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Atelectasis as a Complication of Subarachnoid Hemorrhage: Case Report and Literature Review

Wlla Khaled Alabedalaziz[1*] and Hikmat Mohammed Hadoush[2]

[1]Jordan University of Science and Technology, Faculty of Applied Medical Sciences, Department of Rehabilitation Sciences, Irbid, Jordan

[2]Jordan University of Science and Technology, Faculty of Applied Medical Sciences, Department of Rehabilitation Sciences, Irbid, Jordan

[*]**Corresponding author:** Wlla Abedalaziz, King Abdullah University Hospital, Jordan University of Science and Technology, Tel: +962-781202055; E-mail: wala560@yahoo.com

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Abstract

Introduction: Treatment of atelectasis has improved dramatically in multiple population. These options of treatment and prevention had been used for hemorrhagic patient developed suddenly atelectasis, but all of them had failed. Therefore, the purpose of this study is to highlight the mechanism and etiology of atelectasis as a secondary complication of subarachnoid hemorrhage.

Case presentation: We report an infrequent case of atelectasis after subarachnoid hemorrhage in a 58-year old male patient. After he had get better, he had developed this respiratory complication then sadly passed away even with intensive care and therapy.

Conclusion: This case report tries to highlight the undiscovered mechanism of atelectasis in subarachnoid hemorrhage population, that is because this investigation playing a significant roll in treatment.

Keywords: Atelectasis; Subarachnoid hemorrhage

Introduction

Atelectasis is a pulmonary dysfunction related to collapsed alveoli in one or more lobe, or in the whole lung that leading to poor oxygenation, it subsequent of blockade of the airway that serves the dawned alveoli [1]. This condition is frequently seen in multiple clinical situations such as asthma, post cardiothoracic operation, pediatric with surfactant deficiency, patients with long bed rest and secondary other respiratory problem. In addition, it may develop because of factors like pain and diaphragmatic dysfunction [2].

The prevalence of complications of the respiratory system including atelectasis is difficult to determine, it ranges from 1% - 40% according to the population [3]. Generally, pulmonary dysfunction is a frequent complication in neurological conditions like subarachnoid hemorrhage [4] with a prevalence of 37.6% [5]. But the incidence of atelectasis after SAH concluded that is limited to a tiny fracture ≤ 1% [4] $^{(n=66)}$ [6] $^{(n=70)}$.

Multiple evidences reported the management and treatment modalities of atelectasis including mechanical ventilator with continuous positive airway pressure (CPAP), adjusted with low positive end-expiratory pressure (PEEP) of 5 cm H_2O, nebulizer of bronchodilators and bronchiolitis, chest physical therapy including positioning and suctioning of mucus and secretions, and pain control, and it rottenly resolved in 24-48 hrs [1,3,7].

Complications of atelectasis are serious; they may be fatal. Atelectasis found to impair the exchange of blood gases, pneumonia, or it may lead to ventilation/perfusion (V/Q) mismatching [8]. Cumulative evidence supposed that these conditions of closure alveoli can be a factor in the incidence of post-operation pulmonary complications and respiratory failure [9].

Case Presentation

A male patient (58-year-old, heavy smoker) with history of hypertension came to the emergency department in the King Abdullah University Hospital complaining of acute severe headache, where he was screaming madly, the headache has been described as the worst ever. Besides, his blood pressure was high 148/76, Glasgow Coma Scale (GCS) was 14/15, and able to move his limbs symmetrically, however he was confused, disoriented and suffering from a speech difficulty. After investigation, the computed tomography scan (CTS) showed a primary diagnosis of bilateral SAH with hydrocephalus. He was admitted to a neurosurgical intensive care unit (NICU) to conduct further investigations and interventions.

On the same day, he underwent surgery of external ventricular drainage (EVD) and put on SIMV mechanical ventilator with sedation. On the second day, four vessels angiography had been done. The report showed ruptured anterior communicating artery (ACOM) aneurysm, 60% stenosis of internal carotid artery (ICA), total occlusion of right ICA, and hypoblast of right anterior cerebral artery (ACA) and right posterior cerebral artery (PCA). Echocardiogram showed that the ejection fraction is 45. He complained of bradycardia (heart rate=40). On the third day he underwent coiling for left ACOM.

A few days later, the anesthesia was withdrawn and the mechanical ventilator eliminated, his vital sign became normal but somewhat chesty. The respiratory therapy has been ordered (suction and positioning as needed) also he had an effective cough. The physiotherapy started for mobilization and strengthening the four limbs because of slight weakness. He was fully conscious and responded to the command comprehensively.

The patient doing well until one month after the admission, on that day his temperature raised and the level of consciousness declined. After examination, the CTS showed vasospasm and there was meningitis, and a lab test showed that Acinetobacter baumanni had developed. The patient was given the necessary neurogenic medication and the antibiotic, his condition gets improved, GCS 8/15, and the inflammation was treated.

Suddenly it was noticed that the patient complained of shortness of breath, and the arterial blood gases (ABGs) were all abnormal. The radiological chest test showed atelectasis in left lower lobe so the respiratory therapist put the mechanical ventilator on continuous positive airway pressure (CPAP) mode with judicious parameters according to ABGs , the intensive chest physiotherapy ordered, and a twice-daily bronchodilator has given, he gets little better but after some days all blood gases were abnormal notwithstanding management of atelectasis, he still suffers from CO_2 retention, and elevation of PO_2, the mode of MV turn to BiPAP to get more control but still with no improvement. Then, sadly he passed away.

To note, the medication was used included paracetamol as needed, antihypertensive, antibiotics, Keppra, multivitamins, Somazina, Colistin, Combivent, and vasopressor. In addition, on death death day, a room air ABGs were performed and revealed: HCO_3 33, PCO_2 58mmHg, PO_2 184mmHg, while the normal value should be HCO_3 22-26, PCO_2 35-45mmHg, PO_2 80-100mmHg [10].

Discussion

To the best of our knowledge, this is the first case study for a patient complaining of atelectasis as a complication of SAH. Atelectasis is commonly seen in pediatric population like asthma [11], cardiothoracic population like cardiopulmonary bypass [12,13], and it developed by multiple mechanisms, like changing alveolar lining consequently alters the surface tension of the alveolus (deficit in surfactants) or compression on the parenchyma, so pathophysiology of atelectasis differs from one population to another [11,14].

The nature and the pathophysiology of atelectasis in SAH are not fully understood such as other populations. There were a contradict results about the correlation between atelectasis and vasospasm. In this case study, we highlight the unclear relationship between atelectasis and SAH, we explain it by a presence of a circuit that is still undiscovered, it may have affected by the vasospasm and led to the collapsed alveoli. A recent study conducted on rats to explore the mechanisms that responsible for atelectasis after SAH [15]. It reported that the deflation of alveoli in SAH population associated with proteins of lung-specific surfactant deficiency. However, more translational research is needed to prove this mechanism, in order to investigate more effective treatment.

A study previously reported that CPAP was effective for treating atelectasis induced by surfactant deficiency [16], however this case study demonstrates otherwise. As well, PEEP was confirmed to be effective in management of atelectasis [17], but then again this case study showed that PEEP was not effective. This patient has received evidenced-based treatments and modalities in intensive way and he had a cough reflex that is essential to prevent atelectasis, but all of them were not enough to protect the lung of this patient. Therefore, further investigations and translational studies about the mechanism of atelectasis in SAH population are needed in order to maximize the treatment choices.

Conclusion

Atelectasis was the point of transformation in the patient's life, and he is responsible for the deterioration of his condition. There is a lack of understanding why SAH patient did not respond to the evidenced treatment modalities, although other population responded to them, perhaps the reason is that each population and mechanism has a different treatment method.

Abbreviations: SAH: subarachnoid hemorrhage; ABGs: arterial blood gases; PEEP: positive end expiratory pressure; CPAP: continuous positive airway pressure; BiPAP: biphasic positive airway pressure; CTS: computed tomography scan; NICU: neurosurgery intensive care unit; GCS: Glasgow coma scale; MV: mechanical ventilator; ACOM: anterior communicating artery ICA: internal carotid artery; ACA: anterior cerebral artery; PCA: posterior cerebral artery

Declaration

Ethical approval: The case report entitled "Atelectasis as a complication of subarachnoid hemorrhage: case report and literature review." Received approval.

Consent for publication: Written informed consent was obtained from the patient's family for publication of this case report and any accompanying images.

Availability of data and material: Data sharing isn't applicable because no datasets were generated or analyzed for this study. Competing interests: "The authors declare that they have no competing interests."

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