Case Report

Preoperative neurogenic pulmonary edema: A dilemma for decision making

Siva Kumar Reddy Lakkireddigari, Padmaja Durga, Madhukar Nayak, Gopinath Ramchandran

Department of Anesthesiology, Nizam's Istitute of Medical Sciences, Punjagutta, Hyderabad, Andhra Pradesh, India

Abstract

Neurogenic pulmonary edema may be a less-recognized consequence of obstructive hydrocephalus. The authors report a patient with acute obstructive hydrocephalus due to cerebellar metastatic lesion, who presented with neurogenic pulmonary edema. The edema resolved on placement of the ventriculoperitonial shunt. This report addresses the importance of recognition of neurogenic pulmonary edema as a possible perioperative complication resulting from an increase in intracranial pressure and the issues involved with anesthetic management of co-existing neurogenic pulmonary edema and intracranial hypertension.

Key words: Anesthesia, neurogenic pulmonary edema, obstructive hydrocephalus, postoperative, preoperative, management, resolution

Introduction

Acute neurogenic pulmonary edema (NPE) is an underdiagnosed yet a common clinical entity. It can occur after virtually any form of injury of the central nervous system. NPE is a potential contributor to the pulmonary dysfunction that occurs in these patients. High index of suspicion is required for the diagnosis. Presence of preoperative NPE presents a dilemma to the neuroanesthetist due to the divergent goals of management of raised intracranial pressure and pulmonary edema and also the possible adverse interaction of the two conditions when they co-exist. We report a patient with acute obstructive hydrocephalus due to cerebellar metastatic lesion who presented with NPE that resolved on placement of the ventriculoperitonial (VP) shunt.

Address for correspondence: Dr. Padmaja Durga, Department of Anesthesiology, Nizams Institute of Medical Sciences, Punjagutta, Hyderabad - 500 082, Andhra Pradesh, India.
E-mail: padmajanims@yahoo.com

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Case Report

A woman, about 50–year–old, presented with progressive headache, holocranial and continuous type, associated with multiple episodes of vomiting and swaying gait of two months duration. Her magnetic resonance imaging (MRI) brain revealed fourth ventricular obstruction with obstructive hydrocephalus. As the patient was drowsy though responding to verbal commands and oriented, an emergency VP shunt was planned. She had an unremarkable past history with 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 minute and her blood pressure was normal. Her respiratory rate was 22/min. Electrocardiogram was normal. Chest X-ray showed slight haziness in the right lung suggestive of pulmonary edema-[Figure1]. All other clinical and biochemical investigations were normal. The peripheral oxygen saturation (SpO_2) was around 84% and the arterial blood gases showed a PaO_2 53 mmHg. The SpO_2 increased to 92% on administration of 100% oxygen through the face mask of the anesthetic circuit. As there was no other cause for the respiratory dysfunction such as infection, aspiration, or previous respiratory illness, a diagnosis of NPE was considered. Anesthesia was induced with propofol 2 mg/kg and oral endotracheal intubation was facilitated with vecuronium 0.1 mg/kg body weight. Anesthesia was maintained with air and oxygen mixture, adjusting the FiO_2 to maintain an arterial saturation of >90%, and 1% isoflurane along with atracurium and fentanyl infusions. No

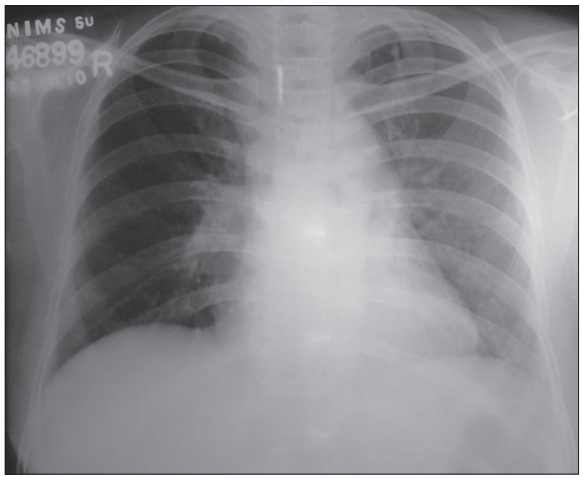

Figure 1: Preoperative chest radiograph of the patient showing pulmonary infiltrates suggestive of pulmonary edema

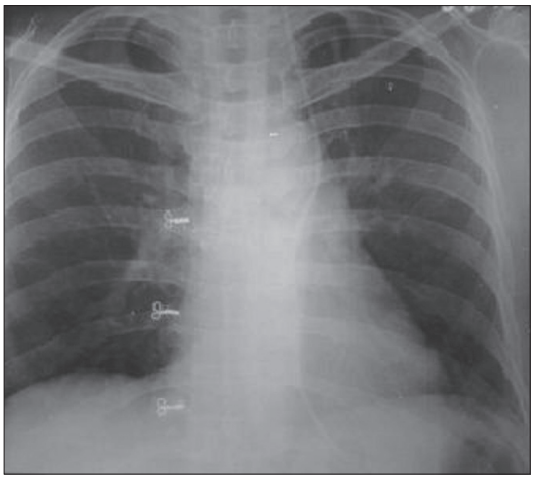

Figure 2: Immediate postoperative chest radiograph of the patient showing resolution of pulmonary infiltrates

positive end expiratory pressure (PEEP) was applied. The SpO_2 was 95% with controlled mechanical ventilation with a FiO_2 of 0.8. Patient remained hemodynamically stable and mean arterial blood pressure (MAP) was maintained at 80 mmHg. VP shunt was performed uneventfully. After the cerebrospinal fluid (CSF) drainage SpO_2 gradually increased to 100% and the FiO_2 could be reduced to 0.5. The neuromuscular blockade was reversed at the conclusion of surgery. The patient was awake, responding to verbal commands with normal motor power and tone. She was breathing spontaneously with no evidence of respiratory distress. Trachea was extubated and patient was monitored in the neurosurgery intensive care unit. Postoperatively, the PaO_2 was 253 mmHg on oxygen supplementation with a face mask delivering a FiO_2 of 0.4. The patient was hemodynamically stable with a MAP of 90 mmHg. Her sensorium improved with return of consciousness and orientation to normal levels. Postoperative chest radiograph showed resolution of pulmonary edema [Figure 2]. Definitive surgery of craniotomy and excision of the lesion was performed one week later. She had an uneventful intraoperative and postoperative course.

Discussion

NPE may be a consequence of a number of diverse central nervous system insults, including head trauma,[1] brain stem lesions,[2] rupture of intracranial aneurysm,[3] excessive irrigation during endoscopic ventriculostomy,[4] during angioplasty for vasospasm,[5] and postictal period.[6] It is often underdiagnosed due to its non-specific clinical manifestation. It may manifest as acute pulmonary distress.[7] 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,[8] endothelial dysfunction,[9] and increased vessel permeability.[10] There are two theories on how it occurs: the blast theory and the permeability defect theory, with evidence in favor of both of them. NPE is probably the result of a combination of these two. The treatment is mainly supportive using mechanical ventilation and alpha-adrenergic blocking agents for managing increased pulmonary arterial pressure.

Anesthetic management of patient with NPE has not been reported widely. Airway interventions like laryngoscopy and intubation may precipitate NPE.[11] It has been hypothesized that deep levels of anesthesia might protect against the development of NPE due to a more pronounced inhibition of the hypothalamic, brainstem, and spinal vasoactive sympathetic centers.[12] 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 NPE. Therefore, maintenance of adequate depth of anesthesia and attenuation of neuroendocrine response to intubation are important.

The use of PEEP in neurosurgical patients is limited by conflicting reports on its effect on intracranial pressure.[13,14] The presence of raised intracranial pressure and the need to provide good brain relaxation for surgery may limit the application of PEEP. Use of PEEP is safe when adequate MAP is maintained.[15] A high FiO_2 was sufficient to maintain optimal blood gases without the necessity for PEEP in this patient. The reduced lung compliance and high intrathoracic pressure during mechanical ventilation in the presence of pulmonary edema may also pose a problem for providing brain relaxation.

The cerebrogenic autonomic and neurohumoral dysregulation

due to intracranial hypertension may cause intraoperative hemodynamic dysfunction. Our patient remained hemodynamically stable throughout the procedure. There was no other detectable systemic cause for the pulmonary edema in this patient. A thorough understanding of the pathophysiological mechanisms behind the development of NPE helps in the management of these patients, thus preventing further complications. Neurogenic pulmonary edema may resolve after treatment of underlying condition. NPE after aneurismal sub-arachnoid haemorrage was shown to resolve after endovascular coiling.[16] and after ventriculoperitonial shunt in a patient with shunt malfunction.[17] NPE in this patient resolved after ventriculoperitonial shunt.

In conclusion, in patients with central nervous system pathology in respiratory distress, the possibility of diagnosis of NPE must be considered and the inciting pathology should be deliberated. Anesthetic management must be carefully titrated considering the divergent goals of NPE and intracranial hypertension.

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