



CASE REPORT

Body position and oxygenation: An intriguing relationship



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Abstract Dyspnea and hypoxemia are among the most common symptoms and signs that need to be assessed in clinical practice.

This case illustrates how simple steps in history taking and physical examination can be crucial for diagnosis.

We present a patient with intermittent hypoxemia, initially attributed to a pulmonary infection. However, the hypoxemia persisted even after successful treatment of the infection. Computed tomography angiography of the chest and ventilation/perfusion lung scan excluded pulmonary embolism.

We then observed that the hypoxemia and dyspnea were triggered by orthostatism. An echocardiogram with a bubble test showed a patent foramen ovale, with a right-to-left shunt, without pulmonary hypertension. After percutaneous closure of the foramen ovale, the symptoms completely resolved.

This is a case of platypnea-orthodeoxia syndrome, which is usually associated with patent foramen ovale or atrial septal defect and is typically observed in the elderly. The features and causes of this curious syndrome are discussed.

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PALAVRAS-CHAVE

Platipneia;
Ortodeoxia;
Foramen ovale
patente;

Posição corporal e oxigenação: uma relação intrigante

Resumo A dispneia e hipoxemia são dos sintomas e sinais mais comuns que avaliamos diariamente na prática clínica.

Este caso ilustra como uma avaliação simples da anamnese e exame físico podem ser cruciais para o diagnóstico.

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Comunicação interauricular

Apresentamos um doente com hipoxemia intermitente, inicialmente atribuída a uma infeção respiratória. Contudo, a hipoxemia manteve-se apesar de tratamento eficaz da infeção. A angiotomografia computadorizada do tórax e cintigrafia de ventilação-perfusão excluíram tromboembolismo pulmonar.

Verificámos posteriormente que a hipoxemia e dispneia eram despoletadas pelo ortostatismo. Um ecocardiograma com administração de soro agitado revelou a presença de foramen ovale patente, com fluxo espontâneo direito-esquerdo, sem hipertensão pulmonar. Após encerramento do forâmen ovale patente, os sintomas desapareceram.

Este é um caso de síndrome de platipneia-ortodeoxia, que geralmente se associa a foramen ovale patente ou comunicação interauricular, e é especialmente frequente em idosos. Discutimos as características e causas desta interessante síndrome.

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Case report

We describe the case of an 83-year old man, independent, with a history of hypertension and multiple lacunar strokes in the past.

The patient was admitted after a syncopal episode; standing in the bathroom, he felt nauseated and lightheaded before losing consciousness.

On admission to the emergency department, he felt dyspneic. He denied chest pain, cough, fever, palpitations, orthopnea or other symptoms.

He had some ronchi in the right lung, fever, type 1 respiratory failure and slightly increased C-reactive protein levels. The chest X-ray revealed a discrete opacity in the right inferior lobe. He began treatment for a respiratory infection, resulting in sustained apyrexia and normalization of laboratory parameters. However, the hypoxia persisted, appearing to follow an intermittent pattern. We began to notice that his blood gases and need for oxygen changed almost every day, with no evident pattern, even when the infection was completely cured.

He had no history of coronary disease or arrhythmia, previous dyspnea, orthopnea, chest pain, edema or other signs of heart failure, and a recent echocardiogram showed no anomalies. He had no background of smoking or pulmonary disease.

A thoracic computed tomography angiogram was ordered, which was normal. Because the possibility of a chronic peripheral pulmonary embolism remained, a ventilation/perfusion scan was performed, which also showed no changes.

There was no apparent deterioration on exertion and when low oxygen saturation was recorded the patient's breathing pattern was normal. His pulse oximetry and blood gases were finally measured in different postural positions and it was found that the hypoxia occurred when he was sitting or standing, and did not resolve completely with oxygen (Table 1). Furthermore, he also complained of dyspnea on orthostatism.

The patient was then diagnosed with platypnea-orthodeoxia syndrome (POS) and we set out to clarify its etiology.

This syndrome is caused by a right-to-left shunt that can be due to an intracardiac shunt through a patent foramen

Table 1 Blood gas analysis in different postural positions.

	Supine	Orthostatism
pH	7.392	7.402
pO ₂ (mmHg)	78	56
pCO ₂ (mmHg)	41	38
HCO ₃ ⁻ (mmHg)	23	23
O ₂ saturation (%)	95	80

HCO₃⁻: bicarbonate; O₂: oxygen; pO₂: partial pressure of oxygen; pCO₂: partial pressure of carbon dioxide.

ovale (PFO), atrial septal defect (ASD) or fenestrated atrial septal aneurysm (ASA), a pulmonary arteriovenous shunt, or pulmonary ventilation/perfusion mismatch.

Thromboembolic disease or arteriovenous malformations had been excluded, as had parenchymal lung disease. There were no abnormal liver function tests or lesions on abdominal ultrasound, so hepatopulmonary syndrome seemed unlikely. A transthoracic echocardiogram and bubble test, with administration of agitated saline through a peripheral vein, was therefore requested. The passage of microbubbles into the left atrium within the first three beats after right chamber opacification is considered diagnostic of PFO; the appearance of microbubbles following the third beat usually corresponds to intrapulmonary shunting.

The echocardiogram showed normal morphology of the cardiac chambers, an aneurysm of the interatrial septum with to-and-fro flow, preserved biventricular systolic function and dilatation of the ascending aorta, without pulmonary hypertension. When the agitated saline was administered, there was an immediate flow into the left atrium.

The transesophageal echocardiogram confirmed a right-to-left shunt and a PFO (Figure 1) with no other changes except for the ectatic aorta. Since the patient was clearly symptomatic and with functional limitations, we proposed percutaneous closure of the atrial septal defect, with which he agreed.

Before the PFO was closed, right-sided catheterization confirmed that the right chamber pressures were not increased. A percutaneous closure device (25-mm Amplatzer) was then implanted, guided by transesophageal echocardiography (Figure 2).

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