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The Risk of Acute Respiratory Distress Syndrome in Patients with Subarachnoid Hemorrhage, About A Case

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Abstract **Case Report**

Aneurysmal arachnoid hemorrhage (AAH) is a rare disease common in the young female population and can cause ma ny neurological complications, namely hydrocephalus, vasospasm and rebleeding. However, it can also lead to extra n eurological complications, including cardiovascular failure, renal failure, metabolic disorders (hypokalaemia, hypergly cemia, dysnatremia) and acute respiratory distress syndrome (ARDS). We report a case of a 50-year-old patient who d eveloped ARDS during her hospitalization in the resuscitation department for an AAH. The occurrence of this complic ation in these patients could be explained on one hand by an activation of the sympathetic nervous system hence the ap pointment of neurogenic pulmonary edema and on the other hand by the presence of a systemic inflammatory response syndrome (SIRS), but future studies will be needed to elucidate these pathophysiological mechanisms. According to r ecent studies, the treatment of ARDS is based on a mechanical ventilation strategy that uses low volumes with high PE EP, while prone position is a therapeutic alternative but with the risk of increased intracranial pressure. The manageme nt of aneurysmal arachnoid haemorrhage relies on the control of as well as other factors responsible for delayed ische mia and aneurysm treatment.

Keywords: Acute respiratory distress syndrome, subarachnoid hemorrhage, catecholaminergic stress, systemic inflamatory response syndrome, protective ventilation.

INTRODUCTION

Aneurysmal arachnoid haemorrhage (AAH) is a rare disease common in the young female population. It can cause many neurological complications, namely h ydrocephalus, vasospasm and rebleeding. However, it c an also lead to extra neurological complications, includi ng cardiovascular failure, renal failure, metabolic disord ers (hypokalemia, hyperglycemia, dysnatremia) and acu te respiratory distress syndrome (ARDS) [1]. We report a case of a 50-year-old patient who developed ARDS d uring her hospitalization resuscitation service for aneury smal Arachnoid hemorrhage.

CLINICAL OBSERVATION

A 50 years old female patient, followed for typ e 2 diabetes for 3 years under metformin, admitted to th e emergency department for intense headaches in thund erclap, vomiting and photophobia. The clinical examina tion at admission found a patient confused (GCS 14/15) , subfebrile (temperature at 37.9° C), hemodynamically stable (BP=130/70 mmHg, Cardiac frequency= 90 beats / min) and respiratory (16 cycles / min, SpO2 at 98% in ambient air), not exhibiting a sensorimotor deficit. The brain CT-scan performed revealed the presence of meni ngeal and intraventricular haemorrhage (Figure-1), the l umbar puncture brought a haemorrhagic liquid, incoagu lable, microscopically characterized by the presence of 14,400 red blood cells / mm3, of 3 white elements / mm 3 and without germs to direct examination, the initial ch est x-ray was normal, the electrocardiogram showed no abnormalities, troponin was slightly elevated (7 times th e normal value) and metabolic status showed hypokale mia at 3.05 mmol / l and hyponatremia at 130 mmol / l. The patient had been transferred to the intensive care un it, placed in a low-light unit, put under oxygen (3l / min), antiemetic to control vomiting, nimodipine (2 tablets every 6 hours by the nasogastric tube) to prevent vasosp asm, paracetamol or morphine to calm headaches. The maintenance of the hemodynamic stability was essential with the aim of a Systolic blood pressure between 160 a nd 180mmHg. The evolution was marked by the respira tory and infectious aggravation of the patient with a pol ypnea at 24 cycles / min, an SpO2 at 86% under oxygen (telescope), bilateral pulmonary opacities at chest x-ray, leukocytosis at 15,680 elements / mm3 and a CRP at 28

0 mg / l. PaO2 / FiO2 ratio was 150 mmHg. Cardiac ultrasound had eliminated cardiogenic hydrostatic edema. The diagnosis of moderate ARDS (Figure-2) and systemic inflammatory response syndrome (SIRS) was made. Protective ventilation was set up with low current volumes (6ml / kg) and high PEEPs combined with antibiotic therapy. The evolution was marked by the improvement of the patient after one week and its transfer to the neurosurgery department for additional support.

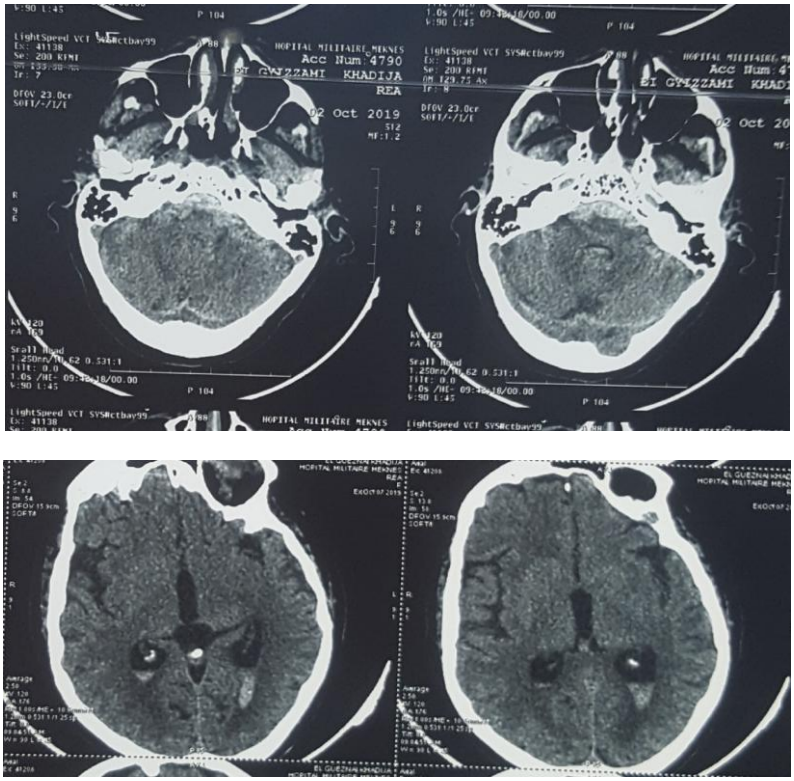

Fig-1: Brain scanner, subarachnoid and intraventricular hemorrhage

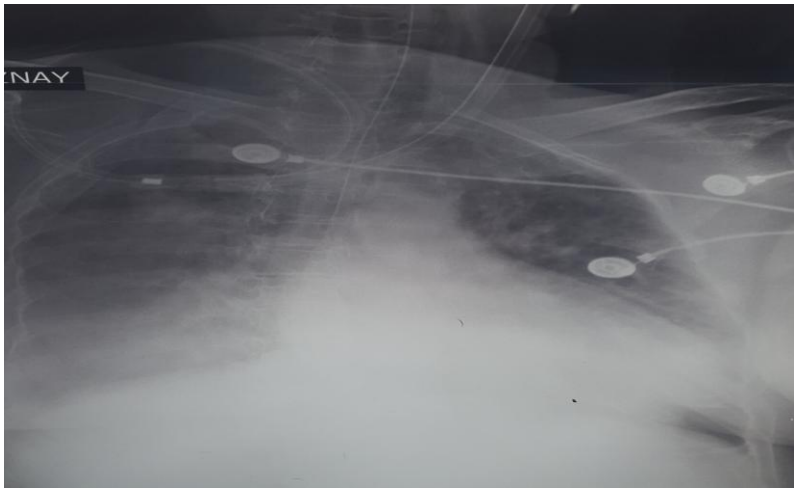

Fig-2: Pulmonary Radiography, bilateral pulmonary opacities

DISCUSSION

According to the new Berlin definition, ARDS is defined by the installation of respiratory symptoms for less than a week after the occurrence of a usual risk factor for ARDS if it is identified, the presence of bilateral pulmonary opacities radiography or chest CT scan, exclusion of left heart failure and hypoxemia with a PaO2 / FiO2 ratio of less than or equal to 300mmHg. There are 3 types of ARDS depending on the severity of this hypoxemia, mild when the PaO2 / FiO2 ratio is between 200 and 300 mmHg, moderate when it is between 100 and 200 mmHg and severe if it is below 100 mmHg. This is valid if the patient is under invasive, noninvasive ventilation or VS-PEP [2]. It is responsible for significant morbidity and mortality in patients with aneurysmal arachnoid hemorrhages. In fact, the mortality is of the order of 50%, and 60% of the survivors will have neurological sequelae [3]. It occurs in severe forms of The AAH (W

FNS > 3 and / or modified Fisher score> 2) [4]. The occ urrence of this complication in these patients could be e xplained on one hand by an activation of the sympatheti c nervous system hence the appointment of neurogenic pulmonary edema and on the other hand by the presenc e of a systemic inflammatory response syndrome (5).Th e massive release of catecholamines secondary to aneur ysmal rupture would lead to myocardial lesions objectif ied by the presence of electrical abnormalities in the ele ctrocardiogram and by an increase in cardiac enzymes i ncluding troponin and CPK mb [6], this catecholaminer gic discharge lasted on average seven at ten days with n ormalization at the 6th month [7]. The presence of SIRS in patients with AAH increases the risk of neurogenic p ulmonary edema and is of poor prognosis if present at a dmission [8]; it is manifested by a fever at admission, h yper leukocytosis and elevation of CRP [9]. In the after math of aneurysmal rupture, there is a sharp increase in systemic and pulmonary vascular resistance that causes ventricular compliance impairment and then hydrostatic edema [10]. Intracranial hyperpressure outbreaks, in cas e of aneurysmal arachnoid haemorrhage, transiently cau se the increase in intravascular pressure at the origin of alveolocapillary membrane damage explaining plasma l eakage in pulmonary interstitial tissue [11]. Several rece nt studies suggested that a mechanical ventilation strate gy using low volumes with high PEEPs could reduce m ortality in these patients [12]; ventral decubitus is a ther apeutic alternative but with a risk of increased intracrani al pressure [13]. A study conducted by the National Hea rt Lung (NHL) had shown a 22% reduction in the risk o f death in patients ventilated at 6ml / kg theoretical ideal weight compared to 12ml / kg [14], the management of the AAH relies on the control of Intracranial pressure as well as other factors responsible for delayed ischemia a nd treatment of the aneurysm.

CONCLUSION

The pathophysiological mechanisms that may explain the association of AAH with ARDS remain to b e elucidated, and future studies are needed. The strategy of mechanical ventilation based on low current volumes and high PEP would nevertheless improve the prognosi s, reduce mortality, and reduce the duration of mechanic al ventilation.

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