CASE REPORT

Distributive shock, cardiac arrhythmias and multiple organ failure following surgery of a fourth ventricular epidermoid

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SUMMARY

A 33-years-old male patient presented with cardiac arrhythmias, acute shock and multiple organ dysfunction after the surgical removal of a massive epidermoid posterior to the brainstem. The patient initially presented with paraesthesia along the right C6 dermatome due to a big tumour at the brain stem. Surgical removal was performed without adverse events and he was transferred to our intensive care unit (ICU) immediately after the operation. Though initially showing a stable postsurgical course he developed cardiac arrhythmias and a state of acute distributive shock with consecutive multi organ failure. Extensive diagnostic measures could not identify a specific cause for this rapid deterioration. However, under carefully monitored symptomatic therapy the patient improved quickly, was extubated 72 h after admission and discharged from the ICU 6 days later. The follow-up did not show any persisting neurological deficits and no evidence of a residual tumour in the MRI-study.

BACKGROUND

Surgical affection of the brain stem can possibly lead to cardiac arrhythmias, distributive shock and consecutive multi organ failure. Neurointensivists should be aware of this life-threatening condition potentially occurring soon after neurosurgical procedures. The diagnosis should always be made by excluding all other known causes and symptomatic treatment should be initiated immediately.

CASE PRESENTATION

We hereby report the course of a 33-years old otherwise healthy male patient (181 cm, 62 kg body weight, body mass index 19) who presented with increasing undulant left-sided paraesthesia. A cranial MRI revealed a 44×31×41 mm tumour posterior to the brainstem with extension from the fourth ventricle down to the craniocervical junction (figure 1 A, B).

At admission the patient showed paraesthesia along the C6 dermatome of his right arm and no paresis or other neurological symptoms. His medical history was empty. After common pre-arrangement he underwent a suboccipital craniotomy for removal of the lesion. The surgery was performed in a semisitting position and monitored by transoesophageal ultrasound for air embolism. There was a futile attempt to puncture the right jugular vein and the patient developed a

subcutaneous haematoma. In a second attempt cannulation of the right subclavian vein was successful.

Surgery and anaesthesia were uneventful except for unexplained hypercapnia which improved after modification of the ventilator settings. There were no signs of air embolism and no cardiac arrhythmias were reported. After the procedure the patient was transferred to the intensive care unit (ICU).

At admission to the ICU he was ventilated in a pressure controlled mode with a fraction of inspired oxygen of 0.3 and had stable haemodynamics without any catecholamine support. The blood gas analysis showed sufficient oxygenation with a partial pressure of oxygen of 91.7 mm Hg and a partial pressure of carbon dioxide of 65.5 mm Hg. Initial sedation and analgesia were performed with propofol (120 mg/h) and remifentanyl (500 µg/h). Regarding this stable situation sedation and anaesthesia were ceased immediately after admission and the patient could be extubated after 1 h.

After extubation the patient presented with left-sided hemiparesis, was agitated and not responsive to any question. Oxygen saturation deteriorated despite oxygen supply (10 L/min) via a face mask and the attending physician decided to reintubate the patient. The blood gas analysis immediately before reintubation showed a PaO₂ of 41.8 mm Hg and a PaCO₂ of 34.1 mm Hg. Patient presented with tachycardia, tachypnea, hypertension and had a cold sweat.

With regard to the hypercapnia during the surgery, the rapid deterioration of gas exchange after extubation, and the postsurgical neurological abnormalities an urgent CT-scan of head and thorax was performed.

The thoracic CT-scan showed bilateral massive atelectasis and a pulmonary embolism could be excluded (figure 2). Additionally, left and right-sided pleural effusion could be detected. The CCT showed regular postsurgical conditions.

During the course of the first day the patient had progressive hypotension, unresponsive to a fluid challenge so that vasopressor therapy had to be started. He had cardiac arrhythmias and a progressive gas exchange disorder. He fulfilled criteria for moderate acute respiratory distress syndrome (ARDS) with a PaO₂/FiO₂ ratio of 116 mm Hg and bilateral infiltrates in the chest X-ray.¹ A transthoracic cardiac ultrasound was performed showing hyperdynamic kinetics with a normal ejection fraction without any indication of acute heart failure or any other myocardial affection. Blood cultures

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Figure 1 (A) MRI of the tumour prior to surgical intervention showing axial T1-weighted image of a presumed epidermoid. The tumour has a space occupying extension from the fourth ventricle down to the craniovertebral junction with strong brainstem compression. (B) Sagittal T2-weighted image of the epidermoid.

and cultures of tracheal secretion did not reveal evidence for an infectious cause. Laboratory tests showed a transient increase in serum creatinine while the patient had always sufficient diuresis.

Furthermore, the patient developed leucocytosis (23.2 Gpt/L) and had fever after the first day on ICU. The norepinephrine dose had to be increased (3.22 µg/kg BW) despite a massive fluid intake of 8700 mL (fluid balance +7200 mL) during the first 12 h. Under an assumed diagnosis of hospital acquired septic shock with unknown focus we started a calculated antibiotic therapy with imipenem/cilastatin and vancomycin. Haemodynamic monitoring was expanded by a catheter for transpulmonary thermodilution and pulse contour analysis (PICCO2, Pulsion medical systems AG, Munich, Germany).

Figure 2 Thoracic CT scan after intensive care unit admission showing pleural effusion and atelectasis.

Measurement revealed a cardiac index of 2.8 L/min/m², an extravascular lung water index of 17 mL/kg and a central venous pressure of 14 mm Hg indicating fluid overload together with massive vasoplegia. Regarding these parameters haemodynamic and fluid therapy was adjusted and diuretics were given in order to reduce lung oedema.^{2 3} Additionally, a therapy with continuous infusion of 200 mg/24 h hydrocortisone was initiated, which had no immediate effect on catecholamine doses. The pleural effusions were drained and due to his severe oxygenation disorder the patient was intermittently put in prone position.⁴ Following these therapeutic measures we saw a rapid improvement of haemodynamic instability and gas exchange that norepinephrine could be reduced and the patient could be weaned from mechanical ventilation. He was finally extubated 72 h after ICU admission.

OUTCOME AND FOLLOW-UP

After extubation he had sufficient muscle power with no persisting signs of the previously diagnosed hemiparesis and was fully orientated. On day 9 after surgery the patient could be transferred from ICU to the general ward and was discharged 5 days later in good health. The histological examination confirmed the presumed diagnosis of an epidermoid without any signs of malignancy. Three months later the patient presented for post-surgical examination without any symptoms with a post-operative MRI showing no residual tumour.

DISCUSSION

We report of a patient with sudden development of distributive shock with multiple organ failure after an uneventful resection of an epidermoid adherent to the brainstem. As no underlying disease or catastrophic event could be identified for the reported course, only symptomatic therapy could be applied.

Postneurosurgical gas exchange disorders can be induced by pulmonary embolism including air embolism, transfusion related acute lung injury, or unexpected bacterial contamination for example, following aspiration of gastric content or by central venous catheterisation. Furthermore, shock following allergen exposure and cardiogenic shock have to be considered. However, pulmonary embolism or acute right heart failure were

excluded via CT scan and cardiac ultrasound, microbiological testing revealed no result, and no transfusions of blood products were made. The patient did not have any known allergies and the appearance of shock had no relation to the application of potential allergens. As a septic shock, allergic reactions or cardiogenic shock have been excluded or at least seemed to be very unlikely we hypothesise that surgical affection of the brain stem alone may have led to vasoplegia, capillary leakage and multiple organ dysfunctions. Even more, because the tumour was adherent to the floor of the fourth ventricle and the brainstem below, including the regions for circulatory and breathing control.

Pulmonary dysfunction following cerebral affection has been described mainly in patients with head injury. A small part of patients with severe head injury present with lung oedema, denominated as neurogenic pulmonary edema (NPE). NPE is a rare clinical sign, mainly described after cerebral insults in combination with raised intracerebral pressure (ICP).⁵ Massive catecholamine release as a main pathophysiological trigger for NPE is discussed. However, in the presented case, there was neither a massive cerebral insult nor indication for raised ICP. Cerebral insults may also cause local or systemic inflammation resulting in a complex pattern of cytokine and mediator release accounting for lung capillary leakage. This has been reported only in experimental brain injury or substantial brain damage.⁶ Few reports describe comparable clinical courses followed by brain stem affection. Brown *et al* report of a patient who developed hypertension, hypoxia and fatal NPE following surgery of the medulla oblongata. The main pathological finding which was suspected of having caused NPE was gross swelling of the

medulla.⁷ Bilik *et al*⁸ describe a patient with symptomatic haemangioblastoma of the medulla oblongata who developed ARDS. On the other hand hypotension has been described as a rare manifestation of medulla oblongata lesions. Patients may present with orthostatic hypotension as a first symptom which is assumed to be induced by affection of sympatoexcitatory neurons.⁹ The combination of hypotension and vasomotor instability also has been described as a result of medullary stroke or arteriovenous fistula in the medulla.^{10–11} Neurogenically caused cardiac arrhythmias as a result of stroke or cerebral haemorrhage, especially subarachnoid haemorrhage have been described extensively.¹² However, the occurrence of arrhythmias have been also reported in isolated brain stem lesions.¹³

All discussed cases and NPE in general have in common that the cause of hypotension or lung failure has to be diagnosed by exclusion. In our case transient affection or local swelling caused by surgical manipulation of the medulla oblongata may explain the specific combination of symptoms as well as the rapid recovery.

Competing interests None declared.

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Learning points

- Shock, cardiac arrhythmias and consecutive organ failure can be caused by surgical affection of the brain stem. Neurointensivists should be highly aware of rapid deterioration after neurosurgical interventions and this life-threatening complication. We hypothesise that this complication could have a good prognosis under cautious critical care and as far as no permanent morphological brainstem defects exist.
- Severe shock with multiple organ failure after neurosurgical interventions can be induced by several reasons including microbiological focus, cardiogenic shock, allergic exposure or transfusion reaction. As this is a life-threatening condition, rigorous diagnostic work-up and immediate symptomatic therapy is mandatory.
- Neurogenic pulmonary oedema is an import differential diagnosis of respiratory distress or gas exchange disorders after cerebral damage.

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