ventriculostomy was crucial in temporizing the patient's intracranial hypertension in the acute period. Definitive management involved neurointerventional embolization of the posterior meningeal artery. Critical care management strategies included hyperosmolar therapy, TXA, and early tracheostomy. Overall, severe TBI is incredibly nuanced, and treatment strategies should be tailored for each individual patient. In certain patients with severe TBI and skull fractures, there may be a role for developing protocols to include CT head angiography in order to not miss meningeal arterial injuries. Early identification of meningeal vascular injuries, coupled with aggressive multidisciplinary management, can be lifesaving and its value cannot be overstated.

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Ethical approval

This report was conducted in compliance with ethical standards. Informed written consent has been obtained and all identifying information is omitted.

Informed consent

Informed written consent has been obtained and all identifying information is omitted.

Registration of research studies

This is a case report study.

CRediT authorship contribution statement

JE, AE, MM, MR– Conception of study, acquisition of data, analysis and interpretation of data, drafting the article, and revision of article.

MR – Management of case, revision of article, and final approval of the version to be submitted.

JE, AE, MM, MR – Approval of the final version for submission.

Declaration of competing interest

No conflicts of interest.

References

[1] V.E. Toro, J.F. Fravel, T.A. Weidman, et al., Posttraumatic pseudoaneurysm of the posterior meningeal artery associated with intraventricular hemorrhage, AJNR Am. J. Neuroradiol. 14 (1) (1993 Jan-Feb) 264–266.
[2] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, SCARE Group, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230, https://doi.org/10.1016/j.ijsu.2020.10.034.
[3] T.N. Lui, S.T. Lee, C.N. Chang, W.C. Cheng, Epidural hematomas in the posterior cranial fossa, J. Trauma 34 (2) (1993 Feb) 211–215.
[4] Q.A. Shah, R.W. Hurst, Endovascular treatment of a traumatic pseudo aneurysm of the middle meningeal artery, Radiol. Case Rep. 1 (3) (2015 Nov 6) 73–76.
[5] F. Goetz, H. Lanfermann, M. Nakamura, J.K. Krauss, B. Hong, Life-threatening hemorrhage from a traumatic aneurysm of the posterior meningeal artery: treatment with coil embolization, Cen. Eur. Neurosurg. 72 (3) (2011 Aug) 162–165.
[6] L. Naraghi, A. Larentzakis, Y. Chang, et al., Is CT angiography of the head useful in the management of traumatic brain injury? J. Am. Coll. Surg. 220 (6) (2015 Jun) 1027–1031.
[7] Committee on Trauma, ACS TQIP Best Practices in the Management of Traumatic Brain Injury, American College of Surgeons, Chicago, 2015.
[8] N. Carney, A.M. Totten, C. O'Reilly, et al., Guidelines for the management of severe traumatic brain injury, in: Neurosurgery, Fourth edition 80(1), 2017 Jan 1, pp. 6–15.
[9] E. Picetti, S. Rossi, F.M. Abu-Zidan, et al., WSES consensus conference guidelines: monitoring and management of severe adult traumatic brain injury patients with polytrauma in the first 24 hours, World J. Emerg. Surg. 14 (2019) 53.
[10] J. Gu, H. Huang, Y. Huang, H. Sun, H. Xu, Hypertonic saline or mannitol for treating elevated intracranial pressure in traumatic brain injury: a meta-analysis of randomized controlled trials, Neurosurg. Rev. 42 (2) (2019 Jun) 499–509.
[11] H. Chen, Z. Song, J.A. Dennis, Hypertonic saline versus other intracranial pressure-lowering agents for people with acute traumatic brain injury, Cochrane Database Syst. Rev. 12 (12) (2019 Dec 30), CD010904.

[12] H.S. Mangat, X. Wu, L.M. Gerber, et al., Hypertonic saline is superior to mannitol for the combined effect on intracranial pressure and cerebral perfusion pressure burdens in patients with severe traumatic brain injury, Neurosurgery. 86 (2) (2020 Feb 1) 221–230.
[13] F. Cheng, M. Xu, H. Liu, W. Wang, Z. Wang, A retrospective study of intracranial pressure in head-injured patients undergoing decompressive craniectomy: a comparison of hypertonic saline and mannitol, Front. Neurol. 9 (2018 Jul 31) 631.
[14] A. Roquilly, S. Lasocki, J.D. Moyer, et al., COBI (continuous hyperosmolar therapy for traumatic brain-injured patients) trial protocol: a multicentre randomized open-label trial with blinded adjudication of primary outcome, BMJ Open 7 (2017), e018035.
[15] J.V. Rosenfeld, A.I. Maas, P. Bragge, et al., Early management of severe traumatic brain injury, Lancet. 380 (9847) (2012 Sep 22) 1088–1098.
[16] CRASH-3 Trial Collaborators, Effects of tranexamic acid on death, disability, vascular occlusive events and other morbidities in patients with acute traumatic brain injury (CRASH-3): a randomised, placebo-controlled trial, Lancet 394 (10210) (2019 Nov 9) 1713–1723.
[17] A.S. Alali, D.C. Scales, R.A. Fowler, et al., Tracheostomy timing in traumatic brain injury: a propensity-matched cohort study, J. Trauma Acute Care Surg. 76 (1) (2014 Jan) 70–76 (discussion 76-8).
[18] Q. Lu, Y. Xie, X. Qi, et al., Is early tracheostomy better for severe traumatic brain injury? A meta-analysis, World Neurosurg. 112 (2018 Apr) e324–e330.
[19] A. Elkbuli, R.I. Narvel, P.J. Spano 2nd, et al., Early versus late tracheostomy: is there an outcome difference? Am. Surg. 85 (4) (2019 Apr 1) 370–375.