Neurogenic Pulmonary Edema -the Lurking Peril

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Article ID: WMC00549
Article Type: Case Report
Submitted on: 07-Sep-2010, 05:50:12 PM GMT  Published on: 07-Sep-2010, 08:02:06 PM GMT
Article URL: http://www.webmedcentral.com/article_view/549
Subject Categories: ANAESTHESIA
Keywords: Neurogenic Pulmonary Edema, Obstructive Hydrocephalus, Ventriculoperitoneal Shunt, Resolution, Perioperative

How to cite the article: Kumar, L S, Durga P, Naik M, Ramachandran G. Neurogenic Pulmonary Edema -the Lurking Peril. WebmedCentral ANAESTHESIA 2010;1(9):WMC00549

Source(s) of Funding:
Nil

Competing Interests:
nil
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Abstract

The authors report on a patient with acute obstructive hydrocephalus due to cerebellar meatal metastatic lesion who presented with neurogenic pulmonary edema. The edema resolved on placement of the ventriculoperitoneal shunt. Because neurogenic pulmonary edema may be a unrecognized consequence of obstructive hydrocephalus, it is important to recognize this unusual association and provide appropriate diagnostic measures and treatment. This report addresses the importance of recognition of neurogenic pulmonary edema as a possible perioperative complication resulting from an increase in intracranial pressure.

Case Report

A female aged about 50 years, presented with progressive headache, holocranial and continuous type, associated with multiple episodes of vomiting and swaying gait for the last two months. Her MRI brain revealed fourth ventricular obstruction with obstructive hydrocephalus. As the patient was drowsy though responding to verbal commands and oriented an emergency ventriculoperitoneal shunt was contemplated. Her past history was unremarkable. She had no previous history of tuberculosis or respiratory illness. Her cranial nerves were intact. There was no motor or sensory deficit. She had a pulse rate of 65 per min. Her B.P was normal. Her respiratory rate was 22/min Electrocardiogram was normal. Chest X ray had haziness in the lung fields. All other clinical and biochemical investigations were normal. The SpO2 was to be around 84%, Her arterial blood gases showed a PaO2 53mmHg. The SpO2 increased to 92 % on administration of 100%O2 through the face mask of anesthetic circuit. As there was no other cause for the respiratory dysfunction like infection, aspiration, previous respiratory illness, a diagnosis of neurogenic pulmonary edema was considered. Anesthesia was induced with propofol 2 mg/kg and oral endotracheal intubation was facilitated with vecuronium 0.1 mg/kg body weight. Anaesthesia was maintained with air and oxygen mixture, adjusting the FiO2 to maintain an arterial saturation of > 90%. and isoflurane in the dial concentration of 1% along with atracurium and fentanyl infusion. No PEEP was applied. The O2 saturation was 95% with controlled mechanical ventilation with a FIO2 of 80%. Patient was haemodynamically stable and mean arterial blood pressure(MAP) was maintained at 80 mm Hg. Ventriculoperitoneal shunt was performed uneventfully. After the CSF drainage the oxygen saturation gradually increased to 100% and the FIO2 could be reduced to 50%.The neuromuscular blockade was reversed at the conclusion of surgery. The patient was awake, responding to verbal commands and normal motor power and tone regained. She was breathing spontaneously with no evidence of respiratory distress. Patient was extubated and monitored in neurosurgery intensive care unit for further observation. Postoperatively the PaO2 253 with O2 supplimentation with a simple face mask delivering a FIO2 of 40%. The patient was haemodynamically stable with a MAP of 90mm Hg. Her sensoriuum improved with return of consciousness and orientation to normal levels. Postoperative chest radiograph showed resolution of pulmonary edema. Definitive surgery of craniotomy and excision of the lesion was performed one week later. She had an uneventful intraoperative and postoperative course.

Discussion

Neurogenic pulmonary edema may be a consequence of a number of diverse central nervous system insults, including head trauma,(1-3) brain stem lesions,(4) resection of an acoustic neuroma,(5) rupture of intracranial aneurysm,(6-12) cerbellar haemorrhage,(13) leptomeningial carcinomatosis (14) excessive irrigation during endoscopic ventriculostomy,,(15) during angioplasty for vasospasm,(16) post-ictal period(17), pneumoencephalus(18) Due to non-specific clinical manifestation it often remains undiagnosed. It may manifest as acute pulmonary distress(19). Diagnosis requires a high index of suspicion, especially in the case of respiratory decompensation in neurosurgical patients. The pathogenesis of NPE probably involves overactivation of the sympathetic autonomic system with pulmonary hypertension(20), endothelial dysfunction(21) and increased vessel permeability.(22) There are two theories on how NPE occurs: the blast theory and the permeability defect theory. There is
Evidence for both theories, and NPE is probably the result of a combination of the two. The treatment is mainly supportive with the use of mechanical ventilation and alpha-adrenergic blocking agents while managing increased intracranial pressure. Anesthetic management of patient with NPE has not been reported widely. Anesthetic interventions like laryngoscopy and intubation may precipitate NPE. (23) It has been hypothesized that high levels of anesthesia might protect against the development of neurogenic pulmonary edema due to a more pronounced inhibition of the hypothalamic, brainstem and spinal vasoactive sympathetic centers. (24) An insufficient anesthesia level may not be able to inhibit the sympathetic nervous system during an injury of the central nervous system and thus predispose to development of neurogenic pulmonary edema. Therefore maintenance of adequate depth of anesthesia and attenuation of neoundocrine response to intubation is important. The presence of raised intracranial pressure and need to provide good brain relaxation for surgery may limit the application of PEEP which is recommended for the management of NPE. The reduced lung compliance and high intrathoracic pressure during mechanical ventilation may also pose a problem. The cerebrogenic autonomic and neurohumoral dysregulation due to intracranial hypertension may contribute to intraoperative haemodynamic dysfunction. This patient remained haemodynamically stable throughout the procedure. A high FiO2 was sufficient maintain optimal blood gases without the necessity for PEEP. Neurogenic pulmonary edema may to resolve after treatment of underlying condition. NPE after aneurismal sub-arachnoid haemorrhage was shown to resolve after endovascular coiling. (25) and after ventriculoperitoneal shunt in a patient with shunt malfunction. (26) There was no detectable systemic cause for the pulmonary edema in this patient. A thorough understanding of the pathophysiological mechanisms behind the development of NPE aids in the management of these patients to prevent further complications. In conclusion, when dealing with respiratory distress patients with CNS injuries, the possibility of additional damage from a NPE must be taken into consideration.

References

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