Neurogenic Pulmonary Edema

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Abstract: Neurogenic pulmonary edema (NPE) is an increase in pulmonary interstitial and alveolar fluid that is due to an acute central nervous system injury and usually develops rapidly after the injury. It is still underappreciated in the clinical arena. Its sporadic and relatively unpredictable nature and a lack of etiologic-specific diagnostic markers and treatment modalities may in part be responsible for its poor recognition at the bedside. Neurogenic pulmonary edema in pediatric age group is largely underreported. Three patients presenting with acute onset neurogenic pulmonary edema following head trauma have been described by Neurogenic pulmonary edema in childhood by J. Ross Milley, Stephen K. Mark C. Rogers Nugent (1). We present a case of neurogenic pulmonary edema in a ten yr old female child following recurrent meningitis.

Keywords: Neurogenic Pulmonary Oedema; Pediatric Age, Chronic Meningitis, Raised Intra-Cranial Pressure.

INTRODUCTION

10 yr old female child initially became symptomatic in July 2015 with fever, headache & clinical signs of meningism. CSF analysis was done at this stage suggestive of acute pyogenic meningitis. Clinical recovery after 14 days of intravenous antibiotics. Seven months later she again gets admitted with fever, headache and projectile vomiting. Clinical signs of meningism present. CSF analysis shows turbid appearance with raised WBC: 40/cmm, neutrophil predominant, CSF proteins: 108 mg/dL, CSF glucose of 41 mg/dL with matching serum glucose: 136 mg/dL, CSF ADA: 10 IU/L, MRI (brain) done at this stage shows communicating hydrocephalus.

In the hospital, she is started on iv antibiotics under steroid cover. While being evaluated for possible underlying etiology of chronic meningitis (infectious/inflammatory/neoplastic), she suddenly develops acute respiratory distress, bradycardia, decerebrate posturing and b/l dilated pupils. In view of clinical features of raised intracranial pressure with impending coning, she is immediately intubated. Pink frothy expectoration is observed from the ET tube. Clinical and radiological picture of acute pulmonary edema present. She undergoes urgent neurological intervention with Rt frontal Omaya shunt. Over the next 24 hrs, she has a complete clinical and radiological resolution of pulmonary lesions. Neurological status improves to a GCS 15/15 and the patient is successfully extubated. Her cardiac evaluation including transesophageal echocardiography is normal. Further evaluation does not reveal any evidence of autoimmune pathology. CECT (Chest+Abdomen): normal study. The individual has been discharged in a stable condition. She is now ambulant and carrying out her day to day activities.

DISCUSSION

The syndrome of NPE has been recognized for over a century. In 1903, Harvey Williams Cushing described the connection between CNS injury and hemodynamic dysfunction. [2] Francois Moutier described the sudden onset of pulmonary edema among soldiers shot in the head in World War I [3]. Similar reports exist of observed alveolar edema and hemorrhage in the lungs of 17 soldiers dying after isolated bullet head wounds in the Vietnam War [4]. Data regarding morbidity and mortality following neurogenic pulmonary edema (NPE) have not been well documented, given the relatively low prevalence and likely underdiagnosis. A series of 457 patients with subarachnoid hemorrhage reported a 6% prevalence of severe neurogenic pulmonary edema. [5]
REFERENCES