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CASE REPORT

Case report

Bronchospasm triggered by spinal anaesthesia. Case report and review of the literature☆

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ABSTRACT

Bronchospasm is a clinical condition that can occur unexpectedly during general anaesthesia, but is extremely rare after spinal anaesthesia. The following is a case presentation of a patient who developed bronchospasm after undergoing spinal anaesthesia not attributable to other causes, and that adds another case to the limited literature. Most publications allude to asthmatic patients, and this is probably the first description about a patient with emphysema-type COPD. Our case shows that although spinal anaesthesia is considered safe for patients with respiratory disease, specifically in asthmatic patients there is a possibility of bronchospasm in susceptible patients.

Broncoespasmo desencadenado por anestesia espinal. Informe de caso y revisión de la literatura

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RESUMEN

El broncoespasmo es una condición clínica que puede aparecer inesperadamente durante la anestesia general, pero es extremadamente rara tras la anestesia espinal. Presentamos un paciente que desarrolló broncoespasmo tras ser sometido a anestesia espinal, no atribuible a otras causas y que añade un caso más a la escasa literatura al respecto. La mayoría de las publicaciones se refieren a pacientes asmáticos, y esta sea probablemente la primera descripción en un paciente con EPOC tipo enfisematoso. Nuestro caso muestra que aunque

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la anestesia espinal se considere más segura para pacientes con patología respiratoria, en concreto en pacientes asmáticos, existe la posibilidad de que ésta produzca broncoespasmo en pacientes susceptibles.

Introduction

Bronchospasm following spinal anaesthesia is very rare and little known to practitioners. However, it is a potential occurrence considering a few published cases, and it has been reproduced and demonstrated experimentally in animals.[1] In humans, the majority of cases have occurred in asthmatics, which points to the fact that regional anaesthesia does not totally reduce the risk of bronchospasm in certain patients. Although the pathophysiological mechanism is not clear, it appears that the neuroaxial block might have a direct or indirect effect on the smooth muscles of the bronchial tree. In the clinical case presented here, the patient had emphysema-type COPD and the respiratory complication was finally attributed to spinal anaesthesia after ruling out all potential reasonable causes of bronchospasm.

Case description

Patient information

Retired 69-year old male Caucasian patient, scheduled for transurethral resection due to benign prostatic hypertrophy. Relevant personal history included no drug allergies, cigarette smoking and a diagnosis of emphysema-type COPD for which he was receiving treatment with inhaled bronchodilators. He had been operated several years before for a fracture dislocation at the level C6-C7, with no reported incidents during anaesthesia or surgery.

Clinical findings, diagnostic assessment and interventions

As part of the preoperative workup, a recent CT scan showed a pattern of panacinar emphysema and findings of multiple bronchiectasis and bullae. All other tests (laboratory, EKG) were within normal ranges.

On the day of surgery, the patient received his usual bronchodilator treatment and reported no recent respiratory infection or current symptoms of exacerbation. Antibiotic prophylaxis with amoxicillin 1 g iv was given 20 min before the intervention. After standard monitoring, premedication with midazolam 0.5 mg iv was given, followed by the initiation of a slow infusion of 500 ml of Ringer's lactate. Spinal anaesthesia was then administered with a 27 G needle at the level of L3-L4 using 0.5% hyperbaric bupivacaine 10 mg, uneventfully. Anaesthesia was demonstrated at the level of T6-T7 before the start of the surgery. Thirty-five minutes into the procedure the patient reported dyspnoea with no abnormal changes in vital signs initially: blood pressure (BP) 120/70 mmHg, heart rate (HR) 85 bpm and oxygen saturation (Sp02) of 98% with nasal cannula at 3 l/m. No changes were identified in the depth of anaesthesia and there were no adventitious breath sounds on lung auscultation. However, 4 min later, dyspnoea worsened and the patient exhibited tachypnea, slight HR increase to 90 bpm, lowering of Sp02 down to 92%, and onset of disseminated wheezing on lung auscultation, with no additional associated symptoms. Bronchospasm was treated immediately with methylprednisolone 100 mg iv and salbutamol spray 2.5 mg, with progressive improvement of the clinical findings. The intervention was completed 10 min after the episode and the patient was transferred to the post-anaesthetic care unit (PACU).

Follow-up and outcome

The clinical manifestations resolved within 20 min of arrival at the PACU. Laboratory testing and portable chest X-rays were performed and no significant abnormalities were found, except for hyponatremia at 119 mEq/L. In view of the possibility of post-TUR reabsorption syndrome, furosemide 10 mg iv was administered, although at the time the patient was already completely asymptomatic.

After 6 h under observation and no additional findings, the patient was transferred to the floor and later discharged after four days from the date of admission.

Discussion

For the review of the literature, a search was conducted in *Pubmed* of all articles published in any language, with no time limitation, using the terms "spinal anaesthesia and bronchospasm" and "spinal anaesthesia and complications".

Described complications following spinal anaesthesia include haematomas, infection, back pain, headache, pneumoencephalus, neurological lesions, needle or catheter rupture, total spinal anaesthesia, thermoregulatory dysfunction, hypotension, and bradycardia, and other more rare complications such as hearing loss, or VIth cranial nerve palsy.[2] In contrast, lung physiology abnormalities are minimal, despite the fact that the blockade obtained reaches thoracic levels. Those changes, such as loss of abdominal contribution to forced expiration – leading to diminished vital capacity – may be more evident in patients with chronic pulmonary disease. However, bronchospasm following spinal anaesthesia is extremely rare in healthy patients as well as in patients with bronchial disease. Airway manipulation is considered the main triggering factor of bronchospasm during anaesthesia.[3] Although a clear consensus is lacking in this regard, subarachnoid or epidural anaesthesia are considered

attractive options when wanting to avoid intubation.[4] Specifically in asthmatic patients, there appears to be a lower risk of bronchospasm under regional anaesthesia compared to general anaesthesia, the main goal being the elimination of neural reflexes caused by intubation which have been found to give rise to the contraction of the smooth muscles of the tracheobronquial tree. However, regional anaesthesia is no guarantee of protection against bronchospasm. In 1982, Mallampati[5] published the first case in a pregnant woman given spinal anaesthesia due to abortion, with an additional two cases reported years later.[6] All three cases happened in patients with a diagnosis of asthma. On the other hand, the case published by Prabhakar[7] is probably the only one published so far in the English language in an otherwise healthy patient. Our case happened in a male patient with bronchial disease and severe non-asthmatic emphysematous pattern.

Possible causes of bronchospasm following spinal anaesthesia are unclear. It has been speculated that parasympathetic stimulation from surgery may be responsible, considering that the smooth muscle of the bronchial tree has muscarinic receptors, which produce bronchial constriction when activated by cholinergic stimulation. On the other hand, it has been proposed that blockade of sympathetic fibres in the thoracic segments may precipitate bronchospasm, considering that they are responsible for the conduction of bronchodilator nerve impulses. This has been replicated in animals, confirming that spinal anaesthesia in animals increase bronchial constriction response to metacholine.[1] Moreover, a sympathetic block above T10-L1 could reduce adrenal production of adrenalin and, considering that clearance of plasma adrenalin is fast, a lower level of this substance during anaesthesia could lead to bronchospasm. Also, anxiety has been considered a favourable factor because the use of anxiolytics could prevent bronchospasm in selected patients.

The diagnosis of bronchospasm attributable to spinal anaesthesia must be made after ruling out all other potential causes. In our case, post-TUR reabsorption syndrome and fluid overload or cardiac asthma were causes that had to be considered as part of the differential diagnosis, but no clinical signs were observed of cardiac overload or ventricular failure, or crepitus or jugular engorgement, and the X-ray taken in the immediate postoperative period was similar to the preoperative radiograph, with no findings of heart failure or acute pulmonary oedema; also important is the fact that the clinical manifestations resolved before the administration of furosemide. Another possible diagnosis that had to be considered was an allergic reaction to local anaesthetics, which occurs typically within 10 min of the injection and is associated, besides bronchospasm, with skin rash or facial blush and hypotension. In our patient, symptoms appeared after 35 min and they were not associated with any other finding attributable to an allergic reaction. No oral or parenteral medications had been administered previously, except for amoxicillin as prophylactic antibiotic, which could not explain the complication on the grounds of either time or symptomatology. Pain or inadequate anaesthesia could have also triggered the symptoms as a result of a vagal reflex, but in our patient the anaesthetic level was the right one and the patient was comfortable. Finally, motor block of abdominal and thoracic muscles may compromise ventilation, and although it is possible that such compromise may have influenced the onset of dyspnoea, the clinical manifestations were indicative of bronchospasm and not of muscle fatigue.

We are aware that, given the multiple causes that may trigger bronchospasm, this may be a limitation of the clinical case presented here. However, we think that this limitation is also a strong point because, having ruled out all potential causes of bronchospasm, and given the precedent in humans and in an animal model of higher bronchial reactivity after spinal anaesthesia, our diagnostic approach was correct.

In summary, bronchospasm is a condition that may occur unexpectedly during anaesthesia, although it is very rare following spinal anaesthesia. In this case, our patient developed bronchospasm following subarachnoid anaesthesia, which could not be attributed to any other cause, adding one more case to the scant literature on this topic. In our opinion, further studies are required to elucidate how neuroaxial blockade affects the smooth muscle of the tracheobronchial tree. Additionally, it is important to recognize that although spinal anaesthesia is considered the safest modality in patients with respiratory disease, specifically in asthmatics there is a possibility that it may induce bronchospasm in susceptible patients.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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