





CONGENITAL PULMONARY ARTERIOVENOUS MALFORMATION DIAGNOSED IN TRAUMA

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INTRODUCTION

Congenital pulmonary arteriovenous malformations is a rare vascular anomaly involving fistulous communications between the pulmonary artery and pulmonary vein. They are typically incidental findings on imaging studies. Its context in trauma is rare and challenging.

CASE REPORT

A 32 years old lady had a low-impact accident where she fell from a stationary motorcycle. She is a vape user with a BMI of 38. Upon presentation, she had no respiratory symptoms, spoke in full sentences but had peripheral cyanosis. Examination shows an oxygen saturation of 92% on high flow mask and clear polycythaemia She had lungs. with haemoglobin of 18g/dL and haematocrit of 57%. Chest radiograph was suspicious for lung contusion.

Blood gas shows persistent Type 1 respiratory failure with a pH 7.35, pO2 55, pCO2 35, SaO2 84 and HCO3 19 despite CPAP. She was intubated two days after trauma due to severe hypoxaemia. Serial chest radiographs did not show worsening haziness.

A cardiac echogram ruled out cardiac contusion but reveals a mild pulmonary hypertension. Difficulty in achieving oxygen saturation (SpO2 of 50-75%) despite lung recruitment strategies resulted delaying a diagnostic CT to day eleven post trauma. CT revealed right middle lobe arteriovenous malformations with bilateral ground glass opacities.

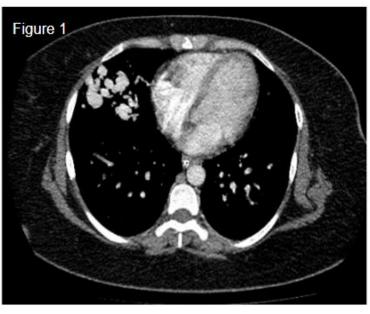
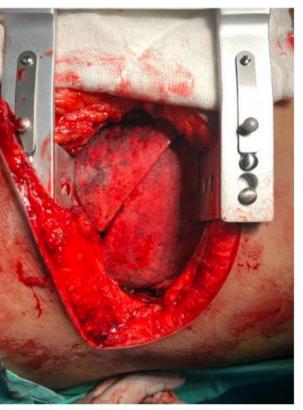




Figure 1 and 2: CT showing right middle lobe arteriovenous malformations

She underwent an open right thoracotomy and middle lobectomy. Intraoperative findings revealed large dark bluish lakes of AVM occupying 50% of the middle lobe. There were two dilated feeding pulmonary arteries and two

pulmonary veins. Her oxygen saturation preoperatively was 51% and immediately post lobectomy was 91%. She eventually succumbed despite intensive post operative care.



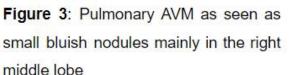




Figure 4: Cross section of the middle lobe of right lung showing dilated pulmonary arteries and veins

DISCUSSION

The fundamental defect of PAVM comes from right-to-left shunting. Patient's peripheral cyanosis and polycythemia indicated that shunting is likely more than 20% of her cardiac output.

Her wide paCO2 and ETCO2 difference indicated a ventilation - perfusion mismatch. Multiple differential diagnoses were considered for her respiratory failure such as pulmonary hemorrhage, ARDS, pulmonary embolism, cyanotic heart disease and haemoglobinopathy.

Embolotherapy was not available at our center. The perioperative mortality for pulmonary lobectomy is about 3% but it is expected to be higher in our patient due to her smoking status, obesity and confounding lung insults such as pneumonia, ARDS and pulmonary oedema.

CONCLUSION

This case report highlights the challenges associated with diagnosing and managing congenital PAVM in trauma patients.

REFERENCE

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