Clinical Reports

Pulmonary arterio-venous fistula: mechanical ventilation and hypoxemia

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Pulmonary arteriovenous malformation (PAVM) is an uncommon congenital anomaly. As PAVM is a direct communication between branches of the pulmonary artery and vein, major disturbances in gas exchange can result. We present a patient with an unsuspected PAVM who came to our institution for drainage of a brain abscess. Arterial blood gas analysis during and after surgery demonstrated a large alveolar-arterial gradient for oxygen in the absence of any obvious pulmonary pathology while the patient was receiving positive pressure ventilation (PPV). Oxygenation improved considerably upon resumption of spontaneous ventilation. A diagnosis of PAVM was made subsequently. We conclude that positive pressure ventilation can worsen right to left shunting in patients with PAVM.

Une malformation artérioveineuse pulmonaire (MAVP) est une anomalie congénitale inhabituelle. Comme elle consiste en une communication directe entre des branches de l'artère et de la veine pulmonaires, il peut en résulter des désordres majeurs dans les échanges gazeux. Nous présentons un patient avec un PAVM méconnu qui s'est présenté dans notre institution pour un drainage d'abcès cérébral. L'analyse de gaz artériels pendant et après la chirurgie démontrait un gradient alvéolo-artériel d'oxygène élevé en l'absence de toute pathologie pulmonaire manifeste, alors que le patient était sous ventilation à pression

positive (PPV). L'oxygénation s'est considérablement améliorée au retour en ventilation spontanée. Un diagnostic de MAVP fut posé ultérieurement. Nous concluons que la ventilation à pression positive peut aggraver un shunt droit-gauche chez les patients avec MAVP.

Case report

A 39-yr-old 82-kg man was hospitalized following an episode of non-generalized seizure activity. Before admission the patient complained of headaches, but denied photophobia, diplopia, neck stiffness, sensory or motor losses, ataxia, nausea, vomiting or fever. A dental procedure had been performed nine days before admission. The past medical history revealed intermittent episodes of sinusitis. No history of pulmonary or cardiac symptoms was reported. Physical examination demonstrated a low-grade temperature and a grade 2/4 systolic murmur heard at the left sternal border. There was no clubbing, cyanosis or telangiectasia of the skin or mucous membranes. Mental status was normal and there were no motor, sensory, cranial nerve or cerebellar abnormalities. The white blood cell count was $16,000 \text{ mm}^{-3}$ without a left shift. Pre-operative chest radiograph and 2D echocardiogram were normal. A contrast-computed tomographic brain scan revealed a right frontal mass and cerebral arteriography indicated an avascular lesion. Preoperative arterial blood gas analysis was not performed.

Eight days after admission a right frontal brain abscess was excised and drained. Anaesthesia was induced with thiopentone 400 mg, metocurine 10 mg, pancuronium 5 mg, and lidocaine 100 mg were given to facilitate oral tracheal intubation.

Anaesthesia was maintained with nitrous oxide, isoflurane and sufentanil ($200 \mu\text{g iv}$). Ventilation was controlled with an FiO_2 of 0.50, rate of 12, zero positive end-expiratory pressure (PEEP) and a tidal volume of 800 ml. Peak inspiratory pressure was 24 cm H_2O . Intraoperative arterial blood gas analysis revealed pH 7.46–51, PaO_2

Key words

HYPOXIA:

LUNG: bloodflow, shunting, vasculature.

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TABLE Relationship between lung ventilation and oxygenation

	OR	PARR	ICU × 2 hr	ICU × 12 hr	ICU × 14 hr	ICU × 15 hr
FiO ₂	0.5	0.6	0.6	0.5	0.6	0.5
PEEP (cm)	0	5	5	5	0	0
PaO ₂ (mmHg)	82	102	135	66	198	154
A-a DO ₂	232	279	251	252	186	157
Ventilation	MV*	MV	MV	MV	T-piece	Face mask

*Mechanical ventilation.

70–90 mmHg, PCO₂ 31–38 mmHg, and oxygen saturation was 95% or greater by pulse oximetry. Breath sounds were clear and equal to auscultation throughout the operation.

The patient was moved to the recovery room continuing to receive mechanical ventilation. In the recovery room and subsequently in the ICU, the increased alveolar-arterial (A-a) gradient persisted (Table) and PEEP (5 cm H₂O) was begun and continued for 12 hr. The patient remained afebrile without evidence of respiratory distress or increase in sputum production. Arterial oxygenation remained poor until positive pressure ventilation was discontinued.

During spontaneous breathing via T-piece (FiO₂.60) and subsequently by face mask, the A-a gradient decreased markedly. Reevaluation of the preoperative chest radiograph revealed a linear density in the left midlung field suggesting a PAVM. There was no evidence of atelectasis, effusion, pulmonary oedema or pneumonitis on subsequent radiography. Pulmonary arteriography confirmed the presence of a large solitary PAVM in the left lung (Figure).

The patient was transferred from the ICU on post-operative day two, neurologically intact with arterial blood gases as shown in the Table. Eight days later he was discharged in good condition to receive antibiotic therapy at home. He returned three weeks later and underwent excision of his PAVM. This procedure was uncomplicated.

Discussion

We describe a case in which arterial oxygen desaturation was induced by positive pressure ventilation in a patient with an undiagnosed pulmonary arteriovenous fistula. To our knowledge, there have been no reports implicating positive pressure ventilation as a cause of worsening hypoxemia in patients with PAVM.

Pulmonary arteriovenous malformation is a rare condition that is most often congenital in origin, although acquired cases have been reported.¹ It exists as a spectrum of abnormality ranging from multiple microscopic anastomoses to large, solitary communications.² Although information about the origin of these lesions is limited, there is a strong association between PAVM and hereditary telangiectasia.¹ Most patients are asymptomatic but

FIGURE The arrows indicate free flow of contrast dye in a large left PAVM on pulmonary arteriogram.

the presence of exertional dyspnea, cyanosis and clubbing may trigger a diagnostic search. The chest x-ray is abnormal in approximately 98% of patients and is the most common clue to the anomaly.³ A peripheral density connected to the hilum by vascular markings is the most characteristic finding.² Our patient had a systolic murmur which is a common but nonspecific finding. Pulmonary angiography is required to make a definitive diagnosis and plan for therapy. Our patient presented with a brain

abscess. Although rare, the association of neurological manifestations with PAVM has been documented.⁴⁻⁸

There was a clear association between removal of PPV and improvement in oxygenation in our patient. Also, PEEP may have played a role as there was an increase in A-a gradient when PEEP was applied after surgery. The most likely cause for the decreased arterial oxygen saturation observed with PPV was increased right to left shunting induced by increased pulmonary vascular resistance (PVR). Alternatively, arterial desaturation could have been due to mixed venous desaturation induced by PPV mediated decreases in cardiac output. It is well known that PPV and/or PEEP can decrease cardiac output.⁹ We did not measure mixed venous oxygen saturation or cardiac output in our patient. Thus, the mechanism responsible for the ventilation-related changes in oxygenation is speculative. However, there was no evidence to indicate a large decrease in cardiac output occurring during mechanical ventilation. Hypoxaemia due to right to left shunting has been shown to occur in a patient with PAVM and obstructive lung disease, a situation in which elevated PVR is probably present.¹⁰ Lung volumes influence PVR: lung volumes above or below normal functional residual capacity (FRC) will increase PVR.^{11,12} At lung volumes above FRC, compression of small intraalveolar vessels occurs; at lung volumes below FRC, increased PVR is due to compression of large extraalveolar vessels.¹³ Therefore, PPV may increase PVR.¹⁴

While the tidal volume which we used, 10 ml · kg⁻¹, seemed appropriate, it may have inflated the lungs above normal FRC and increased PVR. It is unlikely that this tidal volume led to an FRC that was below normal. There was a further reduction in the A-a gradient after extubation of the trachea. This is consistent with the concept that optimal FRC (and PVR) occurs in the presence of a normal glottic closure mechanism.¹⁵

The potentially deleterious effects of positive pressure ventilation and PEEP in patients with intracardiac shunts have been well described.^{16,17} We report a case in which PPV induced a marked decrease in arterial oxygenation in a patient with a non-cardiac right to left shunt, PAVM. When hypoxaemia has been induced by PPV in a patient with a known or suspected PAVM, consideration should be given to cease mechanical ventilation as soon as possible.

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