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CLINICAL INFORMATION

Neurogenic pulmonary edema due to ventriculo-atrial shunt dysfunction: a case report

Ana Sofia Cruz[a,*], **Sónia Menezes**[b], **Maria Silva**[a]

[a] *Anesthesiology Department, Centro Hospital São João, Porto, Portugal*
[b] *Anesthesiology Department, Hospital Distrital de Santarém, Santarém, Portugal*

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KEYWORDS
Neurogenic pulmonary edema;
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Abstract
Background and objectives: Pulmonary edema is caused by the accumulation of fluid within the air spaces and the interstitium of the lung. Neurogenic pulmonary edema is a clinical syndrome characterized by the acute onset of pulmonary edema following a significant central nervous system insult. It may be a less-recognized consequence of raised intracranial pressure due to obstructive hydrocephalus by blocked ventricular shunts. It usually appears within minutes to hours after the injury and has a high mortality rate if not recognized and treated appropriately.
Case report: We report a patient with acute obstructive hydrocephalus due to ventriculo-atrial shunt dysfunction, proposed to urgent surgery for placement of external ventricular drainage, who presented with neurogenic pulmonary edema preoperatively. She was anesthetized and supportive treatment was instituted. At the end of the procedure the patient showed no clinical signs of respiratory distress, as prompt reduction in intracranial pressure facilitated the regression of the pulmonary edema.
Conclusions: This report addresses the importance of recognition of neurogenic pulmonary edema as a possible perioperative complication resulting from an increase in intracranial pressure. If not recognized and treated appropriately, neurogenic pulmonary edema can lead to acute cardiopulmonary failure with global hypoperfusion and hypoxia. Therefore, awareness of and knowledge about the occurrence, clinical presentation and treatment are essential.

PALAVRAS-CHAVE
Edema pulmonar neurogênico;
Hidrocefalia;
Neuroanestesia

Edema pulmonar neurogênico devido à disfunção da derivação ventrículo-atrial: relato de caso

Resumo
Justificativa e objetivos: O edema pulmonar é causado pelo acúmulo de líquido nos alvéolos e no interstício pulmonar. Edema pulmonar neurogênico é uma síndrome clínica caracterizada

* Corresponding author.
E-mail: fyzzita@gmail.com (A.S. Cruz).

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por edema pulmonar de início agudo após um acometimento súbito do sistema nervoso central. Pode ser uma consequência menos reconhecida de pressão intracraniana aumentada por causa da hidrocefalia obstrutiva por derivações ventriculares bloqueadas. Geralmente aparece em minutos ou horas após o insulto e tem uma alta taxa de mortalidade, caso não seja identificado e tratado adequadamente.

Relato de caso: Relatamos o caso de paciente com hidrocefalia obstrutiva aguda por causa da disfunção da derivação ventrículo-atrial, programado para cirurgia em caráter de urgência para a colocação de derivação ventricular externa, que apresentou edema pulmonar neurogênico no pré-operatório. A paciente foi anestesiada e o tratamento de manutenção instituído. No fim do procedimento, a paciente não apresentou quaisquer sinais de distúrbio respiratório, pois a redução rápida da pressão intracraniana facilitou a regressão do edema pulmonar.

Conclusões: Este relato aborda a importância da identificação de um edema pulmonar neurogênico como uma possível complicação no período perioperatório resultante de um aumento da pressão intracraniana. Quando não identificado e tratado adequadamente, o edema pulmonar neurogênico pode levar à insuficiência cardiorrespiratória aguda, com hipoperfusão global e hipóxia. Portanto, a conscientização e o conhecimento de sua ocorrência, apresentação clínica e seu tratamento são essenciais.

Introduction

Pulmonary edema is caused by the accumulation of fluid within the air spaces and the interstitium of the lung. It may form due to intrinsic lung pathology or systemic dysfunction,[1] leading to impaired gas exchange and respiratory failure.

Neurogenic pulmonary edema (NPE) is a clinical syndrome characterized by the acute onset of pulmonary edema following a significant central nervous system (CNS) insult.[2] It usually appears within minutes to hours after the injury and has a high mortality rate if not recognized and treated appropriately.[3] A high index of suspicion is required for its diagnosis, which is based on the occurrence of the edema after a neurologic insult and the exclusion of other possible causes.[4]

The most common causes of NPE are subarachnoid hemorrhages, followed by head trauma, seizures, embolic stroke, neurologic endovascular procedures and increased intracranial pressure (ICP) of any etiology.[1,5] NPE may be a less-recognized consequence of raised ICP due to obstructive hydrocephalus by blocked ventricular shunts.[6] In this setting, a mechanical shunt malfunction should be treated urgently to prevent the neurologic sequels of ICP,[7] but the presence of preoperative NPE presents a dilemma to the neuroanesthesist due to the divergent goals of management of a raised intracranial pressure and pulmonary edema.[6]

We report a patient with acute obstructive hydrocephalus due to ventriculo-atrial shunt dysfunction who presented with NPE.

Case report

A 15-year-old female patient, with history of obesity, asthma, epilepsy, myelomeningocele sequelae and ventriculo-atrial shunt, was proposed to urgent surgery for placement of external ventricular drainage (EVD) to treat obstructive hydrocephalus due to shunt obstruction.

On admission, in the emergency department, she presented with vomit, headache and somnolence (Glasgow Coma Scale (GCS) of 14) and her CT scan showed a triventricular hydrocephalus (Fig. 1).

She was admitted to the emergency operating room (EOR) prostrate, showing progressive signs of respiratory distress (while with oxygen support by facemask with a FiO_2 of 80%) and with bilateral thick scattered crackles on pulmonary auscultation. She had a heart rate of 100 beats per minute, arterial pressure of 140/85 mm Hg, respiratory rate of 25 ventilations per minute and $SatO_2$ of 96%. Her chest X-ray showed bilateral haziness suggesting pulmonary edema (Fig. 2). The arterial blood gas parameters (ABG) were the following: pH 7.44; pCO_2 29 mm Hg; pO_2 86 mm Hg; Oxygen Saturation 97%; Lactates: 2.68 mmol L^{-1}. All other clinical and biochemical investigations were normal.

During pre-oxygenation, and with the supine position, the presence of pink foamy secretions was noticed.

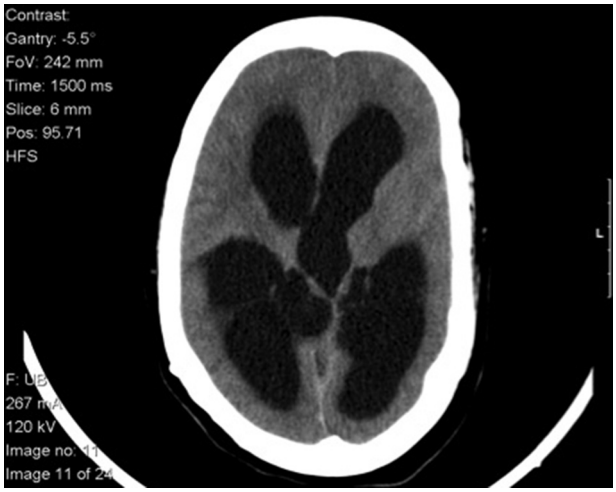

Figure 1 Admission CT scan showing triventricular hydrocephalus.