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Pneumocephalus and Cranio-Cervico-Facial Subcutaneous Emphysema: An Unusual Complication of Craniotomy

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Abstract **Case Report**

Pneumocephalus and cranio-cervico-facial subcutaneous emphysema is an unusual complication in the postoperative period of craniotomy. We report a case of a 74-year-old patient who was operated on for a huge left temporoparietal hematoma, and who presented this complication 24 hours after admission to the intensive care unit. We highlight the rarity of this association in cranial surgery, as well as the possible mechanisms for development of these complications.

Keywords: Cerebral hematoma; Craniotomy; Pneumocephalus; Subcutaneous emphysema.

INTRODUCTION

Pneumocephalus (PNC) is the presence of air in the intracranial cavity secondary to communication with the extracranial compartment, it is frequently found in cranial surgery with variable proportions. Association with extensive subcutaneous emphysema (SCE) is an unusual complication. We report a case of a patient who presented postoperatively 24 hours after a craniotomy for evacuation of cerebral hematoma a PNC with extensive cervical and craniofacial SCE. We highlight the rarity of this association in cranial surgery, as well as the possible mechanisms for development of these complications.

CASE REPORT

A 74-year-old patient presented to the emergency department with a sudden headache accompanied by decreased level of consciousness. The patient was being monitored for diabetes mellitus, arterial hypertension, and dyslipidemia. He was on insulin, losartan 25 mg/day and simvastatin 20 mg/day. Physical examination reveals a CGS of 12 (E3V4M5), symmetrical pupil with positive light reflex in both eyes, a right hemicorporal deficit, blood pressure: 160/90 mmhg, heart rate: 75 beats/min, *pulse oximetry*: 93%. Laboratory test results Hb: 12 g/dl, *WBC*: 8000/µl, platelets: 240000/µl, PT: 75%, aPTT: 31 seconds, CRP: 08 mg/l**.** Cerebral CT scan showed a huge left temporoparietal hematoma with a mass effect on the midline (figure 1), The patient underwent emergency craniotomy, evacuation of the hematoma and placement of a subgaleal drain, he was then transferred to the surgical intensive care unit for postoperative management. The patient showed good neurological improvement, and was ventilator-weaned and extubated on 24 hours later. Nevertheless, the clinical examination was marked by the presence of a facial deformity with swelling of the left hemiface and persistent occlusion of the left eye. Palpation revealed cervicofacial subcutaneous crepitations on the side of swilling. Cranial and cervico-facial CT scan revealed a left temporoparietal PNC with a left cervical and craniofacial SCE (Figure 2 and 3). Respiratory origin was initially suggested because of mechanical ventilation, but examination of the upper airways and *tracheobronchial fibroscopy* revealed no abnormalities. No deterioration in consciousness was observed and the patient remained respiratory stable, he was kept under supervision in ICU for 48 hours, and transferred then to the *department* of *Neurosurgery*. The emphysema regressed spontaneously without complications after 72 hours. The patient was discharged after 7 days.

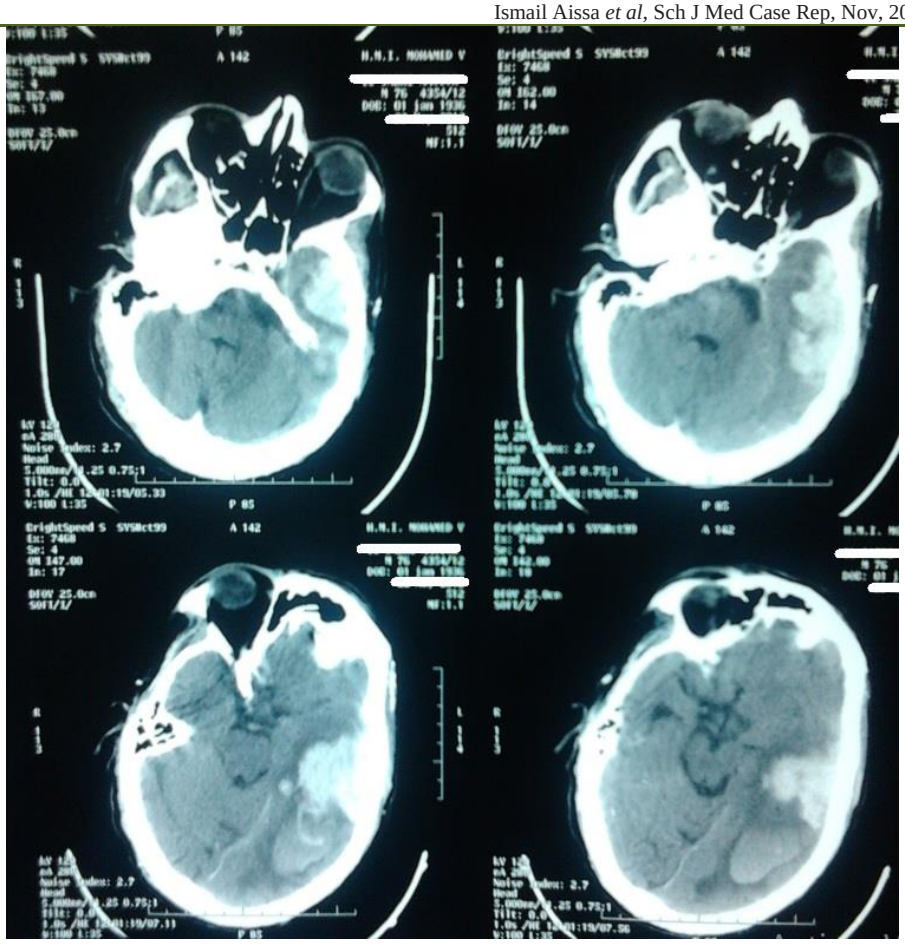

Figure 1: Preoperative axial cranial CT scan showing a left temporoparietal hematoma with a mass effect on the midline

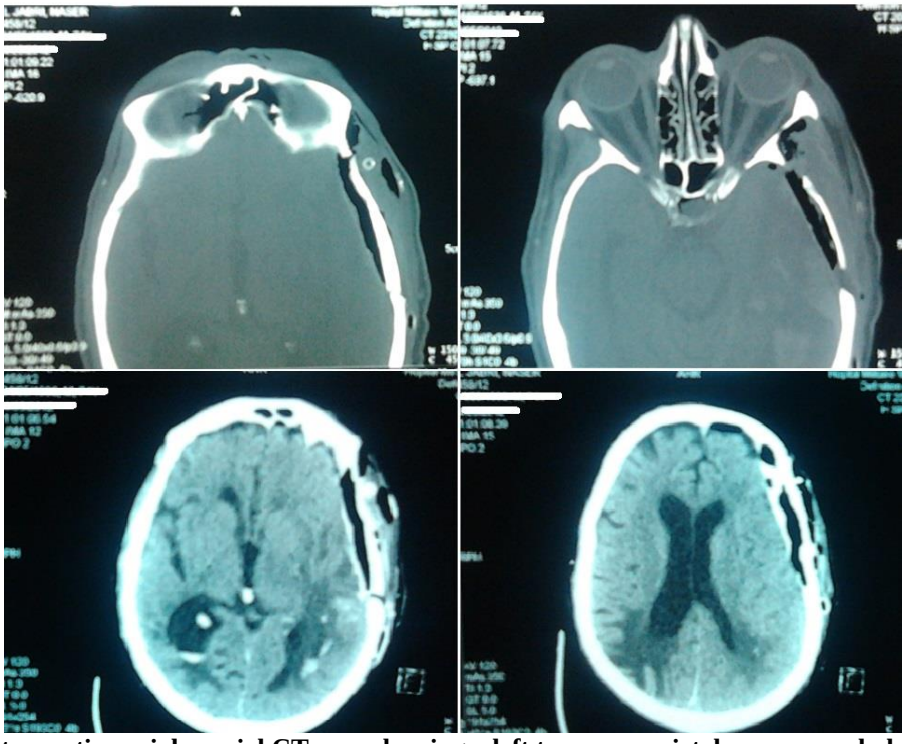

Figure 2: Postoperative axial cranial CT scan showing a left temporoparietal pneumocephalus and cranial subcutaneous emphysema

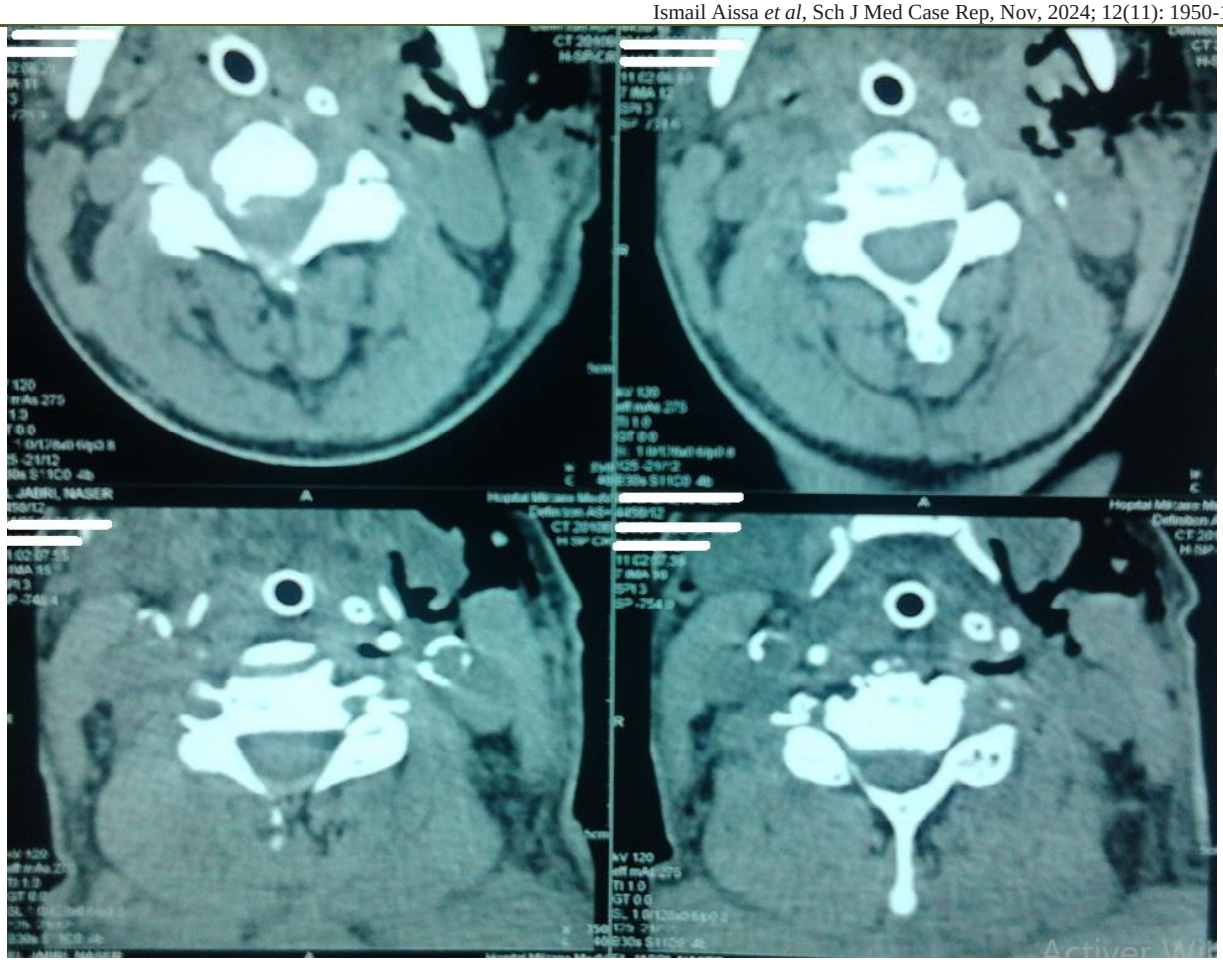

Figure 3: Postoperative axial Cervicofacial CT scan showing a left cervical and cervico-facial subcutaneous emphysema

DISCUSSION

Cervical and craniofacial SCE occurring postoperatively may result from several mechanisms, it may be induced by spontaneous rupture of the esophagus secondary to postoperative vomiting (Boerhaave syndrome) [1], or by oropharyngeal or tracheal trauma which may occurs; during traumatic intubation, inflation of the endotracheal cuff tube, repeated intubation attempts, repositioning of endotracheal tube without cuff deflation, coughing during intubation and movement of patient's head and neck after intubation [2, 3]. Other patient-related factors can induce tracheal injury, such as congenital anomalies of trachea, weakness of the tracheal membrane, chronic steroid use and chronic obstructive pulmonary disease [4, 5]. In our patient *tracheobronchial fibroscopy* and cervicofacial CT scan shows no evidence of oropharyngeal or tracheal injury, and then none of the mechanisms mentioned above explained the observed SCE.

Pneumocephalus is usualy expressed in the subdural space, but can be also located in the subarachnoid, epidural, intraventricular, or intracerebral space [6]. The accumulation of intracranial air can be acute (<72 h) or delayed (≥72 h) [7]. Others authors classified it as early PNC (<7 days) or late PNC (≥7 days) [8]. It can be located in the epidural, subarachnoid, intraventricular, intracerebral, or subdural space, with the subdural space most frequent. The common site is frontal, followed by the occipital and temporal areas [7]. PNC most often results from traumatic or iatrogenic skull fractures. Infections, including chronic otitis media, sinusitis, meningitis and septicaemia have been reported [9]. Other cases of spontaneous PC have also been reported [10]. Common causes of PNC are cranial or spinal surgery as well as some ENT operations, such as paranasal sinus surgery, nasal septum resection, or nasal polypectomy [7]. In some cases, the incidence of PNC after supratentorial craniotomy has been reported to be 100%. 66% of these PNC were judged to be moderate or large, and the incidence decreased to 75% by postoperative day 7. Nonetheless, 11.8% of the scans obtained during the 2nd postoperative week had PNC that were judged to be moderate or large [11].

Many mechanisms have been proposed to explain the occurrence of PNC. In the operative period PNC is hypothesized to be caused by intraoperative loss of cerebrospinal fluid (CSF) and the subsequent intracranial entry of air replacing the CSF as the pressure in the two cavities balance, which corresponds to the theory of “inverted soda bottle” effect [12]. A more

unusual mechanism is the production of gas in situ due to infection by germs forming gas [13]. In our patient there was no evidence of an infectious cause, the possible explanation would be the first theory, so air penetrated into subdural region after evacuation of parenchymal haematoma, the cerebral re-expansion would facilitated the passage of air into the subgaleal region through the craniotomy breaches and then its extension into the subcutaneous region at craniofacial and cervical level favoured by the severe laxity of the affected tissues with age.

The association of PNC and SCE after craniotomy is very rare in literature; we found a case rapported by Muhammad AP *et al*., 2023 of a PNC and SCE following ventriculoperitoneal shunt surgery for hydrocephalus in a baby of 11-month-old [14]. The mechanism explaining immediate PNC associated with SCE following shunt procedures is excessive drainage CSF during insertion of the ventricular shunt leading to decreased cerebral pression, the non-compliant ventricular system does not collapse after excessive drainage of CSF, leading to the influx of air into the ventricular system, they suspected that SCE was related to pneumonia or an infected abdominal wound, and it extend into the scalp and slowly infiltrate the intracranial from the defect present after the ventriculoperitoneal shunt insertion contributed to the development of the PNC.

CONCLUSION

In the case of association of PNC and cranio-cervico-facial SCE post-craniotomy, several factors can cause it to occur especially oropharyngeal or tracheobronchial injuries occurring during anesthetic procedures of airway management. In our patient who did not have this type of lesion the exact mechanism has not yet been fully understood. We hope further researches will shed light on this phenomenon in the future.

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