Traumatic tension pneumocephalus: Two cases and comprehensive review of literature

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ABSTRACT

Introduction. Although traumatic pneumocephalus is not uncommon, it rarely evolves into tension pneumocephalus (TP). Characterized by the presence of increasing amounts of intracranial air and concurrent appearance or worsening neurological symptoms, TP can be devastating if not recognized and treated promptly. We present two cases of traumatic TP and a concise review of literature on this topic.

Methods. Two cases of traumatic TP are presented. In addition, a literature search revealed 20 additional cases, of which 18 had sufficient information for inclusion. Literature cases were combined with the 2 reported cases and analyzed for demographics, mechanism of injury, symptoms, time to presentation (acute <72 hours; delayed ≥72 hours), diagnostic/treatment modalities, and outcomes.

Case reports and literature review. Twenty cases were analyzed (17 males, 3 females, median age 26, range 8-92 years). Presentation was acute in 13/20 and delayed in 7/20 patients. Injury mechanisms included motor vehicle collisions (6/20), assault/blunt trauma to the cranio-facial area (5), falls (4), and motorcycle/bicycle crashes (3). Common presentations included depressed mental status (10/20), CSF rhinorrhea (9), headache (8), and loss of consciousness (6). Computed tomography (CT) was utilized in 19/20 patients. Common underlying injuries were frontal bone/sinus fracture (11/20), emergency burr hole (4), endoscopy (2), and ventriculostomy (2). Most patients responded to initial treatment (19/20). One early and one delayed death were reported.

Conclusions. Traumatic TP is rare, tends to be associated with severe cranio-facial injuries, and can occur following both blunt and penetrating injury. Early recognition and high index of clinical suspicion are important. Appropriate treatment results in improvement in vast majority of cases. CT scan is the diagnostic modality of choice for TP.

INTRODUCTION

Traumatic pneumocephalus – or abnormal presence of air in the cranial cavity following traumatic injury – occurs frequently. Its pathophysiology involves air entry into the cranial cavity following injury to the brain meninges. Tension pneumocephalus (TP) is a clinical entity characterized by continued build-up of air within the cranial cavity, leading to abnormal pressure exerted upon the brain and subsequent neurologic deterioration. The accumulation of intracranial air can be acute or delayed. Knowledge of risk factors, radiographic findings, and clinical signs/symptoms associated with TP is crucial to its prompt identification and treatment. Clinical diagnostic and treatment delays may result in poor neurologic outcome and mortality.

METHODS

Two cases of traumatic tension pneumocephalus are presented. In addition, a detailed literature search (PubMed, Google Scholar, ScientificCommons) revealed a total of 20 other traumatic pneumocephalus cases, of which 18 contained complete data that were sufficient for subsequent collection/analysis of the following variables: (a) patient age; (b) patient gender; (c) mechanism of injury; (d) primary injury associated with TP; (e) associated injuries; (f) signs and symptoms of TP; (g) therapeutic approach to TP; and (h) timing.
of clinical onset of TP. Cases reported in the literature were then combined with the 2 cases reported by our group and analyzed for demographics, mechanism of injury, symptoms, time to presentation (acute or presenting within 72 hours of injury versus delayed or presenting after the initial 72 hours post-injury), diagnostic and treatment modalities used, and outcomes.

**CASE REPORT #1**

An 8-year-old male was admitted to the hospital after being struck by a motor vehicle. During the initial trauma evaluation, he was found to have slight facial deformities and abdominal tenderness. Computed tomography (CT) demonstrated the presence of multiple skull fractures, including right mastoid fracture as well as pneumocephalus (Figure 1A-D). His other injuries included a grade II splenic laceration with no evidence of intravenous contrast extravasation. He was admitted to the surgical intensive care unit (SICU) for observation. The following morning, the patient was found to have dilated and minimally responsive pupils bilaterally. He also experienced several episodes of bradycardia (lowest recorded heart rate of 28 beats/minute). He had no other focal neurologic findings (his Glasgow Coma Score [GCS] remained between 14 and 15), with the only major subjective complaint being significant headache. The patient underwent an urgent repeat CT of the brain, which demonstrated a marked increase in the volume of pneumocephalus, no intra- or extra-axial hemorrhage, and stable multiple skull fractures (Figure 1E-F). The patient, who was initially maintained with 30-degree head elevation, was now repositioned in flat configuration and high-flow oxygen was administered via face mask. His bradycardic episodes resolved after this positional change and his pupillary dilation resolved over the period of approximately 18 hours. Therapy directed at TP was continued for three days, after which his activity was liberalized without any recurrent symptoms. The patient was doing well on three-month follow-up [Glasgow Outcome Score (GOS) of 5, GCS of 15, no focal neurological deficits].

**Figure 1.** Computed tomographic (CT) imaging for Case #1: (A-B) Admission CT shows small foci of pneumocephalus; (C-D) Admission images showing associated skull fractures, including right mastoid process fracture; (E-F) Repeat CT imaging shows the appearance of the “Mount Fuji” sign.

**CASE REPORT #2**

A twenty-year-old man was brought to the trauma evaluation area following a high-speed motor vehicle collision. He sustained massive injuries to his midface after striking the steering wheel. His initial GCS was 14 (Motor 6, Verbal 5, Eyes 3) upon hospital arrival. The initial CT of the head and face demonstrated a small to moderate pneumocephalus, brain contusions, as well as multiple facial fractures (Figure 2A). The patient was initially monitored in the SICU. The patient was placed on 100% oxygen therapy. After approximately 24 hours, his GCS suddenly declined to 10 (Motor 5, Verbal 4, Eyes 1). He was intubated, and a repeat CT demonstrated increasing pneumocephalus (Figure 2B), with slight midline shift and the classic appearance of the “Mount Fuji” sign (Figure 2C). No other obvious causes for such rapid deterioration (i.e., opioid administration, rapidly expanding intracranial hematoma, or other traumatic injuries) were noted. The patient was taken to the operating room for repair of a dural tear as well as fixation of multiple facial fractures. Postoperative CT scan demonstrated resolution of the preoperative pathologic findings (Figure 2D). The patient gradually recovered over a period of 3 weeks and was discharged to a rehabilitation facility with a GCS of 13 and GOS of 3. On three-month follow up, the patient was ambulatory, with GCS of 15, GOS of 4, and continued neurologic improvement.

**Figure 2.** Computed tomographic (CT) imaging for Case #2: (A) Initial CT – note the presence of small amounts of intracranial air; (B and C) Repeat CT of the brain demonstrating increasing pneumocephalus, with slight midline shift (C) and “Mount Fuji” sign; and (D) Postoperative CT showing the resolution of tension pneumocephalus.

**REVIEW OF LITERATURE**

A total of 20 tension pneumocephalus reports were identified. Of those, 18 were suitable for inclusion and further comparisons. Cases outlined by the authors were then added to those identified from the literature search, for a total of 20 patients. There were 17 males and 3 females (median age 26, range 8-92 years). Presentation was acute in 13/20 and delayed in 7/20 patients. Injury mechanisms included motor vehicle collisions (6/20),
Tension pneumocephalus (i.e., intracranial air causing mass effect on the brain) requires conditions that lead to increased intracranial pressure within the intracranial space, and represents further increases in intracranial pressure that are assumed to be due to the mechanisms described above.19, 20 The pressure exerted by the intracranial air upon the brain may lead to extra-axial mass effect with subsequent compression of the frontal lobes (and thus the “Mount Fuji” appearance on CT, Figures 1E-F and 2B-C). Clinical presentation of TP may include agitation, delirium, otherwise altered level of consciousness, pupillary changes, and frontal lobe syndrome. At times, hemodynamic changes may be present, including episodes of bradycardia with or without hypertension (see Case Report #1).21

Trauma is the predominant etiologic factor associated with pneumocephalus, accounting for 67-74% of all pneumocephalus cases in large series.12, 22 While there are no reports large enough to characterize the incidence of presenting symptoms in patients with TP, certain generalizations may be made based on pneumocephalus literature alone. We have noted that headache was the most common, but not universally present, presenting symptom of TP (seen in 44% of cases). This is consistent with previously published data, wherein headache was also found to be the most common, but not predominant, symptom (38% of cases) of pneumocephalus.12 A comparable degree of similarity can also be seen between the current review and previously reported data in terms of the incidence of CSF rhinorrhea (41% versus 31%).12 Of note, the only symptom and physical finding that is pathognomonic of pneumocephalus is the bruit hydro-aérique (a.k.a. “suctioning splash”), defined as the presence of a splashing sound heard only by the patient upon postural change.9,10 However, bruit hydro-aérique occurs in only about 7% of pneumocephalus cases.12 Mental status changes were associated with 44% of TP cases in the current report, with 28% experiencing loss of consciousness at some point during their clinical course.

In terms of clinical management, most cases of pneumocephalus tend to resolve spontaneously with conservative management. Nonoperative management involves oxygen therapy, maintaining the patient supine or in Trendelenburg position, prophylactic antimicrobial therapy (especially in post-traumatic cases), adequate analgesia, frequent neurologic checks, and repeated CT scans. The use of continuous high concentration inspired oxygen as a treatment modality for traumatic pneumocephalus may have certain theoretical benefits (i.e., quicker absorption of the trapped cavitary air via the nitrogen washout effect), although the effectiveness of this approach is yet to be clinically proven in the setting of traumatic pneumocephalus.12-23 Prompt decompression of intracranial air is the initial treatment of symptomatic pneumocephalus.26 The principles of subsequent treatment
parallel those for a cerebrospinal fluid leak. It is important to identify the site where the communication between the air cavity and the external environment occurs. If the site can be identified, the passage should be sealed off, thereby decreasing the possibility of worsening or recurrent pneumocephalus. Effective therapy of TP via a controlled decompression using a closed water-seal drainage system has also been described. 27, 28

Tension pneumocephalus is a neurological emergency, and as such, its early identification is crucial. Diagnostic, Ishiwata et al described the appearance of the "Mount Fuji" sign in a series of patients with tension pneumocephalus. 29 The presence of such "Mount Fuji" sign (Figures 1F & 2B-C) on head CT in trauma patients should be considered a critical finding, and its presence should prompt immediate patient evaluation and appropriate re-appraisal of the therapeutic plan. 29 To diagnose TP, the CT findings should correlate with clinical signs of neurologic deterioration. In the early 1980s, an attempt was made to explain the mass effect of pneumocephalus based on the volume of gas. It has been proposed that the volume of air as little 65 mL is sufficient enough to produce tension pneumocephalus. 28 Subsequent to this finding, however, other authors found no substantial difference between the volume of air and the occurrence of tension pneumocephalus. 29 After both clinical and imaging findings are appropriately recognized and correlated, definitive treatment is initiated. Reported treatment options for tension pneumocephalus include a combination of: (a) drilling of burr holes; (b) craniotomy; (c) needle aspiration; (d) ventriculostomy placement; (e) Administration of 100% oxygen; and (f) closure of dural defect(s). 29 Careful monitoring for clinical deterioration, as well as serial CT scanning of the brain, is recommended.

CONCLUSIONS
Post-traumatic tension pneumocephalus is rare, tends to be associated with severe cranial base and facial injuries, and can be present following both blunt and penetrating mechanisms of injury. Early recognition and high index of clinical suspicion are important and prompt treatment results in improvement in vast majority of cases. Computed tomography is the gold standard for diagnosis of this condition.

REFERENCES


<table>
<thead>
<tr>
<th>Date (Reference)</th>
<th>Age</th>
<th>Gender</th>
<th>Mechanism of Injury</th>
<th>Nature of Brain Injury and Associated Injuries</th>
<th>Symptoms and Signs</th>
<th>Therapeutic Approach</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>1992 / 31</td>
<td>18</td>
<td>M</td>
<td>MVC</td>
<td>Right frontal intracerebral hematoma; Frontal bone fractures</td>
<td>Headache, nausea, vomiting x1 week prior to admission</td>
<td>Emergency craniotomy; TP decompression; Pericranial muscle coverage of CSF fistula; Duraplasty</td>
<td>Presentation after 3 months of delay; Patient was discharged without complications</td>
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<td>1998 / 32</td>
<td>45</td>
<td>M</td>
<td>Gunshot wound to left frontal sinus</td>
<td>Bullet fragment in the left frontal sinus; Dural tear and necrotic brain tissue present</td>
<td>Presentation GCS of 13; CSF rhinorhea</td>
<td>Emergent surgery; Dural repair and debridement of necrotic brain tissue; Removal of the mucosa membrane of the left frontal sinus</td>
<td>Tense fascia lata grafting to the sinus was performed</td>
</tr>
<tr>
<td>1998 / 33</td>
<td>17</td>
<td>M</td>
<td>MVC</td>
<td>Hemorrhagic brain contusions; Frontal and ethmoid sinus fistula leading to TP</td>
<td>Headache, rhinorhea, vomiting after anesthetizing</td>
<td>Operative repair</td>
<td>Presentation one month after acute traumatic injury; Dural involvement</td>
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<td>1999 / 3</td>
<td>39</td>
<td>M</td>
<td>Fall down stairs</td>
<td>Right orbital roof fracture communicating with frontal sinus; Right periorbital contusion</td>
<td>Loss of consciousness; Right eye pain; Headaches; Dizziness; CSF rhinorhea</td>
<td>Conservative (non-operative) therapy with antibiotics, analgesics, and serial neurologic exams</td>
<td>Presented with conservative therapy only</td>
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<td>1999 / 4</td>
<td>25</td>
<td>M</td>
<td>Remote history of blunt traumatic injury</td>
<td>Traumatic brain injury with residual left hemiparesis and behavioral changes; Patient required ventriculo-peritoneal shunt placement for communicating hydrocephalus</td>
<td>Severe headaches; Post-traumatic epilepsy</td>
<td>Bifrontal craniotomy; Exenteration of right ethmoid sinus; Repair of dural defect with fascia lata graft</td>
<td>Three-year delay before clinical presentation; Fascia lata grafting to the sinus; Patient experienced immediate relief of headaches; Recovery was uneventful</td>
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<tr>
<td>2001 / 34</td>
<td>34</td>
<td>M</td>
<td>MVC</td>
<td>Bifrontal brain contusions; Depressed open skull fracture; Comminuted frontal sinus fracture; Nasal-orbital-ethmoid fractures; Comminuted superior orbital roof and nasal bone fractures</td>
<td>Persistent rhinorhea; Progressive lethargy</td>
<td>Open reduction/internal fixation of depressed skull fractures; Cranialization of frontal sinuses; Obliteration of naso-frontal ducts; Ventriculostomy</td>
<td>Muscle and adipose tissue graft utilized; Endoscopic technique described; At 2-year follow-up, no evidence of CSF leak</td>
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<td>2001 / 2</td>
<td>13</td>
<td>F</td>
<td>Stab wound to the neck</td>
<td>Cerebrospinal fluid leak</td>
<td>Headaches</td>
<td>Surgical repair</td>
<td>Immediate onset of symptoms</td>
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<td>2002 / 7</td>
<td>27</td>
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<td>MVC</td>
<td>Traumatic brain injury; Left supraorbital laceration</td>
<td>Loss of consciousness for 10 days; Visual loss; 6th cranial nerve palsy; CSF rhinorhea</td>
<td>Bifrontal craniotomy; Release of subdural TP</td>
<td>Presentation delayed by approximately 2 months; Patient fully regained vision</td>
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<td>2003 / 5</td>
<td>12</td>
<td>M</td>
<td>MCC</td>
<td>Frontal fracture with associated frontal pneumocephalus</td>
<td>Loss of consciousness; Convulsions; Amnesia</td>
<td>Right frontal burr hole placement</td>
<td>Onset of symptoms and presentation delayed by 2 months; Air resolved after 10 days</td>
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<tr>
<td>2004 / 35</td>
<td>8</td>
<td>F</td>
<td>Fall from bicycle</td>
<td>Closed head injury with temporal bone fracture; Large pre-pontine air displacing the brain stem posteriorly</td>
<td>Loss of consciousness; Initial GCS of 8; CSF rhinorhea and right hemotympanum</td>
<td>Conservative (non-operative) management with mechanical ventilation on general supportive measures</td>
<td>The patient developed meningitis which was treated with systemic antibiotics and lumbar CSF drain placement; Partial hearing loss was noted in the right ear</td>
</tr>
<tr>
<td>2005 / 15</td>
<td>38</td>
<td>F</td>
<td>Remote history of blunt cranial trauma</td>
<td>History of cranial fracture following an accident 12 years prior to current presentation</td>
<td>History of nasal discharges and intermittent generalized seizures</td>
<td>Operative reconstruction of both dural and bony defects; Repair of the dural defect; Fascia lata grafting to the left frontal sinus</td>
<td>Delayed presentation (12 years after initial traumatic injury); Patient improved and recovered uneventfully</td>
</tr>
<tr>
<td>2005 / 36</td>
<td>40</td>
<td>M</td>
<td>MVC</td>
<td>Traumatic brain injury with diffuse intracranial air, including the right frontal brain parenchyma, lateral ventricles, and the subarachnoid spaces and cisterns</td>
<td>Initial GCS of 12; Hydrocephalus</td>
<td>Treatment with 100% FIO2 and head elevation; Ventriculo-peritoneal shunt placement</td>
<td>Clinical presentation was delayed until post-injury day #4; Patient discharged on hospital day 20 in stable condition</td>
</tr>
<tr>
<td>2005 / 8</td>
<td>30</td>
<td>M</td>
<td>Assault</td>
<td>Multiple facial and skull base fractures</td>
<td>Agitation; Mental status changes; Initial GCS of 14</td>
<td>Repair of the dural defect; Repair of facial fractures</td>
<td>Immediate post-injury presentation</td>
</tr>
<tr>
<td>2005 / 1</td>
<td>22</td>
<td>M</td>
<td>Stab with golf club</td>
<td>Forehead laceration; Frontal sinus fracture; Comminuted nasal fracture</td>
<td>Bloody rhinorhea; CSF leak</td>
<td>Bifrontal craniotomy with cranioplasty of frontal sinuses; Galeal flap and temporal flap</td>
<td>Immediate post-injury presentation; Delayed CSF rhinorhea noted</td>
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<tr>
<td>2006 / 37</td>
<td>34</td>
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<td>Basal skull fractures</td>
<td>Headaches; Vomiting; CSF rhinorhea; Mental status changes</td>
<td>Emergency burr hole placement with drainage; After failure of the initial therapy (the TP actually increased in size) the patient underwent endoscopic surgical repair of a dural tear</td>
<td>Clinical presentation at 48 hours after initial injury; Patient recovered fully</td>
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<td>2007 / 38</td>
<td>92</td>
<td>M</td>
<td>Traumatic fall</td>
<td>No evidence of cranial fracture</td>
<td>Mental status changes</td>
<td>Emergency burr hole placement with drainage; After failure of the initial therapy (the TP actually increased in size) the patient underwent endoscopic surgical repair of a dural tear</td>
<td>Immediate post-injury presentation; This report describes novel use of an endoscopic technique to repair dural defects; The patient died two weeks after surgery from multi-organ dysfunction</td>
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<tr>
<td>2007 / 9</td>
<td>35</td>
<td>M</td>
<td>Blunt head injury</td>
<td>Right subdural hematoma; Diffuse subarachnoid hemorrhage</td>
<td>Declining mental status; Facial swelling</td>
<td>Placement of intracranial pressure (ICP) monitor with concurrent evacuation of small amount of subdural blood</td>
<td>Traumatic pneumocephalus was causally linked to ICP monitor placement; The patient died approximately one week after initial injury</td>
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<td>2009 / 39</td>
<td>22</td>
<td>M</td>
<td>MVC</td>
<td>Fractures of anterior cranial fossa; Convex right fronto-temporal epidural hematoma</td>
<td>Loss of consciousness; Epistaxis; Headaches</td>
<td>Conservative (non-operative) management</td>
<td>Immediate presentation; Patient recovered fully</td>
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