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Clinical Case Studies

Traumatic pneumocephalus as a possible early sign of acute spinal cord injury: Case report

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ABSTRACT

Background: Traumatic pneumocephalus (TPC) following craniofacial injuries is common, but isolated TPC secondary to pneumorrhachis (PR) is the rare result of upward gas migration from the spinal canal. In the absence of craniofacial and grossly unstable spinal fractures, the etiology of TPC in polytrauma can be elusive and an underlying diagnosis of acute spinal cord injury (SCI) can be easily missed. We report the first polytrauma case where TPC was the most reliable early sign of SCI.

Case Description: A 34-year-old polytrauma female with imaging findings of TPC, which was later found to be secondary to an underlying SCI. As a focused exam could not be performed at admission, the TPC was first attributed to undiagnosed craniofacial injuries. Tertiary survey revealed the patient being paraplegic and MRI workup demonstrated an acute SCI at the T3-T4 level. PR was the most likely cause of TPC in the absence of other craniofacial injuries.

Outcome: The patient did not have a meaningful recovery given the extensive hemispheric infarcts, spinal cord injury, and respiratory failure.

Conclusions: Although uncommon, TPC may be an important radiographic sign suggesting the possibility of an underlying SCI in polytrauma patients. especially when focused neurologic assessment is limited at admission. Polytrauma patients with TPC and PR in the absence of coexisting craniofacial fractures require an urgent spine consultation by the ER physician, with possible early spine MRI workup. We suggest a diagnostic algorithm for the early identification of SCI in polytrauma patients presenting with TPC and propose considering 3 groups which may have different risks for SCI based on their clinical presentation and the presence of PR.

Background

Pneumocephalus can occur following trauma, cranial surgeries, or even spontaneously [1–3]. Traumatic pneumocephalus (TPC) is a frequent radiographic sign of craniofacial injuries. In such cases the route of intracranial gas entry directly involves the fracture site in the skull. TPC can also be caused by air entering intracranially through the sinuses, mastoid, or other aerated air cells [4–7]. TPC in the absence of craniofacial fractures is a much rarer occurrence and has been reported as a possible consequence of upward gas migration from the spinal canal [8–10].

Pneumorrhachis (PR) was first described by Newbold et al. in 1987 and is characterized by the presence of air in the spinal canal, with most reported cases affecting the cervical and thoracic regions [10,11]. Reported etiologies of PR include iatrogenic interventions, anesthetic complications, trauma, asthma, or other indolent causes [12,13]. Traumatic PR is quite infrequent and can manifest itself on imaging as epidural, subdural or subarachnoid gas [1,14]. Even more uncommon, craniofacial fractures resulting in TPC may lead to air migration in the craniocaudal direction toward the spinal canal [15]. Finally, delayed PR can manifest itself as a subarachnoid-pleural fistula in cases of spinal trauma [12,13].

The coexistence of TPC and PR during secondary trauma survey has been previously reported in the setting of penetrating or blunt thoracic trauma resulting in complex skull base fractures and simultaneous spinal fractures [5,16–18]. Similarly, various studies have hypothesized that PR may occur due to subcutaneous emphysema, pneumomediastinum, or pneumothorax with secondary air migration into the spinal canal

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[5,19–24]. To our knowledge, this is the first reported case of TPC with PR caused by blunt chest trauma and no evidence of skull fractures on imaging, or a clear pathway for air migration from the mediastinum into the spinal canal.

Polytrauma patients presenting with TPC, with or without PR, and without other craniofacial fractures are at a special risk of having an undiagnosed SCI, especially when a detailed neurogical exam cannot be obtained at admission. We here report the case of a 34-year-old polytrauma female who arrived in hypovolemic shock, and was found to have a diagnosis of isolated TPC with PR during secondary survey. In the setting of polytrauma and established shock, the patient was intubated and a focused neurologic exam could not be performed. TheTPC was initially attributed to a possible undiagnosed craniofacial fracture. Later, the source of the PR was found to be more likely secondary to a dural tear from an underlying spinal cord injury [25]. We propose a diagnostic algorithm for the early identification of SCI in polytrauma patients with TPC, based on their initial neurologic assessment and the presence of PR. We believe this simple tool may prove valuable to spine surgeons, especially taking into account the uncommon nature of such type of presentation and the value of early identification of an underlying SCI in this population.

Clinical presentation

A 34-year-old Caucasian female with no significant past medical history presented to the ED as polytrauma following a motor vehicle collision without signs of penetrating injuries. She was found unresponsive at the scene for an unknown period of time following the accident and was intubated early for airway protection. The patient arrived at the hospital in hypovolemic shock with blood pressure of 55/40 mm Hg, having received aggressive resuscitation with a massive transfusion protocol. Primary survey was positive for bilateral hemopneumothoraces, so the patient underwent bilateral chest tube insertion and placement of a central line for vasopressor administration. Her initial Glasgow Coma Scale (GCS) was 7 out of 15, but her neurological exam was unreliable, and it was unclear whether she had lower extremity weakness, given her shock status and suspected unstable pelvic fractures. Following successful resuscitation and pelvic stabilization, the patient underwent a CT scan of her head, cervical, thoracic, and lumbar spine. A head CT revealed extensive pneumocephalus involving the bilateral frontal lobes, left sylvian fissure, left lateral ventricle, and basal cisterns (Fig. 1). However, there was no evidence of other intracranial injuries or craniofacial fractures (Fig. 2). Additional injuries sustained included a left superior pubic

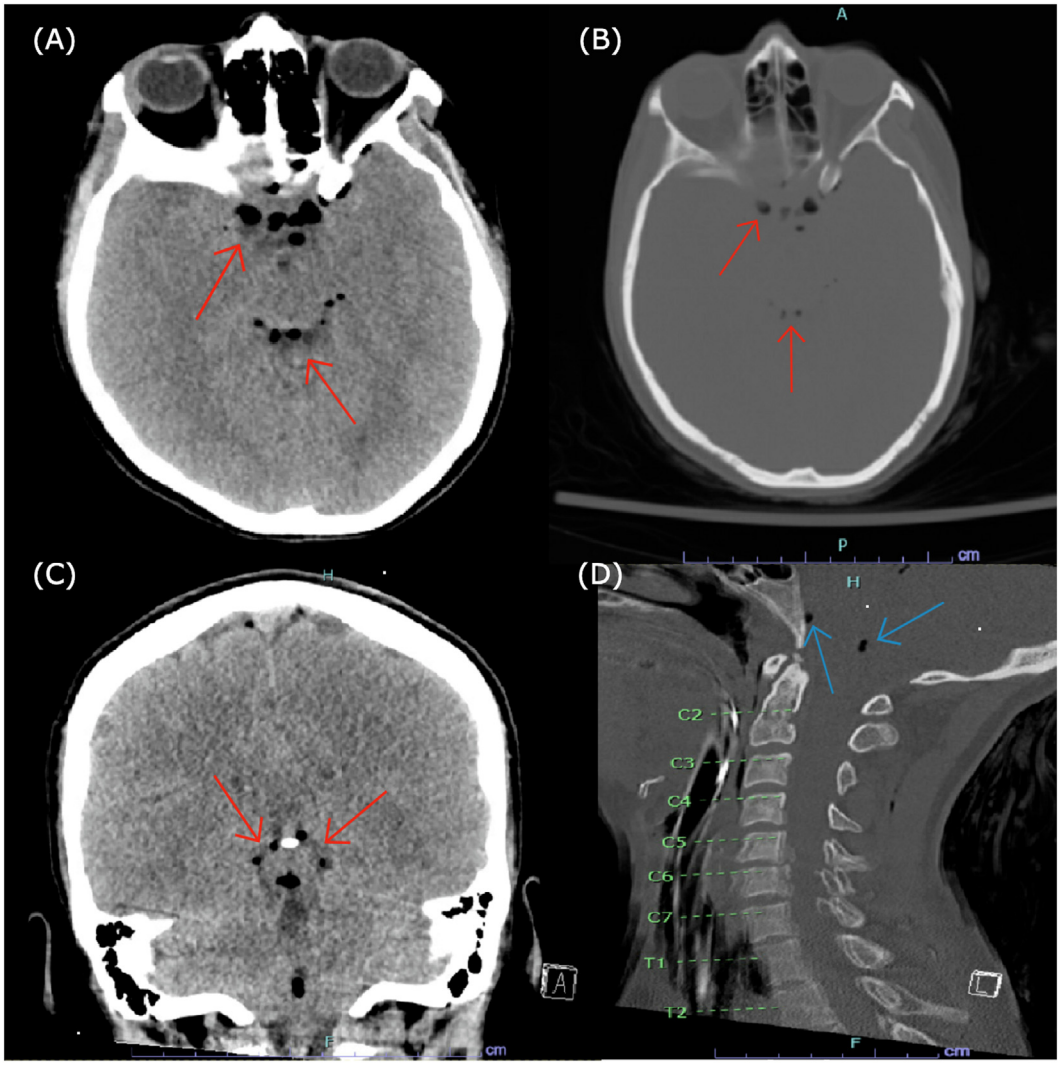

Fig. 1. Axial CT head without contrast showing brain (A) and bone (B) windows, as well as a mid-coronal CT section (C), at the level of the basilar cisterns demonstrating extensive pneumocephalus (red arrows) involving the peri-mesenchephalic and ambient subarachnoid cisterns. There is no evidence of other intracranial injuries or craniofacial fractures. Mid-sagittal section of CT cervical spine without contrast (D) was negative for fractures or pneumorrhachis, but pneumocephalus is present in the pre-medullary cistern and fourth ventricle (blue arrows).

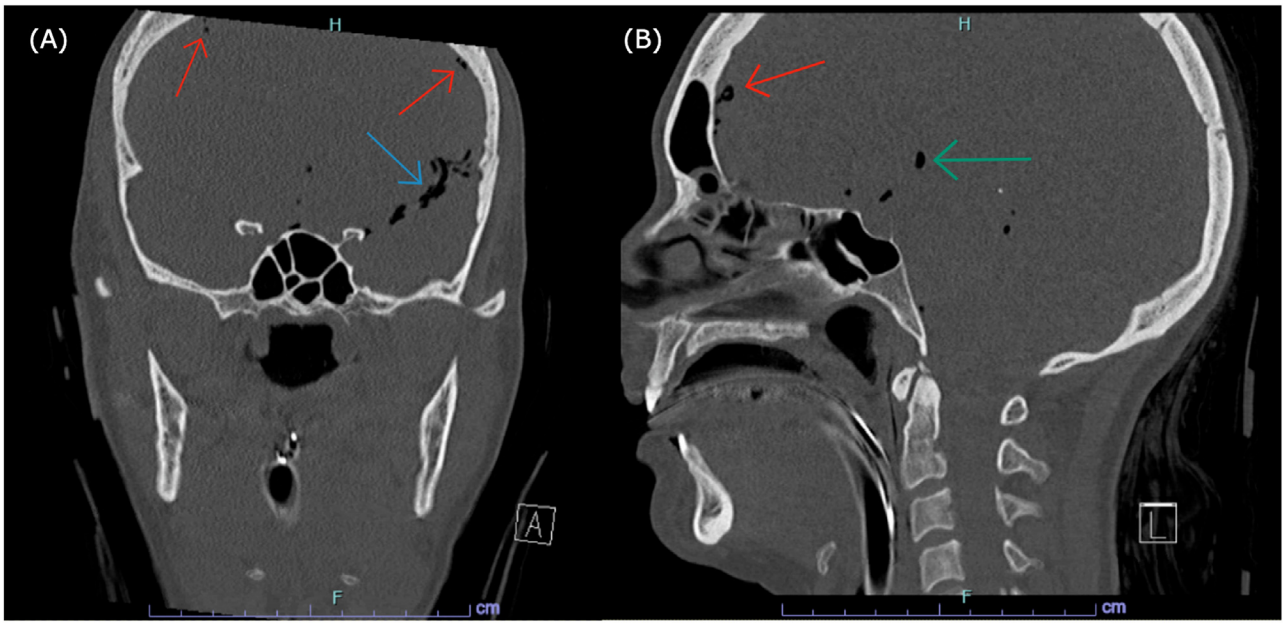

Fig. 2. Coronal CT of the face without contrast- bone window (A) at the level of the ethmoid sinuses and mid-sagittal CT face bone window (B) showing extensive pneumocephalus in the bilateral frontal lobes (red arrow), left sylvian fissure (blue arrow), and the left lateral ventricle (green arrow). There is no evidence of craniofacial or skull base fractures.

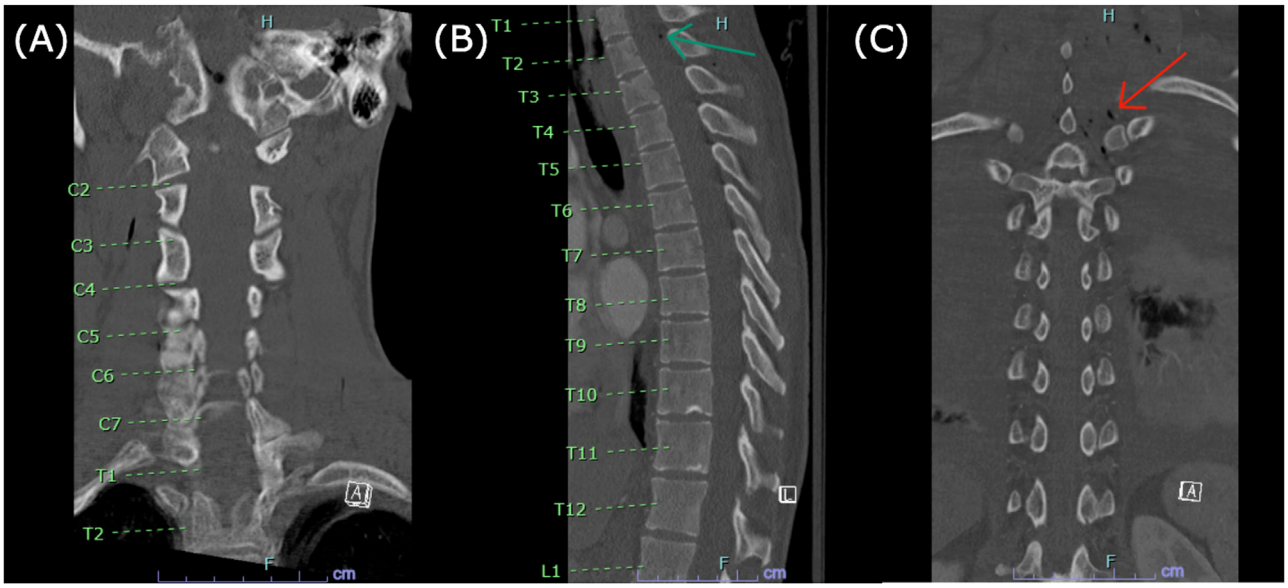

Fig. 3. A coronal section of the CT of the cervical spine (A), sagittal section of the thoracic spine CT (B), and a coronal section of the thoracic spine CT (C) revealed a T3 hairline fracture extending from the anterior cortex to the inferior endplate. It was possible to verify a subtle finding of pneumorrhachis at the level of T2 in Figure B (green arrow). The presence of gas adjacent to the paraspinal muscles and facet joints on the left side in Figure C (red arrow) was initially attributed to the bilateral pneumothoraces and previous chest tube insertion.

ramus fracture, and sacral insufficiency fractures. Initial spine CT-scans revealed a line of fracture at T3 extending from the inferior endplate to the anterior cortex with minimal height loss. The presence of subtle PR was observed (Fig. 3B**).** Initial CT further did not reveal any signs of gross instability or facet joint involvement with preserved interspinous process distance.

Management

Within 24 hours, during tertiary survey, the patient was eventually found to be paraplegic, and therefore, a spine consultation was placed. Mean arterial pressure augmentation (MAP) was initiated for suspected spinal cord injury. An urgent spine MRI was performed which revealed signs of significant cord disruption along with severe compression of the spinal cord at the level of T3/T4. PR was obvious on repeat imaging, now extending from spinal levels C1 down to T6 (Fig. 4**)**. The anterior longitudinal ligament was intact, the posterior longitudinal ligament was disrupted by a disk fragment which extruded into the spinal canal, the interspinous ligament between T3 and T4 and ligamentum flavum was also disrupted. The MRI also revealed that the facet joints were intact.

The patient was scheduled to undergo posterior spinal instrumented fusion for stabilization and remained intubated in the ICU for continued hemodynamic support. On hospital day 3, the patient further deteri-

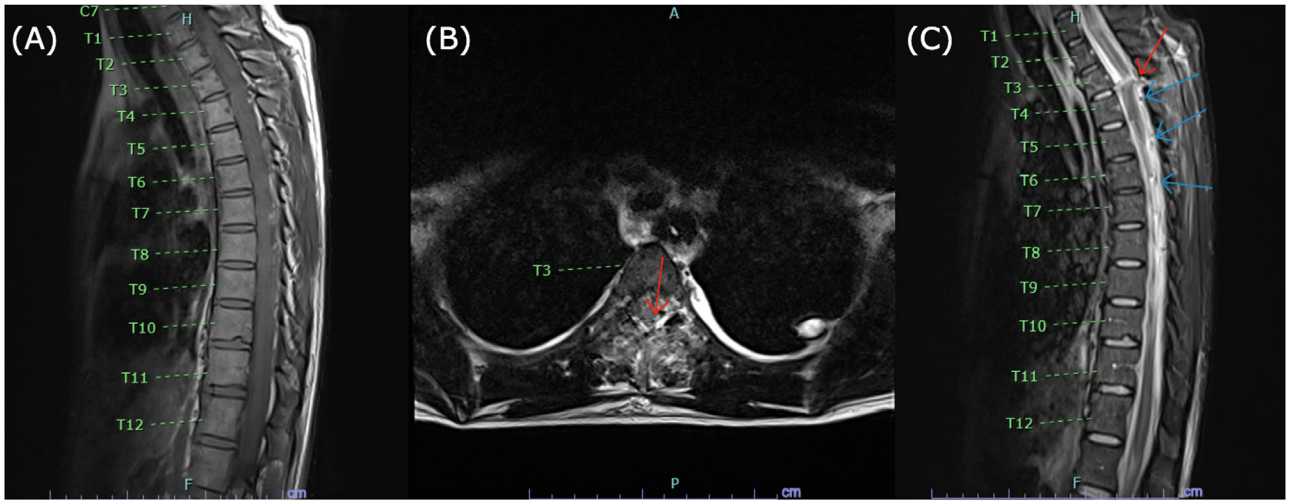

Fig. 4. Mid-sagittal T1(A) and T2(C) sequences of the thoracic spine MRI without contrast, and axial cut of T2 sequence at the T3 level (B) suggestive of acute spinal cord injury (red arrow). Pneumorrhachis is also seen extending down to spinal level T6 (blue arrows).

orated neurologically, and a repeat head CT without contrast showed extensive bilateral hemispheric infarcts with diffuse cerebral swelling. Given concerns for vascular injury, a CT angiogram of the head and neck was performed, which revealed bilateral internal carotid artery dissection resulting in extensive thrombosis and bilateral middle cerebral artery territorial infarcts with patent intracranial internal carotid arteries, presumably from collaterals. Appropriate management steps were taken by the neurosurgical, neurological, trauma, spine, stroke, and orthopedic specialty teams, including hemodynamic support, optimal sedation, MAP augmentation, placement of an intracranial pressure monitor, and seizure prophylaxis. From hospital days 4-9, changes in the neurological exam were closely monitored. She did not have a meaningful neurologic improvement. The risk of hemorrhagic transformation with acute stent-assisted reperfusion and use of anti-platelet agents was also high. Given the extensive hemispheric infarcts, acute spinal cord injury, respiratory failure despite adequate mechanical ventilation, and low probability of a meaningful functional recovery, discussions were held with the family regarding the goals of care. Her family reached the decision to withdraw intensive treatment and enter palliative care. On hospital day 10, the patient was transitioned to comfort care and ultimately passed away.

Discussion

TPC is a rare complication of thoracic trauma [10–14]. It can be caused by air migrating from the spinal canal to the intracranial compartments and, therefore, be a secondary manifestation of traumatic PR [15]. TPC in trauma can coexist with PR, in the presence or absence of underlying craniofacial fractures [8–10]. The diagnosis of SCI in polytrauma patients with TPC may be elusive when there are no evident craniofacial or other grossly unstable spinal fractures. In the presented case, the patient had acute SCI, subtle PR, and TPC as the initial finding during her secondary survey. This case demonstrates how TPC in polytrauma patients, especially in the absence of craniofacial fractures that could explain the intracranial gas source, should immediately alert the emergency physician for early spine workup due to the possibility of an etiology involving PR and acute SCI.

Although our patient had a line of fracture on T3, there were no signs of posterior cortex involvement which might explain the PR and TPC. In our case, the acute spinal cord injury was likely associated with a dural tear, conceivably due to the high amount of energy from the initial impact leading to an associated pleural violation, pneumothorax and air migration initially to the spinal canal and subsequently to the skull [12].

Allard et al. reported the only previous case of TPC with PR as identified in the CT-scan and without spinal fractures. The authors suggested the migration of air into the spinal canal was due to subcutaneous emphysema and a pleural hernia. The use of CT scans to diagnose TPC and PR in the setting of penetrating injury to the spine has also been previously reported [10]. Finally some authors have already previously suggested the use of MRI for patients with PR if the initial neurological examination is suggestive of underlying SCI [26]. In our patient, PR was mostly evident on MRI which also clearly demonstrated the previously masked thoracic spinal cord injury.

As this is an extremely rare phenomenon, there is no current diagnostic algorithm for identifying those polytrauma patients with TPC or PR which may be at high risk of SCI and, therefore, may deserve early MRI imaging [8–10]. Assessing for clinical signs of SCI may be challenging in polytrauma patients, especially in the presence of pelvic or lower extremity fractures, or overt shock. In the presented case, although initially attributed to underlying craniofacial fractures, TPC was the only early sign of SCI and a possible dural tear which, in the absence of a reliable neurological exam, could have suggested SCI. Due to the rare and unreported nature of such a finding, we believe that there is a significant value to report this event so that future healthcare providers may more promptly recognize TPC in the absence of craniofacial injuries as a potential sign of spinal cord injury.

Diagnostic algorithm

We propose a diagnostic algorithm for the early identification of spinal cord injury in TPC polytrauma patients with, or without, coexisting PR **(**Fig. 5**).** Initially, after successful resuscitation and completion of the primary trauma survey, a full body CT should be conducted as usual. We identified 3 distinct TPC trauma group categories with possible different risks for spinal cord injury, based on their initial neurologic assessment and the presence of PR. TPC patients with craniofacial fractures, but no PR, could be considered as low risk for SCI. We propose that TPC patients with PR and coexisting skull fractures with a normal neurological exam, and polytrauma patients with TPC but without PR in the absence of craniofacial fractures and an intact neurologic exam, could be considered as having an intermediate risk group of SCI. For this category, an early spine consultation should be considered by the emergency physician soon after completion of the secondary survey. We propose that all TPC patients with PR in the absence of craniofacial fractures on imaging, as well as all TPC patients with PR and underlying skull fractures with an unreliable neurologic exam should be considered high risk for SCI **(**Fig. 5**).** Our patient would have

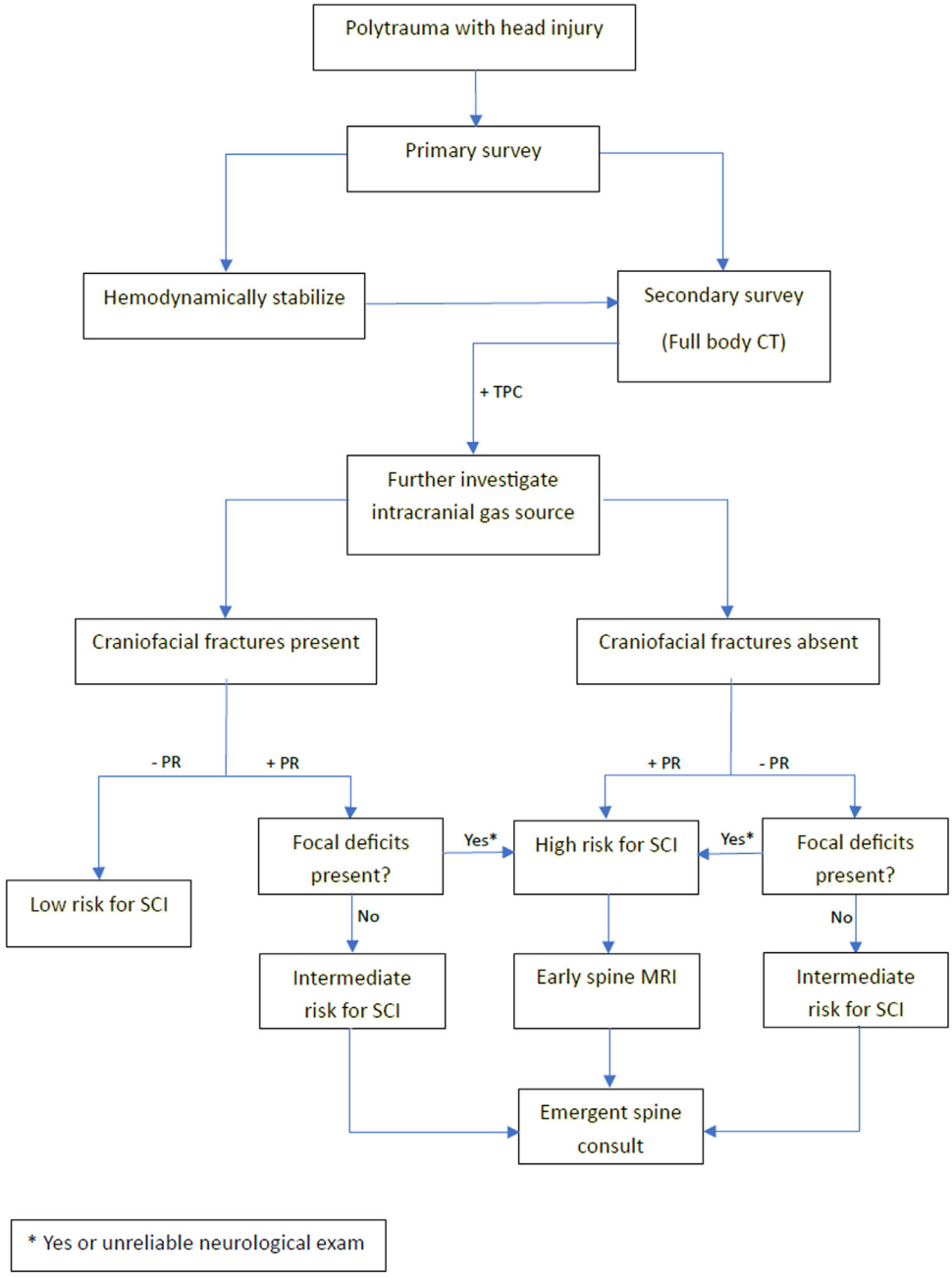

Fig. 5. A suggested algorithm for the early identification of spinal cord injury (SCI) in polytrauma presenting with pneumocephalus in the absence of coexisting spinal fractures. We identified 3 distinct groups of patients with polytrauma and traumatic pneumocephalus (TPC) with possible different underlying risk for SCI, based on their initial neurologic assessment and the presence of pneumorrhachis (PR) on imaging. After stabilizing patients during primary trauma survey, a full body CT revealing traumatic pneumocephalus would suggest further investigation to identify the intracranial source of the air. In the presence of craniofacial fractures and in the absence of PR, the intracranial gas source is most likely at the fracture site. The presence of PR and focal deficits or an unreliable neurological exam should raise a high suspicion for further investigation. TPC patients with craniofacial fractures, but no PR, should be considered as low risk for SCI (left). TPC patients with PR and coexisting skull fractures but a normal neurologic exam, as well as polytrauma patients with TPC but without PR in the absence of craniofacial fractures (right) and with an intact neurologic exam, should both be considered as the intermediate SCI risk group. For the intermediate group category, an early spine consultation should be considered. All TPC patients with PR in the absence of craniofacial fractures on imaging and all TPC patients with PR and underlying skull fractures but an unreliable neurological exam (middle) should be considered high-risk patients. In these patients urgent MRI should be considered by the trauma team or ED physician even before the spine team is contacted. Our patient was included in the high-risk TPC trauma category since she presented with PR but an unreliable exam (right); the inability to perform a focused assessment at presentation together with the absence of other spine fractures on imaging masked the diagnosis of underlying SCI. Therefore, we advocate that such type of patients with TPC without PR at the initial evaluation but also without any craniofacial injuries which may justify the TPC and with an unreliable neurological exam should also be included in the high-risk group for SCI. In these patients early MRI may play an importat role in expediting surgical treatment for the SCI. TPC, traumatic pneumocephalus; PR, pneumorrhachis; SCI, spinal cord injury.

been classified as high-risk due to the inability to perform a focused neurological assessment at presentation and the presence of TPC and PR. Lastly, the most challenging subgroup of TPC polytrauma patients, which we believe should also be included in the high-risk group, is composed of TPC patients without initial PR but an unreliable neurological exam.

By utilizing the proposed algorithm, emergency physicians and trauma surgeons can better stratify polytrauma patients with TPC regarding the underlying risk of SCI. Adoption of the proposed algorithm may lead to the early diagnosis of SCI, maintenance of spinal precautions, immediate initiation of MAP augmentation protocol, early completion of MRI and early involvement of spine surgery teams, and, in patients who can tolerate, earlier surgical intervention, all of which could increase the chances of a meaningful neurological recovery.

Conclusions

It is not uncommon for polytrauma patients to present with TPC. Spine surgeons, ED physicians and trauma surgeons should be aware that, in some cases, TPC may be the only sign of spinal cord injury, especially in the setting of a polytrauma patient without a reliable neurologic assessment. We suggest that all isolated TPC trauma patients with PR in the absence of coexisting craniofacial and spinal fractures should initially be considered as high-risk for SCI; early MRI should also be considered by emergency physicians and trauma surgeons and spine surgeons should be consulted. Ultimately the most challenging polytrauma group is probably those patients with isolated TPC without PR and an unreliable physical exam, a scenario in which an underlying SCI diagnosis can still be easily missed.

Patient informed consent statement

Complete written informed consent was obtained from the patient's family for the publication of this study and accompanying images.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Cunqueiro A, Scheinfeld MH. Causes of pneumocephalus and when to be concerned about it. Emerg Radiol 2018;25(4):331–40.
[2] Dowd GC, Molony TB, Voorhies RM. Spontaneous otogenic pneumocephalus. Case report and review of the literature. J Neurosurg 1998;89(6):1036–9.
[3] Lee IH, How CK, Chen JD, Hsu TF. Spontaneous pneumocephalus. Intern Med J 2011;41(12):842–4.
[4] Anorbe E, Aisa P, Saenz de Ormijana J. Spontaneous pneumatocele and pneumocephalus associated with mastoid hyperpneumatization. Eur J Radiol 2000;36(3):158–60.
[5] Allard E, Selim J, Veber B. Pneumocephalus and pneumorachis after blunt chest trauma without spinal fractures: a case report. J Med Case Rep 2019;13(1):317.
[6] Mohammed el R, Profant M. Spontaneous otogenic pneumocephalus. Acta Otolaryngol 2011;131(6):670–4.
[7] Yucel S, Gunbey HP, Kutlar G, Ozdemir M, Aslan K, Incesu L. Spontaneous epidural pneumocephalus. J Craniofac Surg 2016;27(8):e748–e7e9.
[8] Kara H, Akinci M, Degirmenci S, Bayir A, Ak A. Traumatic pneumorrhachis: 2 cases and review of the literature. Am J Emerg Med 2015;33(6):861 e1-e3.
[9] Koktekir E, Tatarli N, Ceylan D, Koktekir BE, Akdemir G. Symptomatic pneumorrhachis. J Neurol Surg A Cent Eur Neurosurg 2014;75(2):140–5.
[10] Osunronbi T, Sofela A, Sharma H, Muquit S. Traumatic pneumorrhachis: systematic review and an illustrative case. Neurosurg Rev 2021;44(2):731–9.
[11] Newbold RG, Wiener MD, Vogler JB 3rd, Martinez S. Traumatic pneumorrhachis. AJR Am J Roentgenol 1987;148(3):615–16.
[12] Al-Mufarrej F, Gharagozloo F, Tempesta B, Margolis M. Spontaneous cervicothoracolumbar pneumorrhachis, pneumomediastinum and pneumoperitoneum. Clin Respir J 2009;3(4):239–43.
[13] Eroglu U, Yakar F, Zaimoglu M, Ozates O, Ozgural O, Ugur HC. Pneumorrhachis. Asian J Neurosurg 2016;11(2):172–3.
[14] Pfeifle C, Henkelmann R, von der Hoh N, et al. Traumatic pneumorrhachis. Injury 2020;51(2):267–70.
[15] Gelalis ID, Karageorgos A, Arnaoutoglou C, et al. Traumatic pneumorrhachis: etiology, pathomechanism, diagnosis, and treatment. Spine J 2011;11(2):153–7.
[16] Harandou M, Khatouf M, Kanjaa N, Adnet F, Lapostolle F. [Intraspinal air, a rare complication of blunt chest trauma]. Ann Fr Anesth Reanim 2005;24(4):421–4.
[17] Katz DS, Groskin SA, Wasenko JJ. Pneumorachis and pneumocephalus caused by pneumothorax and multiple thoracic vertebral fractures. Clin Imaging 1994;18(1):85–7.
[18] Khalili H, Niakan A, Ghaffarpasand F. Massive pneumocephalus and pneumorrhachis after severe skull base fracture. Bull Emerg Trauma 2016;4(4):248–9.
[19] Chun BJ, Moon JM. Symptomatic epidural pneumorrhachis associated with an occult pneumomediastinum due to minor trauma. Spine (Phila Pa 1976) 2009;34(26):E979–82.
[20] Derner M, Drugova B, Horejsi L, Skvara D, Druga R. Massive pneumorrhachis, pneumocephalus and pneumoopticus following thoracic trauma and avulsion of the brachial plexus: case report and review of the literature. Prague Med Rep 2011;112(1):56–66.
[21] Kim SW, Seo HJ. Symptomatic epidural pneumorrhachis: a rare entity. J Korean Neurosurg Soc 2013;54(1):65–7.
[22] Mahajan PS, Al Maslamani NJ, Purayil NK. Rare case of pneumorrhachis, pneumomediastinum, pneumothorax, and surgical emphysema secondary to bronchial asthma. Int Med Case Rep J 2014;7:35–9.
[23] Niemann MJ. [Valsalva-induced subcutaneous emphysema, pneumomediastinum and pneumorrhachis in a young man]. Ugeskr Laeger 2019;181(9):V10180693 Danish.
[24] Pangtey GS, Das CJ, Javan N. Airlessness in airspace. Simultaneous occurrence of spontaneous pneumothorax with pneumomediastinum and pneumorrhachis: report of a case. Surg Today 2008;38(1):49–51.
[25] Kasimatis GB, Panagiotopoulos E, Megas P, et al. The adult spinal cord injury without radiographic abnormalities syndrome: magnetic resonance imaging and clinical findings in adults with spinal cord injuries having normal radiographs and computed tomography studies. J Trauma 2008;65(1):86–93.
[26] Oertel MF, Korinth MC, Reinges MH, Krings T, Terbeck S, Gilsbach JM. Pathogenesis, diagnosis and management of pneumorrhachis. Eur Spine J 2006;15(Suppl 5):636–43.