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Case report

Flow phenomenon, a differential diagnosis in refractory hypoxaemia in patients with anterior mediastinal mass. Case report☆

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ABSTRACT

Introduction: Refractory hypoxaemia is a critical, life-threatening condition if not resolved promptly. The flow phenomenon is implicated in its development.
Objectives: Clinical case presentation and non-systematic review of the literature on refractory hypoxaemia and flow phenomenon. Incidence, aetiology and pathophysiology are described.
Materials and methods: Clinical case presentation authorised by the Ethics Committee of our institution of a young patient who presented with progressive dyspnoea, reaching functional class IV/IV. A search of the literature was conducted in Pub Med, Scielo and Bireme.
Results: There is a growing interest in the physiological flow phenomenon leading to the development of refractory hypoxaemia in the absence of increased pressure in the cardiac cavities. Few reports are found.
Conclusions: Flow phenomenon-related refractory hypoxaemia must be suspected as an exclusion diagnosis in patients with a mediastinal mass. Prone ventilation is proposed as a bridging therapy in order to revert a life-threatening condition.

Fenómeno de flujo, diagnóstico diferencial de la hipoxemia refractaria en pacientes con masa mediastinal anterior. Reporte de caso

Palabras clave:
Foramen ovale, permeable

RESUMEN

Introducción: La hipoxemia refractaria es una condición crítica potencialmente mortal si no se corrige rápidamente. El fenómeno de flujo está implicado en su desarrollo.

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Disnea
Neoplasias del mediastino
Anoxia
Manejo de la vía aérea

Objetivos: Presentación de un caso clínico y revisión no sistemática de la literatura sobre hipoxemia refractaria y el fenómeno de flujo, describimos su incidencia, etiología y fisiopatología.
Material y métodos: Con autorización del comité de Ética de nuestra institución, presentamos un caso clínico de un paciente joven quien consulto por disnea progresiva hasta clase funcional IV/IV. La búsqueda bibliográfica se realizó en Pub Med, Scielo y Bireme.
Resultados: Se ha despertado un creciente interés en el fenómeno fisiológico de flujo que conlleva al desarrollo de hipoxemia refractaria en ausencia de aumento de presión en las cavidades cardiacas. Se encuentran escasos reportes.
Conclusiones: La hipoxemia refractaria por el fenómeno de flujo debe ser sospechada como diagnóstico de exclusión en pacientes con masa mediastinal. Proponemos la ventilación en prono como terapia puente para revertir una condición potencialmente mortal.

Introduction

Hypoxaemia is characterised by a partial oxygen pressure in arterial blood lower than 60 mm Hg. Under normal conditions, the circulatory and respiratory systems work together to keep it within normal ranges. The absence of homeostasis in those systems to ensure normal oxygen content in arterial blood is known as hypoxaemic respiratory failure and is explained on the basis of five pathophysiological mechanisms, namely: inspired oxygen pressure reduction, development of alveolar hypoventilation, right-to-left shunting, alveolar-capillary exchange membrane diffusion abnormalities, and dead space development. Prompt identification of its aetiology is the best strategy for instituting effective treatment.[1–4]

Case description

A 23-year old male patient, construction worker of mestizo ethnic origin considered otherwise previously healthy who presented with progressive dyspnoea that had lasted for 30 days, oppressive left chest pain not exacerbated by exercise and weight loss of approximately 2 kg over the previous three months. On physical examination, the patient was tachycardic but haemodynamically stable, with evidence of pericardial rub, hepatomegaly, splenomegaly and jugular engorgement grade II. The chest X-ray made in the emergency service revealed a very enlarged cardio-mediastinal silhouette. Echocardiography showed pericardial effusion with normal biventricular function, undilated cavities, no signs of tamponade, and intact septum. A suspected anterior mediastinal mass prompted an axial chest computed tomography scan which confirmed the presence of a 15 cm × 11 cm mass (see Fig. 1). Pericardiocentesis was performed with removal of 750 cc of blood, and an ultrasound-guided biopsy was performed. Later, the patient went into rapid clinical decline due to respiratory distress and hypoxaemia, leading to the suspicion of pneumothorax. Clinical confirmation was obtained from the general surgeon, and left thoracostomy was performed. The patient was then transferred to the intensive care unit (ICU) for haemodynamic monitoring. After initial stability, the patient exhibited signs of respiratory distress, hypoxaemia and fever. The decision was made to initiate invasive ventilation support, awake intubation due to the risk of airway collapse from extrinsic compression, blood gases control in acid–base balance with moderate/severe oxygenation disorder. The use of vasopressors was also initiated. Tables 1 and 2 show the haemodynamic profile, blood gases and laboratory results, respectively.

The ventilation mode used was controlled-assisted with 50% fraction of inspired oxygen and a positive end-expiratory pressure (PEEP) of 8. There was initial improvement with a saturation greater than 90%. One hour later the patient went again into progressive hypoxaemia with no improvement despite FiO_2 titration at 100%. Blood gases showed a pH of 7.31, PaO_2 of 21 mm Hg, $PaCO_2$ of 71 mm Hg and base excess of less than 5, with a PaO_2/FiO_2 ratio of 23. On chest X-ray there was partial atelectasis of the upper lobe that did not explain the current picture. Lung recruitment with PEEP titration was tried with no improvement. A suspected acute pulmonary embolism prompted performance of an angio-CT which was not available at the time, but given the urgency, a pulmonary angiography was performed instead, and was normal. Bacteraemia was suspected considering the presence of fever and clinical decline, and antibiotic therapy was initiated.

In view of refractory hypoxaemia, and having tried all the ventilation resources described, with adequate sedation and thinking about a mechanical cause, the patient was put on prone ventilation. This resulted in almost immediate recovery of oxygenation, with the following blood gases: pH 7.29, PaO_2 205 mm Hg, $PaCO_2$ 42 mm Hg and excess base of – 6.9 with a PaO_2/FiO_2 ratio of 205. Severe hypoxaemia was estimated to have lasted 2 h. Later, hyperkalemia, hypercalcemia and hyperphosphatemia were documented, constituting a tumour lysis syndrome with progressive shock that did not respond to management, resulting in death due to multiple organ dysfunction. The final pathology results confirmed the presence of an adenocarcinoma of the thymus (see Fig. 2).

Discussion

Life-threatening severe hypoxaemia requires prompt differential diagnosis in order to institute effective, live-saving treatment.[1,2] If pathophysiological causes are considered at