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Pneumocephalus as a cause of seizure after lumbar spine surgery. A case report and review

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Abstract : We report an atypical case of pneumocephalus following lumbar spine surgery for degenerative purposes. This clinical presentation is characterized by a seizure in the recovery room. The underlying etiology is a peroperative dural tear. After an initial assessment with CT-scan the evolution is rapidly favorable under conservative treatment. Our review of literature compares nine similar cases and the management options. Anesthesiologists play a major role in the prevention of risk factors and in the differential diagnosis of symptomatic cases, particularly to exclude postoperative life-threatening situations.

Keywords : Pneumocephalus ; spine surgery : seizure ; dural tear ; surgical complication.

Introduction

Pneumocephalus is the presence of air in the intracranial cavity. The clinical presentations are multiple and non-specific, but most cases remain asymptomatic. A wide variety of etiologies exists. Intracranial or cervical procedures are the most commonly reported. We describe an atypical case of seizure in the recovery room as a result of pneumocephalus following a dural tear during lumbar degenerative surgery.

Case report

A sixty-year-old man developed a first episode of seizure in the recovery room after a lumbar surgery for spinal stenosis. His medical history includes obesity (119 kg, BMI 33.3), arterial hypertension and type 2 diabetes. The patient undergoes his 8th lumbar surgery due to refractory sciatica in the right lower limb despite previous operations in other institutions. He does not describe any postoperative complication besides chronic pain. The preoperative medical exams (complete blood count, electrocardiography (EKG) and echocardiography) show no significant anomaly.

The surgical procedure lasted 98 minutes for an occupation time of 157 minutes. The patient is installed in prone position. The technique performed is a median 8 cm skin incision, L2-L4 paraspinal muscle release and L2-L4 recalibration without laminectomy or discectomy. The blood loss is less than 100 ml and a non-suction drain is placed at the end of the procedure. A dural tear, described in the postoperative report, is not sealed due to difficult access localization.

No anesthetic incident is recorded. The induction is achieved with sufentanil 15 µg, lidocaine 100 mg, propofol 200 mg and rocuronium 50 mg. Clonidine 150 µg, ketamine 25 mg, tranexamic acid 1 g and cefazolin 2 g are administered after the intubation. Recurrent episodes of arterial hypotension are noticed during the procedure (the lowest mean arterial pressure recorded is 43 mmHg), but rapidly resolved with several boluses of ephedrine (total dose of 24 mg). The fluid management requires 500 ml of crystalloids (Ringer-lactate) and 500 ml of colloids (Volulyte 6%). The anesthesia maintenance is provided with volatile anesthetic (desflurane).

In the recovery room the patient reports unusual and intense frontal headaches without nausea, photo-/phonophobia or other signs of intracranial hypertension. He is fully orientated. The vital parameters are constant with a blood pressure of 120/80 mmHg. After a few minutes he developed a tonic-clonic seizure, initially localized in the right lower limb, but rapidly generalized. A treatment with intravenous midazolam is administered and effective. Normal serum glucose is measured. The

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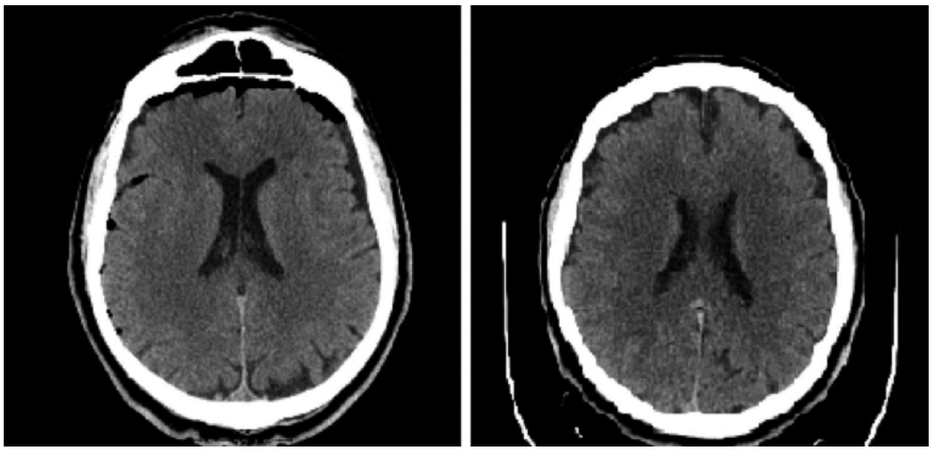

Fig. 1. — Postoperative axial brain CT-scans (day 0 and D +5).

physical examination performed is non-contributive with no signs of motor or cranial nerve deficit. Finally a head CT-scan is quickly carried out to exclude acute intracranial pathology and reveals mild frontal pneumocephalus with no mass effect or cerebral hemorrhage (Fig. 1).

Conservative management with bed rest and analgesics in an orthopedic surgery unit is introduced after prolonged monitoring in the recovery room. The patient remained hospitalized for six days. His clinical status was encouraging with a rapid attenuation of the headaches and no seizure recurrence. The electroencephalogram (EEG) performed two days after surgery showed no significant anomaly. A control CT-scan at day five showed a significant reduction of the pneumocephalus (Fig. 1).

Informed consent was obtained from the patient for the publication of this case report.

Discussion

The concept of pneumocephalus (PNC) is defined by the presence of gas anywhere in the intracranial cavity (intraventricular, intra-parenchymal, subarachnoid, subdural, epidural) as a result of various etiologies (Table 1). Intravascular air embolism can be included in this definition. PNC is constant after craniotomy and significant in two thirds of cases, albeit not symptomatic nor pathological (1). Anesthetic causes consist of traumatic nasotracheal intubation; spinal or epidural anesthesia, especially when air was used for the loss-of-resistance technique (2). Nitrous oxide inhalation (due to its preferential transfer compared to nitrogen into air-filled cavities) and positive end-expiratory pressure above 5 cm H_2O (increasing the cerebrospinal fluid (CSF) pressure and favoring its leakage in case of dural tear) are known as contributing factors. The typical clinical presentation is non-specific and includes nausea and vomiting, headache and confusion. Confirmation of diagnosis is achieved by CT-scan due to its availability, high sensitivity and specificity (3). In rare severe cases the PNC induces intracranial hypertension associated with marked neurological symptoms (motor deficit such as cranial nerve palsy or consciousness impairment). This situation is called tension pneumocephalus. As a result of the high amount of intracranial air the CT-scan images are often characterized by the separation of the frontal tips called 'Mount Fuji Sign' and gas located in the cisterns called 'air bubble sign' (3,4). In extreme cases cerebral herniation can appear (5).

PNC is seldom described following lumbar surgery for degenerative pathologies, i. e. without planned meningeal incision. Our literature review, adapted from Gauthé et al. (6), highlighted nine cases among adult patients (Table 2). An iatrogenic dural tear is systematically the suspected cause

Table 1

Described etiologies of pneumocephalus

Neurosurgery	Intracranial surgery (transsphenoidal surgery, shunt insertion, drainage), spine surgery.
Other surgeries	Endoscopic sinus, maxilla-facial surgery.
Trauma	Facial and head, cervico-thoracic, spinal.
Anesthesiology	Spinal and epidural anesthesia, naso-tracheal intubation, nitrous oxide inhalation, nasogastric tube insertion, CSF drainage.
Others	Congenital skull defect, intracranial neoplasm, infection with gas-producing organisms, barotrauma, chronic otitis, air embolism. Uncommon : recurrent colorectal carcinoma, diverticulitis.

Table 2

Review of cases of pneumocephalus after lumbar spine surgery for degenerative pathologies

Authors	Age (years)	Symptoms	Delay of symptoms	Management	Duration of symptoms	Peroperative DT assessed	Drain
Ayberk *et al.* (13)	55	HA, N/V	Day 2	BR, ST, OT	?	No	S
Cesar *et al.* (24)	41	HA, PP, stiff neck, dysarthria	Day 1	BR, ST	7	No	S
Dhamija *et al.* (25)	63	HA, CO, subfebrile	Day 1	BR, OT	5	Yes, sealed	?
Gauthé *et al.* (6)	69	SZ, lumbar pain	Day 1	BR, OT, anti-epileptics	? DC: 10	No	S, RR dis-connexion
Karavelioglu *et al.* (14)	56	HA, NV, DZ	Day 1	BR, ST, OT	9	No	S, CF
Kizilay *et al.* (16)	30	HA, NV	Day 2	BR, antibiotics	3	Yes, not sealed	S, CF
Pirris *et al.* (12)	65	Diplopia	RR	OT	< 1	Yes, sealed	?
Turgut *et al.* (15)	47	HZ	Day 2	BR	? DC: 14	Yes	S, CF
Yun *et al.* (17)	59	HA, DZ	Day 1	BR	? DC: 14	Yes, sealed	?
Current case	60	HA, SZ	RR	BR, midazolam	<1	Yes, not sealed	No S

HA : headache, NV : nausea or vomiting, PP : photophobia, CO : confusion, SZ : seizure, DZ : dizziness, RR : recovery room, BR : bed rest, ST : symptomatic treatment (antalgics, antiemetics), OT : oxygenotherapy, DC : discharge (days), DT : dural tear, S : suction, CF : described as contributing factor.

and was objectified peroperatively in six cases. The physiopathology of PNC remains unclear. The presumed mechanisms include air entry due to 'inverted soda bottle' phenomenon, wherein a loss of CSF by the tear leads to negative intracranial pressure (7,8). A second explanation is the 'ball valve phenomenon': a valve effect allows air to pass the meningeal barrier, but not to escape (9). We suggest that a combination of these hypotheses occurs in practice. Regarding our patient a dural tear was described in the postoperative report and we also support it as the cause of the PNC. It was not sutured because of difficult surgical access, but no CSF leakage was visualized at the end of surgery. Given the early symptoms and the rapid favorable evolution, we assume that the air entrance appeared during the procedure and was not later aggravated. Dural tears occur in up to 7.6 % of primary surgeries and 15.9 % of revision lumbar surgeries (10). Evidence shows that dural tears visualized during surgery should be sealed if possible (11). The most frequent complications are infections (abscess, meningitis, arachnoiditis), dural fistula and pseudomeningocele, intracranial hypotension and subdural hematomas (10,12). PNC is unusually reported. The lack of symptoms and, as a result, the non-systematic approach to imaging performed could result in a significant underestimation of its incidence after procedures such as cervical surgery (13). A similar situation can obviously occur after lumbar surgery, making the estimation of its incidence difficult.

A suggested contributing factor for PNC is the use of vacuum suction devices aggravating the CSF loss. These are underlined for this purpose in three cases of our review (14,15,16). A fourth author infers a disconnection of the drain in the recovery room as a source of air entry (6). The benefit/risk balance of their utilization remains uncertain in case of sutured tears. In the presence of non-closed breach, vacuum drainage is not recommended due to the risk of amplified CSF loss (11). However, no correlation has been established yet regarding PNC, but an aggravation of the inverted soda bottle phenomenon is possible. Considering the possibility of missed lesions, the utilization of suction devices should be carefully considered in situations of high risk of dural tear such as revision surgeries, even if no breach is directly objectified.

The position of the patient could be another contributing factor for PNC. Prone position, associated with obesity in our case, increases intra-abdominal and secondarily CSF pressures, promoting the CSF leakage or the opening of occult defects. Some authors suggest head down position during surgery (16,17). Others identify reverse Trendelenburg positioning as a risk factor (12). Fully supine position in the recovery could also prevent cephalad migration of entered air, even if the classic Fowler position (elevated bed head) is mostly adopted for its respiratory benefits. Adapting the position of the patient per- and postoperatively is a simple tool that could be used to prevent PNC in high risk situations, e. g. if a dural tear is objectified.

Postoperative seizure has numerous origins such as major hypoxic events, hypoglycemia, spinal cord injury, massive loss of CSF, anesthetic agents (high dose of tranexamic acid or local anesthetic, seizure-like phenomena with propofol) and intracranial events (hemorrhage, infection, ischemic pathology). In the absence of these causes, given the time of onset of the symptoms and in view of the CT-scan findings, PNC appears to be the most likely cause of seizure in our case.

Seizure is described but unusual in the presence of PNC, even in high risk situations as craniotomy (1). The appearance of symptoms is also mostly delayed. In a review of 295 confirmed cases of symptomatic PNCs from various origins, in 1967 Markham reports a seizure in 24 of them (8.1%) (19). Only one author reports it, in our specific cases review, the day after surgery. No specific anti-epileptic medication is recommended. However, the use of benzodiazepines as treatment or anti-epileptic agents for recurrence prevention is described and seems justified in case of persistent seizure (6,18). Since the favorable evolution of our patient no additional treatment was administered after the initial bolus of midazolam. The neurological follow-up, including the electroencephalogram, was carried out after the end of the hospitalization.

The treatment in the surgical unit included painkillers, rest in semi-sitting position and appropriate hydration. PNC is mostly reabsorbed without complications after a few weeks (14), the usual management is conservative. The use of osmotic diuretics is described without clear evidence of effectiveness. Another efficient therapy consists of the administration of high concentration oxygen (optimal 68%) (20) or hyperbaric oxygen, and could be used to increase the nitrogen and thus the air resorption (21). The potential side effects of hyperoxymia and the practical requirements limit these techniques to highly symptomatic cases. For severe situations such as intracranial hypertension, decompression by drainage of the air cavity or craniotomy is necessary (5,22).

Neuro-imaging is needed for the initial assessment. For uncomplicated cases, conservative treatments and follow-up with daily clinical examinations are indicated. Repeated CT-scans can be avoided in view of the excellent evolution. We suggest non-invasive techniques such as transcranial Doppler ultrasonography, allowing an easy and reliable investigation without X-ray exposition (23). However, the presence of intracranial air can be a source of artefacts.

Conclusion

This paper adds to the literature a rarely described complication of lumbar spine surgery for degenerative pathologies. Pneumocephalus remains asymptomatic in most cases, resulting in an underestimation of its incidence. It must yet be kept in mind for the differential diagnosis of immediate and delayed postoperative seizure or other symptoms as intense headache and neurological impairment. These symptoms are non-specific, particular attention must be paid in case of peroperative dural tear and lumbar revision surgeries. The examination of the patient with regards to identify signs of intracranial hypertension must be systematic in light of the potential life-threatening consequences. CT-scan is the standard exam for the initial assessment. Further researches should investigate the effectiveness of transcranial Doppler ultrasonography for the follow-up. Regarding the numerous etiologies of pneumocephalus and the variability of the clinical presentations, an updated and extended review of literature is needed to establish practical guidelines for its management. Anesthesiologists play a perioperative key role for the diagnosis, treatment and follow-up, but also for the avoidance of contributing factors, while adapting their anesthetic technique and the patient position in case of high-risk situations.

References

1. Reasoner D.K., Todd M.M., Scamman F.L. and Warner D.S. 1994. The incidence of pneumocephalus after supratentorial craniotomy. Observations on the disappearance of intracranial air. Anesthesiology 80(5): 1008-1012.
2. Kuczkowski K.M. and Benumof J.L. 2003. Images in anesthesia: headache caused by pneumocephalus following inadvertent dural puncture during epidural space identification: is it time to abandon the loss of resistance to air technique? Can. J. Anaesth. 50(2): 159-60.
3. Schirmer C., Heilman C. and Bhardwaj A. 2010. Pneumocephalus: case illustrations and review. Neurocrit. Care 13: 152–158.
4. Ishiwata Y., Fujitsu K., Sekino T., Fujino H., Kubokura T., Tsubone K. and Kuwabara T. 1988. Subdural tension pneumocephalus following surgery for chronic subdural hematoma. J. Neurosurg. 68(1): 58-61.
5. Çelikoğlu E., Hazneci J. and Ramazanoğlu A.F. 2016. Tension pneumocephalus causing brain herniation after endoscopic sinus surgery. Asian J. Neurosurg. 11(3): 309-10.
6. Gauthé R., Latrobe C., Damade C., Foulongne E., Roussignol X. and Ould-Slimane M. 2016. Symptomatic compressive pneumocephalus following lumbar decompression surgery. Orthop. Traumatol. Surg. Res. 102(2): 251-3.
7. Osborn A.G., Daines J.H., Wing S.D. and Anderson R.E. 1978. Intracranial air on computerized tomography. J. Neurosurg. 48(3): 355-9.
8. Donovan D., Iskandar J., Dunn C. and King A. 2008. Aeromedical evacuation of patients with pneumocephalus:

outcomes in 21 cases. Aviat. Space Environ. Med. 79(1): 30-35.
9. Ozturk E., Kantarci M., Karaman K., Basekim C.C. and Kizilkaya E. 2006. Diffuse pneumocephalus associated with infratentorial and supratentorial hemorrhages as a complication of spinal surgery. Acta Radiol. 47(5): 497-500.
10. Khan M.H., Rihn J., Steele G., Davis R., Donaldson W.F., Kang J.D. and Lee Y. 2006. Postoperative management protocol for incidental dural tears during degenerative lumbar spine surgery: a review of 3183 consecutive degenerative lumbar cases. Spine 31: 2609-13.
11. Wolff S., Kheirredine W. and Riouallon G. 2012. Surgical dural tears: prevalence and updated management protocol based on 1359 lumbar vertebra interventions. Orthop. Traumatol. Surg. Res. 98(8): 879-86.
12. Pirris S.M. and Nottmeier E.W. 2013. Symptomatic pneumocephalus associated with lumbar dural tear and reverse trendelenburg positioning: a case report and review of the literature. Case Rep. Neurol. Med. 2013.
13. Ayberk G., Yaman M. and Ozveren M. 2010. Symptomatic spontaneous pneumocephalus after spinal fusion for spondylolisthesis. J. Clin. Neurosci. 17: 934-936.
14. Karavelioglu E., Eser O. and Haktanir A. 2013. Pneumocephalus and pneumorrhachis after spinal surgery: case report and review of the literature. Neurol. Med. Chir. 54(5): 405-7.
15. Turgut M. and Akyüz O. 2007. Symptomatic tension pneumocephalus: an unusual postoperative complication of posterior spinal surgery. J. Clin. Neurosci. 14: 666-668.
16. Kizilay Z., Yilmaz A. and Ismailoglu O. 2015. Symptomatic pneumocephalus after lumbar disc surgery: a case report. Open Access Maced. J. Med. Sci. 3(1): 143-5.
17. Yun J.H., Kim Y.J., Yoo D.S. and Ko J.H. 2010. Diffuse pneumocephalus: a rare complication of spinal surgery. J. Korean Neurosurg. Soc. 48(3): 288-90.
18. Lam K., Prasad A., Fehlings M. and Venkatraghavan L., 2014. Pneumocephalus: an unusual case of postoperative seizure after intradural spine surgery. Can. J. Anesth. 61: 969-970.
19. Markham J.W. 1967. The clinical features of pneumocephalus based upon a survey of 284 cases with report of 11 additional cases. Acta Neurochir. 16(1): 1-78.
20. Gore P.A., Maan H., Chang S., Pitt A.M., Spetzler R.F. and Nakaji P. 2008. Normobaric oxygen therapy strategies in the treatment of postcraniotomy pneumocephalus. J. Neurosurg. 108: 926-9.
21. Paiva W.S., de Andrade A.F., Figueiredo E.G., Amorim R.L., Prudente M. and Teixeira M.J. 2014. Effects of hyperbaric oxygenation therapy on symptomatic pneumocephalus. Ther. Clin. Risk Manag. 10: 769-773.
22. Dabdoub C.B., Salas G., Silveira Edo N. and Dabdoub C.F. 2015. Review of the management of pneumocephalus. Surg. Neurol. Int. 6: 155.
23. Cardim D., Robba C., Bohdanowicz M., Donnelly J., Cabella B. and Liu X., et al. 2016. Non-invasive monitoring of intracranial pressure using transcranial Doppler ultrasonography: is it possible? Neurocrit. Care 25: 473-491.
24. Cesar A.B., Pedro C.G., Ulises H.P.M. and Bernabe D.R. 2015. A rare complication of lumbar spine surgery: pneumocephalus. J. Spine. S. 6: 2.
25. Dhamija B. and Saxena A. 2011. Pneumocephalus-a possible cause of post-spinal surgery confusion. Journal of the Royal Society of Medicine 104(2): 81-83.