

# Fatal Cerebral Arterial Air Embolism Following Uneventful One-level Lumbar Decompressive Surgery

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## Abstract

Several cases with fatal cardiac output failure following coronary and ventricular air embolism and few cases with cerebral venous gas and air emboli have been reported after lumbar surgery. This is, to the best of our knowledge, the first case presented with a fatal cerebral arterial embolism in the course of lumbar spine surgery without any cardiac and pulmonary symptoms or necessity of CPR. Conceivable pathways of the air embolus towards left posterior cerebral artery were either from right into left atrium or, far less probable, nevertheless having been reported for large air masses, trans-pulmonary. No patent oval foramen, however, had been detected prior to surgery by transthoracic sonography. This paper should create awareness about that rare complication and its caveats further be discussed by experts.

**Keywords:** Lumbar spine surgery • Air embolism • Cerebral infarction • Fatal outcome

## Introduction

A 79-year old male obese (BMI 36.2) patient underwent decompressive lumbar spine surgery for left side dominant spinal canal stenosis L3-L5 (Figure 1) accompanied by neurogenic claudication and partial loss of quadriceps function. The patient had reported a history of collapses during a mountain hike, resulting in a Search-and-Rescue salvage via helicopter. Radiation for a non-metastasized prostatic cancer had been scheduled, yet was delayed for spinal surgery.

There was a history of a single event of a Transient Ischemic Attack (TIA) without sequelae, postoperative bleeding under enoxaparine bridging after endoscopic colon surgery, and prostatic cancer without metastases. 18 months preoperatively, the vertebral and carotid arteries had been checked for patency. A Simvastatin therapy had been administered since.

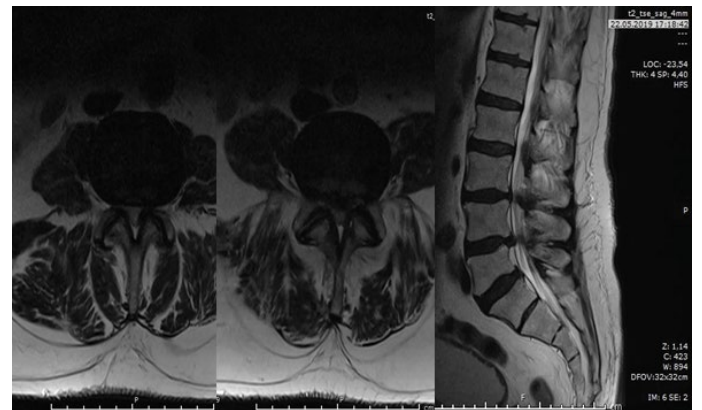
## Case Presentation

He suffered from concomitant atrial fibrillation (AF) and was thus preoperatively bridged with Tinzaparine in exchange for his usual phenprocoumon prophylaxis. InnoHep® 4.500 I.E. was administered s.c. until 16 hours prior to surgery. Patient had been under oral anticoagulants with Procoumon (Marcumar®) until 11 days prior to surgery. Coagulation related parameters 4 ½ hours prior to surgery were INR 1.1, PTT 24.7, Quick 89.

The procedural and patient risk of bleeding under continued Phenprocoumon was estimated via our standardized preoperative Patient Blood Management Assessment as being high (not for massive bleeding but in terms of avoiding spinal compression even from small amounts of hemorrhage). Thus phenprocoumon was interrupted early and, due to the comorbidities, the

above-mentioned bridging, according to risk stratifying considerations was put in place.

There was no preoperative evidence of cardiac insufficiency and hemodynamically relevant patent oval foramen (PFO) according to



**Figure 1.** Preoperative axial MRI scans (from left) of L3/4, L4/5, and corresponding lateral view, depicting the spinal stenosis in both levels and a disc protrusion at L4/5. Additional functional radiographs had been taken to rule out instability (not depicted).



**Figure 2.** Type of position of the patient on the OR table as applied in the presented case. Note the horizontal position of the trunk, avoidance of hyperlordosis, and semi soft cushions in place.

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preoperative transthoracic sonography, but admittedly, standard transthoracic echocardiography has a low sensitivity (high false negative rate) for detection of PFO. Chest radiography (Figure 4) was normal. ECG showed atrial fibrillation at an average frequency of 63 bpm. GFR appeared reduced (42 ml/min), CRP slightly elevated (2.7 mg/dl), and Hb slightly reduced (12.7 g/dl).

The patient was placed prone on the OR table, avoiding any kneeling or head-down position. No Wilson-type frame was used. (Figure 2) The operation site thus was about level with the heart. No central venous line was placed during surgery. Surgery itself comprised of uneventful hemi laminectomy at L4 and hemifacetectomy L3/4 on the left side with crossover decompression. No burr was used, no bleeding or accidental laceration of the dural sac ensued. There was no relevant bleeding noted during surgery. Neither H<sub>2</sub>O<sub>2</sub> nor hemostatic agents were utilized. Positive End expiratory Pressure (PEEP) during entire surgery/anesthesia never exceeded 5mmHg.

We noted no drop in the end-tidal CO<sub>2</sub> which stayed normal (31 – 42 mmHg) throughout surgery lasting 83 minutes. The lowest systolic blood-pressure was 100/60 mmHg, single event, 13 minutes after skin incision. Also no significant hypertension occurred. We used a 4-channel SedLine® sedation monitor (Masimo®) to delineate the depth of anesthesia which showed an uneventful course of surgery and anesthesia and normal values all the way.

Permanent ECG, pulse oximetry, noninvasive blood-pressure monitoring (oscillometric method), and invasive intra-arterial blood-pressure monitoring were employed, alongside with neuromuscular function monitoring (StimPot®). No neuromonitoring of lumbar nerve roots was considered necessary.

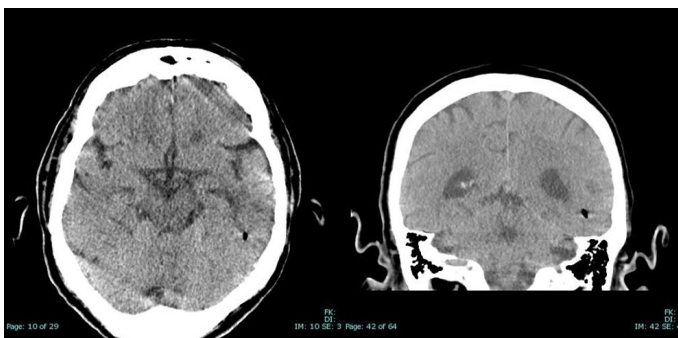
After the patient had been turned back supine and extubated he exhibited an epileptiform convulsion during transfer from carriage to bed. The patient relaxed briefly under Midazolam and was awake further on. A cerebral CT scan (CCT) 100 minutes after the first seizure exhibited air embolism in a temporal branch of the left posterior cerebral artery (Figure 3).

There had been no evidence, whatsoever, of insufflation of considerable amounts of air via the peripheral I.V. line which had been under supervision via the anesthesiologist's team throughout the surgery. Being cardiopulmonarily stable, the patient did not receive a central venous catheter (CVC). He was administered steroids for reduction of brain edema via peripheral venous lines. Neurological deterioration on the following morning resulted in loss of consciousness and reintubation.

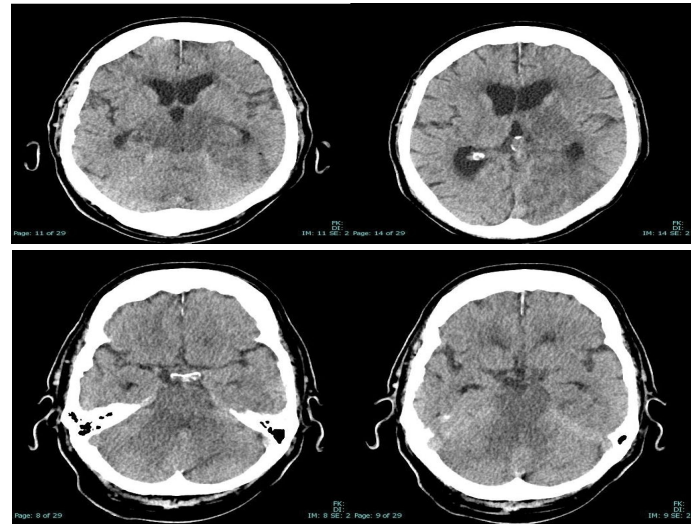
A second CCT without angiography, obtained 16 hours later, presented overt hypodense demarcation of the left thalamus while the intravascular air bubble had almost disappeared (Figure 4). All neurological symptoms were concordant with these findings. No cardiac and/or pulmonary failure had occurred to this point and a chest radiograph still appeared normal.

## Results and Discussion

The patient succumbed to the arterial cerebral infarction after artificial ventilation was successively withdrawn due to his dismal prognosis on the third day after surgery. Prior to best supportive care, informed consent was obtained from the relatives according to guidelines of the hospital's Ethics Committee.



**Figure 3.** The final CT scans show marked left-hemispherical posterior infarction. The affected sector exceeds the area supplied by the posterior cerebral artery and also comprises of the coverage areas of basilar and upper cerebellar artery.



**Figure 4.** The early CT scans show a major air bubble in the suspected course of the left temporal branch of the posterior cerebral artery, still without infarction.

Hyperbaric oxygenation [1] had been considered not useful according to consultation of in-house stroke unit experts. An autopsy was refused.

### Historical review of the condition

According to an exhaustive PubMed research, few partly lethal and non-lethal outcomes with cardiac arrest and/or output failure following coronary and ventricular air or gas embolism after or during lumbar spine surgery have been described [2, 4,6,7]. Also, one lethal case after cerebral venous gas embolism due to H<sub>2</sub>O<sub>2</sub> wound irrigation [3] and one non-lethal case with cerebral arterial air embolism [5] have been reported. Yet, the latter was caused not by surgery itself but by concomitant lumbar peridural anesthesia with air insufflation during catheter placement.

No other source of air entry than lumbar epidural veins (e.g. via arterial monitoring or false placement of central venous catheter) could be detected. What makes our tragic case unique is the isolated and fatal arterial cerebral infarction in the course of an otherwise "standard" lumbar procedure.

Neither any AV-malformations in the patient's or his family's history were recorded. None such malformations were ever detected during imaging. No hereditary haemorrhagic telangiectasia bleeding was reported in the preoperative Patient Blood Management questionnaire which is employed in our hospital. The following diagnostic procedures were also retrospectively analyzed in this respect:

Cranial CT 02.10.2013 (after concussion of head and thorax) Chest radiograph 02.10.2013 Cranial CT 24.06.2018 (after suspected TIA) Sonography of the neck, carotid and vertebral arteries 24.06.2018 MRI of lumbar spine 22.05.2019 Chest radiographs had not shown any signs of pulmonary hypertension. Of course, small intrapulmonary shunts cannot be detected that way. Yet, intrapulmonary shunts had never been suspected in this particular case since the patient preoperatively had no clinical signs indicative for that disorder, was physically active, an active hiker and biker, etc.

This case is, to the best of our knowledge, the first published case of a fatal cerebral arterial air embolism in the course of lumbar spine surgery without any cardiac and pulmonary symptoms. This obviously very rare incident must not be confused with cerebral air embolism of cortical and subdural veins via azygos/hemiazygos veins. These are generally sparing the right atrium, and enter the brain via the left internal jugular vein from retrograde.

Thus, the term "venous cerebral air embolism" as commonly used in the heterogeneous references may be quite confusing and deserves clear definitions: Since no gas emboli from spinal arteries and spinal dural arteriovenous fistulae are known do date, the lumbar and lower thoracic origin of an (Figure 3) intracerebral air mass is presumably always "venous" but its trajectory, and thus its final destination, i.e. cerebral veins or arteries, maybe different.

## Rationale for diagnostics and treatment

Since already an obviously small amount of cranial intra-arterial air was fatal in this case, the common salvage procedures for removal of larger amounts of air from the heart by immediate aspiration via CVC from the right atrium, as recommended for cardiac failure, may not presumably not have been successful.

In cardiac catheter laboratories air embolism is often recognized by inferior ST elevation on the electrocardiogram due to air preferentially entering the upward facing origin of the right coronary artery when the patient is in a supine position. In this case, surgery was carried out with the patient in the prone position and air ejected from the left ventricle would probably have 'floated' along the posterior aspect of the ascending aorta and away from the downward-facing and anterior origins of the right, and to a lesser extent, left coronary arteries. This may explain why there were no cardiac complications to alert clinicians to the possibility of air embolism [6].

## Conclusion

In current cardiology practice the standard test for a PFO is contrast (agitated saline) transthoracic (or tranoesophageal) echocardiography with provocative manoeuvres (e.g. 'sniff' or Valsalva) to transiently open the inter-atrial communication. A PFO is generally not 'haemodynamically' important as left atrial pressure usually keeps the PFO closed. Yet, this is no standard preoperative procedure, at least in asymptomatic patients. So far, there is neither a published recommendation to exclude PFO patients from lumbar surgery, nor any guideline how to proceed in its presence, except "being aware" of the risk and avoiding positioning of the operating site above heart level. The aforementioned exams for PFO may be advisable in patients in whom a sitting position must be employed in the O.R. and laceration of bigger veins may be a considerable risk (e.g. neurosurgical and orthopaedic anterior neck surgery).

A PFO screening recommendation for all patients undergoing spine surgery cannot be derived from our experience.

The peripheral line was equipped with the usual air trap and was under continuous supervision, but special I.V. line filters for patients with PFO are available on the market, mainly for use with central I.V. lines. However, their use will not protect from air entry into epidural veins. Nevertheless, in the future we will advocate their use in case PFO is a reported condition, although their benefit in this respect is not statistically proven so far. This is owed to the still small number of symptomatic (and reported) air embolism cases in comparison to the increasing surgical activity worldwide over the last years.

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