A rare case of tension pneumocephalus after head trauma

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Abstract

OBJECTIVES: We present a rare case of tension pneumocephalus (TP) after head trauma not involving mask ventilation but based on pathological respiration pattern, kussmaul breathing.

TP is a rare condition and exceptionally rare when positive airway pressure has not been applied. In this particular case, the vacuum and one-valve intracranial effects causing pneumocephalon were instead accentuated by a pathological pattern of respiration due to severe ketoacidosis – a condition not previously reported in the literature.

METHODS: This is a case report showing a rare cause of pneumocephalon caused by patients’ own respiration. We retrospectively reviewed the patients’ journal to find the cause of his severe pneumocephalon. The patient has not been ventilated and the only cause of TP in this case seems to be his own rapid and pathological breathing caused by ketoacidosis.

CONCLUSION: In the presented case, pathologic deep and rapid respiration exerted additional pressure on the dural fistula allowing ambient air at a pressure above ICP to drive itself into the intracranial space.

The patient has not been ventilated and the only cause of TP in this case seems to be his own rapid and pathological breathing caused by ketoacidosis.

The patient had at no time received positive airway ventilation. He was intubated only immediately prior to the trauma scan. The neurosurgeon conferred advised making a burr hole in the right temporal region to relieve the pressure pneumo-encephalon before transport to the neurosurgical department. The burr hole was performed and air bubbled out of the incised dura. Upon arrival at the neurosurgical department the patient underwent another CT-scan showing sustained pneumocephalon with compression on the brain bfrontally and air compressing the brainstem. Furthermore an acute subdural haematoma was noted located under the burr hole. The patient was at that point sedated to GCS three, intubated, non-responsive and had normal-responsive pupils. The pt. underwent acute operation; making use of the fracture lines in the skull and the subdural haematomas was evacuated. The brain that had been exposed to air for 20 h had a yellowish and dry surface and was hyperaemic. An epidural haematoma was removed. A dural tear was sutured. Finally an intracranial pressure (ICP) monitor was inserted for follow-up.

1. Introduction

Tension pneumocephalus is infrequent and the limited number of cases reported all involve positive airway pressure ventilation after head trauma with an undiagnosed skull fracture. We present a rare case of violent pneumocephalus after head trauma not involving mask ventilation but based on pathological respiration pattern, kussmaul breathing.

2. Case report

A 53 year old man, known with insulin dependant diabetes mellitus, sustained a heavy blunt trauma to the back of the head from a crane arm. He was found estimated 15 h after the incident and was transported to the nearest hospital without intubation. On admission to the local trauma centre his Glasgow Coma Score (GCS) was eight (Eyes two, Motor five, Verbal one). Pupillary response was normal. There was bleeding from the external meatus of the right ear and a periorbital haematoma on the right side. There were no other exterior signs of trauma. Initial blood gas results showed Plasma glucose 36 mmol/l, PH 7.3, Plasma lactate 5.8 mmol/l, base excess −8.3 mmol/l. There was diminished skin turgor and a strong smell of acetone emanating from the patient. He had deep mechanical type respiratory movements of the thorax corresponding to kussmaul breathing. The patient had severe metabolic acidosis with blood glucose level 36 mmol/l at admission. CT scan (Fig. 1) revealed multiple skull base fractures through the clivus, the petrus bone to the foramen magnum and through the frontal sinus and the sphenoid sinus and massive tension pneumencephalon (TP) bi-frontally compressing the cerebrum to about 2/3 of the cranial volume (Figs. 1 and 2).

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Figs. 1 and 2. Initial CT scan at trauma centre showing multiple skull base fractures through the clivus, the petrus bone to the foramen magnum and through the frontal sinus and the sphenoid sinus and massive tension pneumencephalon. Inserted through a small twist drill hole into the parenchyma pre-coronally on the opposite site. Post-operative CT showed reduction of the intracranial air and a normal calibrated brain with normal surface contours and parenchyma (Fig. 3).

The patient was admitted to neurointensive care unit, under surveillance with contiguous ICP monitoring. He gradually gained consciousness. Two weeks later the patient was still intubated due to pulmonary causes, GCS ten (Eyes three, Motor six, Verbal one) and with no obvious focal neurological deficits and was referred to a neurorehabilitation centre. Two years later, the patient is awake, has GCS 15 and no cognitive deficits. The patient suffers chronic regional pain syndrome and has daily headaches.

3. Discussion

Ketones are weak acids and the hydrogen ions produced must be buffered. When the bicarbonate ions buffering capacity is used, plasma pH and bicarbonate decline and respiration is stimulated. A rapid, deep breathing termed ‘Kussmaul respiration’ therefore follows metabolic acidosis. Abdominal pain and vomiting are common presenting features that may result from the acidosis. Coma is uncommon; most patients are lucid or exhibit mild-to-moderate disturbance of consciousness. We believe that the rapid and shallow respiration in this case has pushed the air intracranial, has caused the increasing pressure to the brain and the result is pneumocephalon.

Pneumocephalus develops due to a cranial or facial fracture through which air enters the intracranial cavity. TP is characterized by continued build-up of air within the cranial cavity, due to valve-effect usually in relation to a skull base fracture. Other common causes are surgery, tumours and infections.

Head position during cranial surgery, duration of surgery, nitrous oxide (N2O) anaesthesia, intraoperative osmotherapy, hyperventilation, spinal anaesthesia, barotrauma, continuous CSF drainage via lumbar drain, epidural anaesthesia are suggested as surgical causes.

Meningitis, otitis media, orbital emphysema and high pressure air applied into the orbit is also suggested as reasons to pneumocephalus without bone fracture. The air is considered to enter from tenon fascis.

Although the condition is generally asymptomatic, pneumocephalus of sufficient volume is implicated in postoperative lethargy, headaches, confusion, hemiparesis, abducens nerve palsy, mental changes and even seizure, in which cases treatment may be necessary.

Two mechanisms have been proposed to explain TP. 1. The pathophysiologic process starts with cerebral spinal fluid leak in the presence of associated discontinuity of the cranium and leptomeningeal disruption. Subsequent development of relative negative intracranial pressure results in a sufficient “vacuum effect” to cause additional accumulation of air within the cranial cavity. This air is distributed in the subarachnoid space.

2. In the presence of a “one-way valve” at the site of the leptomeningeal tear, the positive end tympanic pressure exceeds the intracranial pressure, and air is forced into the cranial cavity. When the intracranial pressure exceeds the pressure within the air collection, the “one-way valve” closes, thus preventing the egress of the trapped air. In this mechanism, the abnormal air is usually distributed in the extradural space.

In the presented case, pathologic deep and rapid respiration exerted additional pressure on the dural fistula allowing ambient air at a pressure above ICP to drive itself into the intracranial space. The loss of CSF from skull base fractures created a void space and relative negative pressure, allowing air to bubble in and fill the void.

4. Conclusion

TP is rare condition and exceptionally rare when positive airway pressure has not been applied. In this particular case, the...
vacuum and one-valve effects were instead accentuated by a pathological pattern of respiration due to severe ketoacidosis and have caused tension pneumocephalon. This is a condition not previously reported in the literature.

Conflict of interest

The authors declare that there are no conflicts of interest.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Authors’ contribution

Dr. Frederikke Rosendal has found the case and written the case report part of the article. Also he has reviewed and made relevant changes in the article. Shima Bjerrum has written the rest of article along with the title page and has contact with the patient.

References

1. Pillai P, et al. Traumatic tension pneumocephalus: two cases and comprehensive review of literature. Opus 12 Scientist 2010;4(1):6–11.
2. Schirmer CM, Heilman CB, Bhardwaj A. Pneumocephalus: case illustrations and review. Neurocrit Care 2010, http://dx.doi.org/10.1007/s12028-010-9363-0.
3. M. Natrress, Diabetic ketoacidosis.