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Clinical and Radiological Characteristics of Traumatic Pneumocephalus after Traumatic Brain Injury

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Conflict of Interest
The authors have no financial conflicts of interest.

ABSTRACT

Objective: Traumatic pneumocephalus (TP) is a common complication of traumatic brain injury (TBI), which is characterized by the abnormal entrapment of air in the intracranial cavity after TBI to the meninges. The purpose of this study was to investigate the clinical and radiological characteristics related to TP associated with TBI.
Methods: From January 2013 to March 2018, the data from 71 patients with TP after TBI were collected. Demographic and clinical characteristics were investigated and the distribution of TP was investigated as radiological characteristics. The author compared the demographic characteristics of TP to the data from the Korean Neurotrauma Data Bank System (KNTDBS).
Results: There was a higher ratio of males in patients with TP compared with KNTDBS. The mean age was 48.4±20.5 years and the incidence was highest in those 41–60 years of age (42.3%). Surgical treatment was performed in 23.9% patients. The mortality associated with the TP was 4.2%. The most common cause of injury was a traffic accident (52.1%). TP was mainly located in the epidural space (53.5%) and showed a scatter pattern (60.6%). It was mostly located in the frontal and temporal area (81.7%). Skull fractures were observed in 40.8% in the temporal bone and 25.4% in the frontal bone.
Conclusion: The author identified epidemiology, clinical, and radiological characteristics of TP associated with TBI. Although this study has many limitations, the author believes it is worthwhile as it examines various characteristics of TP, which previously had relatively little clinical interest.

Keywords: Traumatic pneumocephalus; Traumatic brain injury; Epidemiology

INTRODUCTION

Intracranial air is known as an “aerocele” or “pneumocephalus” and it was initially described at autopsy by Chiari in 1884.[1] Luckett[10] first used the term “pneumocephalus” in 1913 when he reported the case of a man whose skull radiography showed air in the ventricles after a traffic accident (TA). The incidence of pneumocephalus depends on the etiology and can be seen in almost all cases of craniotomy. Pneumocephalus is mostly caused by trauma and the incidence of pneumocephalus after traumatic brain injuries (TBIs) varies from 1% to 82% depending on the series.[7,18]

Traumatic pneumocephalus (TP) is a common complication caused by TBI, which is characterized by the abnormal entrapment of air in the intracranial cavity after TBI to the meninges.[9] Pneumocephalus can be sub-divided by location.[13] Extra-axial locations include the epidural, subdural, and subarachnoid space, and intra-axial locations include parenchymal, intraventricular, and intravascular space. TP also present as a single lesion or multiple air bubbles scattered in several cisterns. In addition, it alone may occur after TBI and may be accompanied by intracranial lesions such as acute traumatic subarachnoid hemorrhage (TSAH), acute subdural hematoma (ASDH), acute epidural hematoma (AEDH), traumatic intracerebral hemorrhage (TICH), and traumatic intraventricular hemorrhage (TIVH). Conservative treatment alone is often sufficient for TP that occurs alone. Furthermore, the clinical significance of TP is often less than that of the accompanying traumatic intracranial lesions. For these reasons, TP has been often overlooked compared to other traumatic intracranial lesions, so there is not many clinical studies which consider TP. The purpose of this study was to investigate the clinical and radiological characteristics of TP after TBI.

MATERIALS AND METHODS

The study was approved by the Institute Ethical Committee of Wonkwang University Hospital (WKUH) and in compliance with institute's requirements. (WKUH 201908022). From January 2013 to March 2018, the author collected data from 71 patients with TP after TBI. Data were retrospectively reviewed from the medical and radiological records. All patients with TP confirmed by cranial computed tomography (CT) scan were included in the study. Exclusion criteria were pneumocephalus due to other non-traumatic reasons such as intracranial gas-forming bacterial infection, post-operative pneumocephalus, and fracture compound comminuted depressed (FCCD). In this study, the reason for the exclusion of pneumocephalus due to FCCD is that clinical and radiological findings have no significance because FCCD-induced pneumocephalus naturally occurs just below the head trauma site due to the compound depression of the skull. The author compared demographic characteristics of TP and data from the Korean Neurotrauma Data Bank System (KNTDBS) and statistically analyzed the characteristics. The Korean Society of Neurotraumatology recorded data from 2,617 patients with TBI from 20 institutions between September 2010 and March 2014 and provided various demographic data related to TBI in Korea though an established KNTDBS website.[17] The author investigated the gender, age distribution, Glasgow coma scale (GCS), surgical treatment, and survival before comparing this study with various data from KNTDBS after obtaining permission from KNTDBS to analyze demographic characteristics. Other demographic and clinical characteristics of patients with TP, including cause of injury, leakage of cerebrospinal fluid (CSF), clinical manifestation (review of system) except headache, presence of scalp laceration, Glasgow outcome scale (GOS), and hospital stay, were investigated. The leakage of CSF was confirmed through otolaryngology consultation when patients complained of clinical symptom that the leak of clear and watery drainage from the nose and ear with a positional dependency. The outcome of TP was identified using the GOS at 6 months after TBI. Distribution of TP was investigated as radiological characteristics and the distribution was analyzed according to the location (epidural, subdural, intracerebral parenchyma, intraventricular, sella) (**FIGURE 1A-E**), side (left, right, bilateral, and midline), depth (superficial and deep) (**FIGURE 1F & G**), and brain area (frontal, temporal, parietal, occipital, and sella) of TP. Sites of skull fracture and intracranial traumatic lesions (none, TSAH, AEDH, ASDH, TICH, and TIVH) associated with TP on CT were also investigated (**FIGURE 2**).

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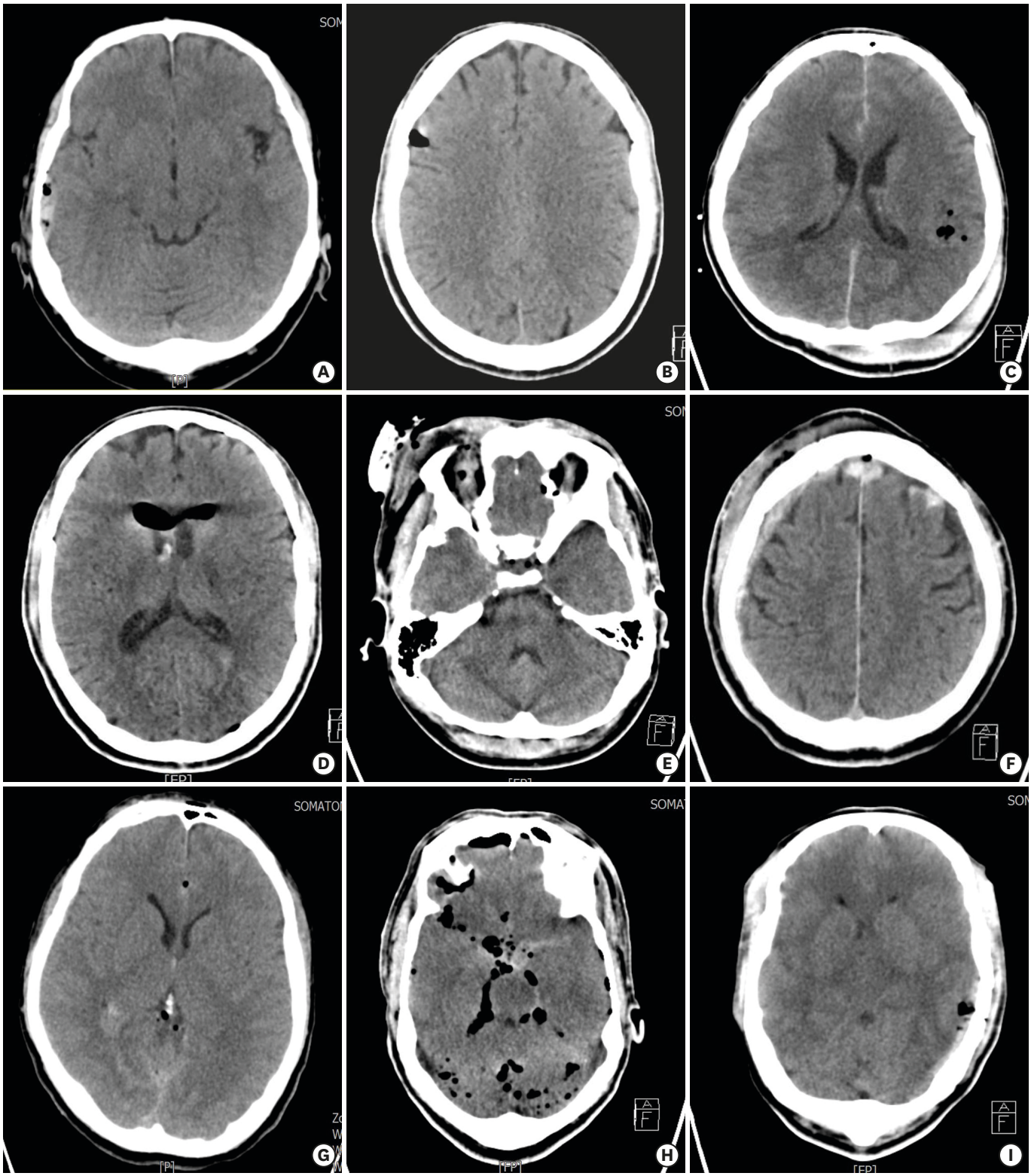

FIGURE 1. Distribution of traumatic pneumocephalus according to location and depth. (A) Epidural, (B) subdural, (C) intracerebral parenchyma, (D) intraventricular, (E) sella area, (F) superficial, and (G) deep location. (H) Scatter and (I) concentrated pattern.

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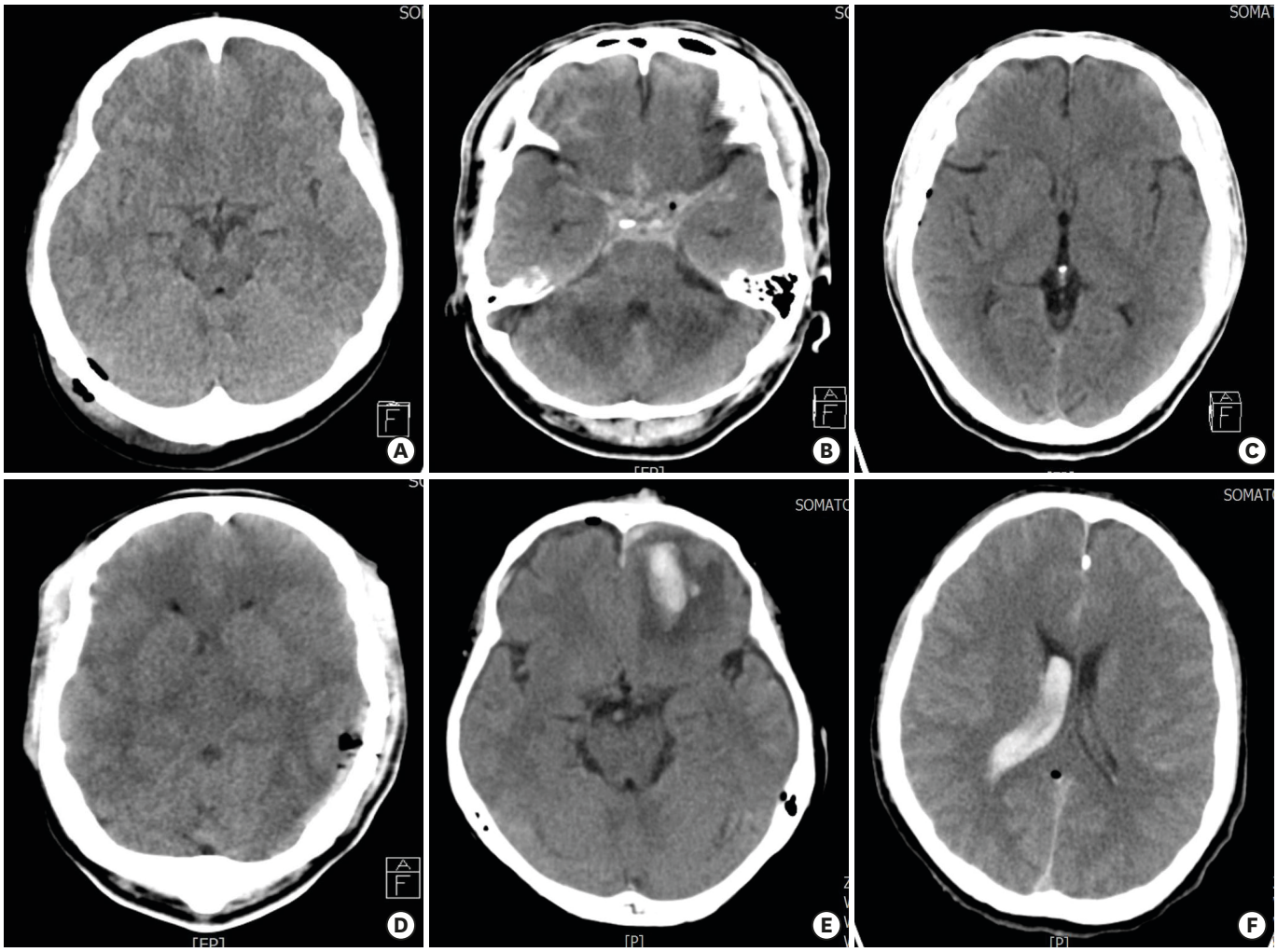

FIGURE 2. Intracranial traumatic lesions associated with traumatic pneumocephalus. (A) None, (B) traumatic subarachnoid hemorrhage, (C) acute epidural hematoma, (D) acute subdural hematoma, (E) traumatic intracerebral hemorrhage, and (F) traumatic intraventricular hemorrhage.

Statistical analyses

SPSS version 22.0 (IBM SPSS Inc., Armonk, NY, USA) was used for statistical analyses. The Mann-Whitney *U* test was used for comparing non-categorical data. Differences were considered statistically significant if *p*-values were <0.05.

RESULTS

Demographic characteristics

A total of 71 TBI patients with pneumocephalus were included in this study. The demographic characteristics of patients are summarized in **TABLES 1** & **2**. In order to determine the difference between TP and general TBI patients in relation to demographic characteristics, the data of the KNTDB which could be compared in this study were selected and analyzed. This is presented in **TABLE 1**. In this study, the proportion of male patients was 83.1%, and this proportion was higher in patients with TP compared with 70.8% of KNTDBS and this difference was statistically significant (p=0.025). The mean patient age was 48.4±20.5 years.

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TABLE 1. Demographic characteristics of patients with traumatic pneumocephalus comparable to KNTDBS

Characteristics	This study	KNTDBS	*p*-value
Sex			0.025*
Male	59 (83.1)	1,854 (70.8)	
Female	12 (16.9)	763 (29.2)	
Age (yr)			0.073
≤20	9 (12.7)	292 (11.2)	
21–40	11 (15.5)	422 (16.1)	
41–60	30 (42.3)	834 (31.9)	
≥61	21 (29.6)	1,069 (40.8)	
GCS			0.643
3–8	12 (16.9)	402 (15.3)	
9–12	8 (11.3)	332 (12.7)	
13–15	51 (71.8)	1,883 (72.0)	
Treatment			0.763
Surgical	17 (23.9)	668 (25.5)	
Nonsurgical	54 (76.1)	1,929 (73.7)	
Outcome			0.830
Survival	68 (95.8)	2,492 (95.2)	
Death	3 (4.2)	125 (4.8)	

Values are presented as number (%). Mann-Whitney *U* test.
KNTDBS: Korean Neurotrauma Data Bank System, GCS: Glasgow coma scale.
*The $p<0.05$ indicates statistical significance.

TABLE 2. Other demographic and clinical characteristics of patients with traumatic pneumocephalus

Characteristics	No. (%)
Cause of injury	
Slip	14 (19.7)
Fall	13 (18.3)
Blunt injury	7 (9.9)
TA	37 (52.1)
Passenger car TA	22
Pedestrian car TA	6
Motorcycle TA	3
Bicycle TA	6
Leakage of cerebrospinal fluid	7 (9.6)
Otorrhea	2
Rhinorrhea	5
Review of system	47 (66.2)
Nausea	16
Vomiting	2
Dizziness	24
Tinnitus	5
Scalp laceration	19 (26.8)
GOS	4.3±1.1
Death	3 (4.2)
2	5 (7.0)
3	5 (7.0)
4	10 (14.1)
5	48 (67.6)

TA: traffic accident, GOS: Glasgow outcome scale.

The incidence of TBI according to age was highest in those 41–60 years of age (42.3%; n=30), followed by 21 patients (29.6%) aged ≥61 years, 11 patients (15.5%) aged 21–40 years, and 9 patients (12.7%) aged ≤20 years. In contrast, the age distribution of KNTDBS was the highest in the group aged ≥61 years (40.8%), followed by 41–60 years (31.9%), 21–40 years (16.1%), and ≤ 20 years (11.2%). However, there was no significant difference in this study (p=0.073). Patients were divided into 3 groups based on their GCS score: mild (GCS score 13–15 points), moderate (GCS score 9–12 points), and severe (GCS score below 8). The GCS score of TP vs.

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KNTDBS groups were not statistically significant (p=0.643), with similar patterns in the mild group (TP vs. KNTDBS: 16.9% vs. 15.3%), moderate group (11.3% vs. 12.7%), and severe group (71.8% vs. 72.0%). A total of 54 patients (76.1%) received non-surgical treatment, all of which were surgery for intracranial lesions associated with TP, not direct surgery for TP. Seventeen patients (23.9%) received surgical treatment; this difference with KNTDBS was not statistically significant (p=0.763). Among the 71 patients, 3 patients (4.2%) died and 68 patients (95.8%) survived and this mortality rate was similar to that of the KNTDBS (4.8%) (p=0.830). Other demographic and clinical characteristics of patients with TP are summarized in **TABLE 2**. The causes of injury were 14 slips (19.7%), 13 fall down (18.3%), 7 blunt injury (9.9%), and 37 TA (52.1%). The cases of TA comprised of 22 passengers TA, 6 pedestrians TA, 3 motorcycle TA, and 6 bicycle TA. Leakage of CSF was found in 6 patients (8.5%), including 2 patients with otorrhea and 5 patients with rhinorrhea. Clinical manifestation (upon systemic examination) except headache was associated with 47 patients (66.2%), including 16 patients with nausea, 2 patients with vomiting, 24 patients with dizziness and 5 patients with tinnitus. Scalp laceration was present in 19 patients (26.8%). The mean GOS score was 4.3±1.1. Three patients (4.2%) had a GOS score of 1, 5 patients (7.0%) had a score of 2, 5 patients (7.0%) had a score of 3, 10 patients (14.1%) had a score of 4, and 48 patients (67.6%) had a score of 5. Mean hospital stay was 20.8±12.3 days.

TABLE 3 shows that the distribution of TP. The most common location of TP was the epidural space (53.5%; n=38), followed by the subdural space (22.5%; n=16), intracerebral parenchyma (5.6%; n=4), sella (5.6%; n=4), followed by the epidural and subdural space (4.2%; n=3). TP mostly occurred unilaterally. The most common side was the right (40.8%; n=29), followed by the left (32.3%; n=23), bilateral (15.5%; n=11), and midline (5.6%; n=4). TP showed a scatter pattern in 60.6% (n=43) and a concentrated pattern in 39.4% (n=28) of cases. The most common depth of TP was superficial (63.4%; n=45), followed by deep (26.8%; n=19), followed by superficial and deep (9.9%; n=7). The brain area TP was most commonly located in was the temporal area (29.6%; n=21) and in the frontal area (23.9%; n=17). The frontal and temporal area were involved in 81.7% (n=58) and sella area were involved in 11.2% (n=8) of TP.

TABLE 4 shows the site of skull fracture associated with TP. The most common site of skull fracture was the temporal bone (32.4%; n=23), followed by the frontal bone (18.3%; n=13), skull base (12.7%; n=9), skull base with other bones (12.7%; n=9), and none (7.0%; n=5). The temporal bone was involved in 40.8% (n=29) and the frontal bone was involved in 25.4% (n=18) of cases. Sella was involved in 12.7% (n=9).

TABLE 5 shows the intracranial traumatic lesions associated TP. The most common intracranial traumatic lesions were ASDH (19.7%; n=14) and none (pneumocephalus alone) (19.7%; n=14), followed by AEDH (18.3%; n=13), TSAH (15.5%; n=11), ASDH with TSAH (7.0%; n=5), ASDH with TICH (5.6%; n=4), AEDH with TSAH (4.2%; n=3), AEDH with ASDH (4.2%; n=3), TICH (2.8%; n=2), and TIVH (1.4%; n=1) and AEDH with TICH (1.4%; n=1).

DISCUSSION

The gold standard diagnostic tool for TP is CT. CT only needs 0.55 mL of air to detect pneumocephalus, while skull X-rays need at least 2 mL of air.[15] The earlier the patient with TBI is examined by CT, the greater is the likelihood is of detecting a TP. This is consistent with the well-known fact that air trapped the intracranial cavity can be absorbed within

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TABLE 3. Distribution of traumatic pneumocephalus

Distribution	No. (%)
Location	
Epidural space	38 (53.5)
Subdural space	16 (22.5)
Epidural+subdural space	3 (4.2)
Intracerebral parenchyma	4 (5.6)
Epidural+subdural+intraventricular space	2 (2.8)
Sella	4 (5.6)
Epidural space+sella	2 (2.8)
Subdural space+sella	2 (2.8)
Side	
Left	23 (32.3)
Right	29 (40.8)
Bilateral	11 (15.5)
Midline	4 (5.6)
Midline+left, right, bilateral	2 (2.8), 1 (1.4), 1 (1.4)
Dispersion	
Scatter	43 (60.6)
Concentrated	28 (39.4)
Depth	
Superficial	45 (63.4)
Deep	19 (26.8)
Superficial+deep	7 (9.9)
Brain areas	
Frontal	17 (23.9)
Frontotemporal	7 (9.9)
Frontoparietal	2 (2.8)
Frontotemporoparietal	7 (9.9)
Temporal	21 (29.6)
Temporoparietal	2 (2.8)
Temporoocipital	2 (2.8)
Parietal	2 (2.8)
Occipital	3 (4.2)
Sella	4 (5.6)
Sella with other lesions	4 (5.6)

TABLE 4. Site of skull fracture associated with traumatic pneumocephalus

Location of skull fractures	No. (%)
None (pneumocephalus alone)	5 (7.0)
Frontal bone	13 (18.3)
Temporal bone	23 (32.4)
Parietal	1 (1.4)
Occipital	3 (4.2)
Lamdoid	1 (1.4)
Skull base	9 (12.7)
Frontotemporal	3 (4.2)
Frontoparietal	1 (1.4)
Temporoparietal	2 (2.8)
Frontotemporoparetal	1 (1.4)
Skull base+other bones	9 (12.7)

hours.[18] The author predicts that the incidence of TP after TBI will increase in future research. As the resolution of CT continues to improve and the patient transportation system to the hospital continues to increase in speed, the possibility of detecting pneumocephalus on CT will increase.

There were no significant differences in age distribution, GCS score at admission, surgery rate (23.9%) and mortality (4.2%) of the patients with TP when compared to TBI patients

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TABLE 5. Intracranial traumatic lesions associated traumatic pneumocephalus

Intracranial lesions	No. (%)
None (pneumocephalus alone)	14 (19.7)
TSAH	11 (15.5)
AEDH	13 (18.3)
ASDH	14 (19.7)
TICH	2 (2.8)
TIVH	1 (1.4)
AEDH+TSAH	3 (4.2)
AEDH+TICH	1 (1.4)
AEDH+ASDH	3 (4.2)
ASDH+TSAH	5 (7.0)
ASDH+TICH	4 (5.6)

TSAH: traumatic subarachnoid hemorrhage, AEDH: acute epidural hematoma, ASDH: acute subdural hematoma, TICH: traumatic intracerebral hemorrhage, TIVH: traumatic intraventricular hemorrhage.

enrolled through KNTDBS (**TABLE 1**). However, the proportion of males in the patients with TP group was significantly higher (83.1%) compared to the KNTDBS (70.8%). In addition, TP tended to be more prevalent in the younger age group (41–60 years) than general TBI patients (≥61 years). In this study, some demographic characteristics of patients with TP, such as gender and age distribution, were similar to previous TP related studies.[16,18] Sharma et al.[16] reported a rate of 90% males, 70% of mild GCS, and 9% of surgical rates and 21–40 years old was the most common at 46% in a study of 100 patients with TP. This was similar to the author's study. Steudel et al.[18] found that the proportion of males was 79.6%, highest between 21–40 years old was 34.7%, but the mortality (40.8%) was due to severe GCS (77.6%) in the study of 49 patients with TP. Yucetas et al.[20] reported that the proportion of males was 78.1% and the mean age was 32 in 73 patients with TP who performed conservative treatment. The mortality rate was 20% in TP patients with meningitis. The number of patients with TP who died in this study was 3 and the mortality was 4.2%, similar to mortality of KNTDBS (4.8%). Surgery rate (23.9%) was also similar to that of KNTDBS (25.5%). These suggest that the outcome of TP was more affected by other traumatic intracranial lesions than TP.

Among 2,617 head trauma patients registered in the KNTDB, TAs accounted for 39.5% (n=875) excluding non-available (n=201) and unknown cause (n=198) cases. On the other hand, the most common cause of TP was TA (52.1%, n=37), and the proportion of passenger car TAs (n=22) was significantly higher among total TA in this study (**TABLE 2**). Sharma et al.[16] reported that motor vehicle accidents were the most common cause (50%), followed by falls (33%) and penetrating brain injury (10%). Yucetas et al.[20] also reported that the most common cause of TP was TAs (38.4%). The incidence of post-traumatic meningitis after TBI is 0.2–17.8%, which increases significantly in the presence of basal skull fractures, pneumocephalus or CSF leakage.[4,6] In 1971, North[14] reported 50% CSF leakage and 25% meningitis in 41 traumatic intracranial aerocells diagnosed by X-ray. He also reported a mortality rate of 16% and insisted that antibiotics should be given while determining a neurosurgical opinion.

In 2004, Eftekhar et al.[5] reported that the overall meningitis rate in TBI with TP was 21.5%. That study again disapproved that the use of the prophylactic ceftriaxone in patients with TP is useful. However, it was shown that CSF rhinorrhea and intracranial hemorrhage are primary risk factors, and that without such factors, intradural location of air and air volume above 10 mL may be considered as secondary risk factors for meningitis in patients with TP. However, even in patients with CSF rhinorrhea or intracranial hemorrhage, the prophylactic ceftriaxone was found to be ineffective in preventing meningitis.[5] The study of Steudel et

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al.,[18] which included a large number of severe patients, reported 12 with CSF leakage (24.5%) (rinorrecha 7, otorrecha 5). They performed prophylactic antibiotic therapy with cefotaxim and gentamycin in TP patients with CSF leakage, protruding brain tissue, aspiration pneumonia, scalp lacerations and multiple injuries. None of the patients who underwent prophylactic antibiotics therapy showed evidence of meningitis, either clinically or at autopsy. Sharma et al.[16] reported that CSF leakage was reported as a common complication in 18% of cases, and rhinorrhea (10%) was more common than otorrhea (8%). There is still a controversy in the use of prophylactic antibiotics with TP or meningitis due to CSF leakage. The most commonly used antibiotics are ceftriaxone and ampicillin/sulfadiazine, but there are no significant difference in the overall incidence of meningitis depending on the type of antibiotics.[3] There were 7 CSF leakages (9.6%) (rhinorrhea 5, otorrhea 2) in this study, which was significantly less than previous studies. Prophylactic antibiotics with ceftriaxone (2 g per day) were injected intravenously in all patients. Although the duration of use of antibiotics varied depending on the degree of intracranial and extracranial injury, general condition, and surgery, an average of 10 days in groups that performed the surgery and an average of 7 days in groups that did not perform the surgery. In all patients, there were no infection-related complications such as meningitis. This was may be due to the exclusion of patients with FCCD and that a diagnosis was possible even in cases of small TP as the resolution of CT was high. This may also suggest that the immediate administration of antibiotics in the emergency room and rapid treatment after TBI reduced the likelihood of infection-related complications. The overall prognosis of TP is not significantly different from other patients with otherwise similar TBI.[18] Steudel et al.[18] reported that a group of patients with frontobasal injuries or depressed fractures combined with TP had a good prognosis. The prognosis in the group of patients with TP was more favorable in those with fractures of the frontal or ethmoid sinuses compared to patients with fractures of the laterobasal fracture of the skull. They reported that the prognosis was poor due to several air bubbles in different intracranial positions because it of a very serious acceleration or deceleration injury. In this study, there was also no difference in mortality compared with mortality in the KNTDBS. This could be because prognosis is more often determined by other accompanying intracranial TBI lesions than by TP itself.

Most of the TP was located in the epidural or subdural space (**TABLE 3**). Even though the TSs were scattered, it includes an epidural or subdural space (88.7%, n=63). This is thought to be because the air from the outside has to pass through the epidural and subdural space while it enters the deep portions of the brain. Eight cases (11.8%) occurred in the sella region, which is relatively susceptible to TP. The air inflow into the intraventricular space was the least common cause (n=2, 2.8%). This is because the air inflow paths are fractures of skeletal pneumaticity include the paranasal sinuses and some of the mastoid cells or deep scalp lacerations just over the skull fractures, TP is most likely to occur in the frontotemporal region. The dura is also thin and tightly attached to the frontotemporal bones and is prone to laceration.[8,12] In this study, a scatter pattern (60.6%) was more common than a concentrated pattern (39.4%) and was mostly located in the frontal and temporal areas (81.7%). This pattern was similar to previous studies.[16,18]

Although less than 3% of all skull fractures are accompanied by TPs, about 8% of fractures of the paranasal sinuses and skull base and 41% of fractures of sella turcica are accompanied by TP.[8] In this study, most fractures were identified in the frontotemporal region and 60.6% of all fractures included the frontotemporal region (**TABLE 4**). It is noteworthy that bone fractures could not be identified in CT in 5 patients with TP (7%). Although TP can also

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occur even without the evidence of skull fracture, this is rare.[2] Two mechanisms have been suggested to account for TP without skull fractures, both of which have low intracranial pressure resulting in the "suction" of air through an epidural defect.[2,11,19] While the first mechanism includes a vertical pressure creating pressure gradients within the CSF system, the second mechanism includes a ball valve effect that allows air to enter the intracranial space through the multiple foramens of the skull base or the craniocervical junction pathway. All 5 patients without fractures identified on CT also showed no evidence of CSF leakage in this study. Another possibility is that a very small skull fracture may have been located between slices of CT and failed fracture detection, even if CT was taken with thin-slices.

ASDH and AEDH are the primary causes of TBI. Therefore, the most common intracranial lesions associated with TP were ASDH and AEDH, and 43 cases (60.6%) included both traumatic conditions (**TABLE 5**). However, while ASDH (37.5%) had a much higher incidence than AEDH (15.1%) in the data from the KNTDB, the incidence of the 2 traumatic conditions were similar in this study on TP associated with intracranial lesions. It is thought that the reason for this difference of incidence was that AEDH was more often accompanied by a fracture than ASDH. Fourteen patients (19.5%) did not have any intracranial traumatic lesions on CT except TP. Therefore, more careful confirmation is needed because TBI patients with only simple fractures may be accompanied by TP.

Study limitations

There are several limitations of this study. First, among the locations of TP on CT, there were ambiguous cases between the epidural and subdural spaces, therefore, it is possible that the author's subjective judgment was biased. Second, because the number of patients included in this study was small, the risk factors associated with death and prognosis could not be investigated. Third, because this study is not a prospective study, it may not have accurately represented the overall characteristics of the PT and it is possible that it gave false information about the use and effectiveness of antibiotics in relation to the complications. Fourth, among the site of skull fractures, basal skull fractures of frontal and temporal bones were classified as frontal and temporal bone fractures, which may cause confusion in the total number of fractures of the skull base (**TABLE 4**). Sixth, no significant clinical significance has been achieved by the TP alone without the influence of other traumatic intracranial lesions.

CONCLUSION

The author identified epidemiology, clinical, and radiological characteristics of patients with TP caused by TBI. Demographic characteristics of patients with TP and KNTDBS were similar except for the proportion of males. Although there are many limitations to this study, the author believes this study is worthwhile as it considers the various characteristics of TP, which have had relatively little clinical interest. This study could be used to obtain useful information as a reference in conducting research related to TBI and may also be helpful in developing treatment plans for traumatic intracranial lesions associated with TP.

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