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Anaesthesia Cases

Dexmedetomidine to enable MRI scanning in a patient with airway compromise.

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Summary

We present the case of an obese gentleman with airway compromise due to a large tumour within the left thoracic inlet, who required a magnetic resonance imaging (MRI) scan to plan further treatment. He was profoundly claustrophobic and required sedation in order to undergo the scan. We describe the use of dexmedetomidine supplemented with a small amount of midazolam to successfully sedate the patient whilst avoiding respiratory depression and obtundation of airway reflexes associated with higher doses of midazolam or propofol sedation.

Introduction

It is well known that general anaesthesia in the patient with an anterior or superior mediastinal mass can precipitate cardiorespiratory collapse or even fatal airway obstruction. The maintenance of spontaneous ventilation in 'at risk' patients is key to avoiding these complications. Dexmedetomidine is a highly selective α2 adrenergic receptor agonist with sedative and anxiolytic properties which causes minimal respiratory depression even in high dose [1].

Report

This 48-yr-old male presented after he noticed a growth in his left supraclavicular fossa. His previous medical history included hypertension, gastro-oesophageal reflux (controlled on omeprazole) and he was obese with a BMI of 37.8 $kg.m^{-2}$.

The growth was confirmed on CT scanning as a massive bi-lobed tumour on the left side of the patient's neck extending through the thoracic inlet into the left side of the superior mediastinum, from the C6 to T3 vertebral levels. A subsequent biopsy confirmed a schwannoma arising from the C7 region of the cervical spine. The tumour had eroded the left C7 facet joint and a significant proportion of the C7 vertebral body. It was also causing tracheal deviation and compression, especially at the T1/T2 intervertebral disc level, resulting in a crescent shaped narrowed trachea with a measured diameter of 6 mm. Despite this degree of tracheal compression, the patient denied any respiratory symptoms, including cough, dyspnoea or orthopnoea.

MRI images were required to adequately plan the complex, multi-stage operative combined procedure between the neurosurgeons and cardiothoracic surgeons. Unfortunately the patient had, on a previous unrelated occasion, become trapped in an MRI scanner and developed severe claustrophobia.

Committed to his treatment, the patient attempted to undergo the scan without sedation but became acutely distressed and was unable to complete the scan. Plans were therefore made to sedate him, but as his degree of distress when approaching the MRI suite was so profound, and complicated by his superior mediastinal mass with tracheal compression and obesity, we developed a treatment plan.

We decided to avoid sedative medications associated with airway reflex obtundation and respiratory depression as much as possible, and therefore chose to use dexmedetomidine as the primary agent for sedation. We administered a loading dose of 1 $mcg.kg^{-1}$ over 15 min followed by an infusion of 0.7 $mcg.kg^{-1}.hr^{-1}$ in a clinical area adjacent to the MRI scan room. Following the loading dose, the patient felt able to be transferred to the MRI table, however on transfer, he once again became acutely distressed. We titrated an additional 0.02 $mg.kg^{-1}$ midazolam (3mg in total) and the patient was able to comply with the scanning process without distress.

The patient's vital signs remained within 20% of baseline, although he did require supplemental oxygen by nasal cannulae to maintain oxygen saturations >92%. He recovered rapidly on cessation of the dexmedetomidine infusion and was discharged home later the same day. The MRI images further demonstrated the tumour-related tracheal

deviation and compression (Figure 1).

Discussion

General anaesthesia, muscle relaxation and positive pressure ventilation in patients with extrinsic compression of the major intrathoracic airways can precipitate complete airway obstruction. This is due to loss of the transpleural pressure gradient that is present during spontaneous ventilation and which serves to 'pull open' the intrathoracic airways thus protecting them against the external compression. Relaxation of bronchial smooth muscle further increases the risk of airway collapse, as does the reduction in lung volume which occurs under anaesthesia, resulting in a proportional reduction in tracheo-bronchial diameter.

Evidence from paediatric studies suggests that compression of >50% of the tracheal diameter is a particular risk factor for respiratory complications under general anaesthesia [2-3]. Although paediatric patients are likely to be at higher risk due to their more compressible airways, caution is still advised when approaching anaesthesia for adults with this degree of airway obstruction, even in the absence of respiratory symptoms. Some authors recommend that patients with a 50% or more reduction in tracheal cross-sectional area should have their femoral vessels cannulated prior to induction of general anaesthesia in readiness for cardio-pulmonary bypass in the event of cardiorespiratory collapse [4].

Our approach to the problem was to avoid general anaesthesia, aiming to sedate our patient in order for him to tolerate the MRI scan. However, because of his profound claustrophobia and procedure-related anxiety, we were concerned that the high dose of our standard sedative medications (propofol and midazolam) that would be required would potentially lead to apnoea and airway obstruction. We therefore elected to use dexmedetomidine as our primary sedative agent.

Dexmedetomidine is a highly selective α2 adrenergic receptor agonist with sedative and anxiolytic properties and produces minimal respiratory depression even in doses greater than ten times the maximum recommended dose [1]. It has been advocated for use when sedating children with obstructive sleep apnoea for MRI scanning where its use is associated with a significantly reduced need for artificial airway support compared with propofol [5].

Dexmedetomidine provides a unique form of sedation as its sedative effect is mediated by agonism at the α2 adrenoceptors in the locus coeruleus. This sub-cortical sedation more closely resembles natural sleep in electroencephalography (EEG) studies [6] and is characterised by co-operative and communicative patients when stimulated, compared with the GABA-mimetic sedative agents. As dexmedetomidine has an α2:α1 selectivity ratio of 1600:1, compared with 200:1 for clonidine, it is a more effective sedative agent. We did not observe bradycardia, which can complicate the use of dexmedetomidine, nor hypertension that may be seen at higher doses due to the activation of vascular smooth muscle α2 receptors. The predominant effect of dexmedetomidine at lower doses is hypotension due to a reduction in sympathetic tone, and our patient did demonstrate a modest reduction in blood pressure, however this did not require intervention.

In the European Union, dexmedetomidine is licensed for sedation in adult intensive care unit patients only. However, in October 2008, the United States Food and Drug Administration approved the use of dexmedetomidine for procedural sedation in non-intubated patients, and its use for a variety of indications is described in the literature. These include sedative premedication to facilitate awake fibreoptic intubation and awake transoesophageal echocardiography, as a perioperative opioid-sparing agent in bariatric surgery, for procedural sedation for radiological and gastrointestinal endoscopic procedures. The use of dexmedetomidine as the primary anaesthetic agent in certain clinical situations where the maintenance of spontaneous ventilation is essential, such as to allow tracheal laser tumour debulking and tracheal stenting, has been described [7]. Our own experience of the drug is mainly from using it to allow neurocognitive testing during awake craniotomy procedures.

We used a dose of 0.7 $mcg.kg^{-1}.hr^{-1}$ following a bolus dose of 1 $mcg.kg^{-1}$ over 15 min as directed by the manufacturer. Many authors find this dose inadequate as a sole agent [8], but as higher doses may cause cardiovascular side effects, some opt to add small doses of other agents as adjuncts when required. The benzodiazepine sparing effect of dexmedetomidine is well established [1] and with dexmedetomidine as our primary sedative agent, we found a low dose of only 0.02 $mg.kg^{-1}$ midazolam was sufficient to supplement the sedation, avoiding the respiratory and airway tone depressant effects of midazolam at higher doses.

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Published with the written consent of the patient.

Competing Interests

No external funding and no competing interests declared.

Image

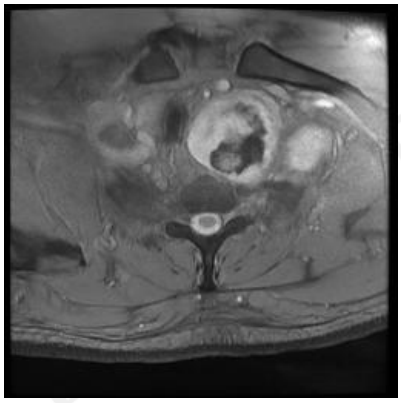

Figure 1. MRI scan demonstrating tumour-related tracheal deviation and compression

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