

## Case Report

# Relieving Intracranial Pressure Cured Hypoxemia-where does the Mystery Lie?

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## Abstract

Respiratory malfunction is associated with a variety of intracranial abnormalities. Neurogenic ventilation-perfusion mismatch due to intrapulmonary shunting is a less described phenomenon. Here we report a case of acute or chronic subdural hematoma complicated by acute preoperative arterial hypoxemia due to neurogenic intrapulmonary shunt (NIS). Hypoxemia subsided with surgical drainage of the subdural blood. One hypothesis for the mechanism of this phenomenon is that a disturbance is created in nervous system control of perfusion-ventilation relationships of the lung. Such a mismatch can be diagnosed using Giola's diagnostic criteria. Quick and definitive relieving of intracranial pressure is the formula to treat NIS.

## Keywords

- Raised intracranial pressure
- Neurogenic intrapulmonary shunt

## INTRODUCTION

Respiratory malfunction is associated with a variety of intracranial abnormalities. Precipitous drops in arterial SpO<sub>2</sub> may worsen the degree of brain injury and affect the degree of recovery and ultimate outcome. Neurogenic pulmonary edema associated with arterial hypoxemia is a well known complication of a variety of neurological conditions. But neurogenic ventilation-perfusion mismatch due to intrapulmonary shunting is a less described phenomenon. Here we report a case of acute or chronic subdural hematoma complicated by acute perioperative arterial hypoxemia in the absence of neurogenic pulmonary edema. Hypoxemia subsided with surgical drainage of the subdural blood.

## CASE REPORT

A 74-year-old gentleman admitted with us, suffered from right fronto parietal acute subdural hematoma (Figure 1) with a weakness of his left upper and lower extremities. His Glasgow coma scale (GCS) was 9/15. Patient's history included emergency Bentall procedure for ascending aortic dissection aneurysm 5 days ago. His trachea was extubated on the second postoperative day. Post extubation room air oxygen saturation was 100%. But he de saturated to <92% on room air with the onset of weakness on day 5. His SpO<sub>2</sub> was maintained at 96 to 97% with 6 liters of oxygen supplementation via face mask. His Chest X ray was unremarkable and transthoracic ECHO was essentially normal with an ejection fraction of 55%. Among blood investigations, platelets were 72000 and INR was 1.12. This patient was posted for emergent burr hole evacuation of subdural hematoma under general anesthesia. In the operating room, with standard

monitoring, the patient was pre oxygenated adequately and general anesthesia was induced and his trachea was intubated. Invasive monitoring included a radial arterial line. After tracheal intubation, SpO<sub>2</sub> dropped gradually over 5 minutes to 88% with FiO<sub>2</sub> of 50%. To achieve SpO<sub>2</sub> of 94%, FiO<sub>2</sub> was increased to 100% with a positive end expiratory pressure of 7 cm H<sub>2</sub>O. An arterial blood gas analysis showed a PaO<sub>2</sub> of 142mmHg. Auscultation of the chest was normal bilaterally. There were



Figure 1 Right Fronto Parietal subdural hematoma (Arrows).

no added sounds. Lung ultrasonography showed normal line artifacts without any pathology. Scalp incision was made and 2 burr holes were drilled on the right fronto-parietal region. Patient received one unit of aphaeresis' platelets in the intraoperative period. Subdural hematoma was drained with a cruciate incision on the dura. As the hematoma was being cleared, the SaO<sub>2</sub> started climbing to 100 % and we could gradually decrease FiO<sub>2</sub> to 50% over 30 minutes. Postoperatively, trachea was extubated when the patient was obeying and breathing well. He was off oxygen supplementation two hours postoperatively. His power on the left side improved and CT scan showed clearance of hematoma.

## DISCUSSION

Arterial hypoxemia may result from several mechanisms: inadequate alveolar ventilation, alveolar capillary diffusion abnormalities, or ventilation perfusion mismatch [1]. Inadequate alveolar ventilation is unlikely in our patient since he received controlled ventilation with FiO<sub>2</sub> 100% and PEEPS of 7 cm of H<sub>2</sub>O without any improvements in SpO<sub>2</sub>. Development of a diffusion abnormality in hours and instantaneous recovery within minutes is an unlikely explanation. Ventilation-perfusion mismatch due to neurogenic intrapulmonary shunting (NIS) in animals with brain trauma with and without raised ICP has been described [2]. There are also reports of a number of patients with increased intracranial pressure after isolated head trauma immediately exhibiting arterial hypoxia due to venous admixture in lungs [3]. To the best of our knowledge, this is the first case reported of NIS in a non brain trauma patient. The mechanism for this phenomenon is obscure, but one hypothesis is that a disturbance is created in nervous system control of perfusion-ventilation relationships of the lung. Maxwell et al. [2], calculated from their animal experiments that increased venous admixture is a significant factor in the production of arterial hypoxemia during high intracranial pressure. Venous admixture may be due to three factors: a) inhomogeneous distribution of ventilation, i.e., perfusion of poorly ventilated alveoli; b) perfusion of non ventilated alveoli; and c) opening of anatomic arteriovenous anastomoses or redistribution of flow through preferential channels. The development of intrapulmonary shunts with increased cardiac output during exercise in healthy humans has been reported in several recent studies. Similarly increased intracranial pressure produces reversible increases in venous

admixture closely associated with increases in cardiac output [4]. These findings suggest that mechanism of venous admixture secondary to increased intracranial pressure may be reversible distension of portions of the pulmonary vascular bed. Vascular distension may compromise alveolar ventilation or cause relative over perfusion of lung areas with resultant increases in venous admixture. This mismatching of the distribution of ventilation and perfusion was confirmed using the multiple inert gas elimination technique in patients with an increased shunt fraction. As per Giola et al. [5], patients were considered to have Neurogenic intrapulmonary shunt (NIS) if the following criteria were met: 1) absence of chest or abdominal trauma; 2) normal clinical and radiologic examination of the chest; 3) normal total respirator compliance; 4) normal airway secretions as judged by gross appearance and bacteriologic exam and 5) the PaO<sub>2</sub>/FiO<sub>2</sub> ratio is persistently <300. Our patient met all the above criteria except one. The patient had a recent thoracotomy. Hence presence of lung atelectasis may be a possibility. But no improvement in oxygen saturation in spite of controlled ventilation with PEEP rules out atelectasis as sole contributor of hypoxia. To conclude, Neurogenic intrapulmonary shunt (NIS) is commonly accompanied by CNS dysfunction and increased ICP. And also high FiO<sub>2</sub> and PEEP fail to alleviate the degree of intrapulmonary right to left shunting in NIS. Quick and definitive relieving of intracranial pressure is the formula to treat NIS.

## REFERENCES

1. Karcz M, Papadakos PJ. Respiratory complications in the postanesthesia care unit: A review of pathophysiological mechanisms. *Can J Respir Ther*. 2013 winter; 49: 21-29.
2. Maxwell JA, Goodwin JW. Neurogenic pulmonary shunting. *J Trauma*. 1973; 13: 368-373.
3. Schumacker PT, Rhodes GR, Newell JC, Dutton RE, Shah DM, Scovill WA, et al. Ventilation-perfusion imbalance after head trauma. *Am Rev Respir Dis*. 1979; 119: 33-43.
4. Eldridge MW, Dempsey JA, Haverkamp HC, Lovering AT, Hokanson JS. Exercise-induced intrapulmonary arteriovenous shunting in healthy humans. *J Appl Physiol*. 2004: 797-805.
5. Giola, Frank R, Stidham, Gregory L, Rogers, Mark C. Neurogenic intrapulmonary shunting without intracranial hypertension or pulmonary edema in head trauma patients. *Critical Care Medicine*. 1980; 8: 254.

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