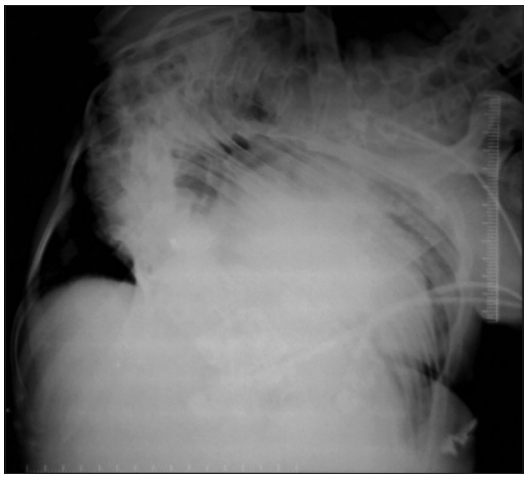

Figure 1: Chest X-ray anteroposterior view

Dexmedetomidine to wean patient of severe kyphoscoliosis with cerebral palsy in intensive care unit

Sir,

Patients with severe kyphoscoliosis with cerebral palsy may present with an acute respiratory failure (ARF) precipitated by an acute respiratory infections, requiring invasive mechanical ventilation.[1] Weaning from ventilator support may be challenging due to agitation and lack of patient co-operation.

A 27-year-old male patient suffering from cerebral palsy and congenital kyphoscoliosis (scoliotic angle>100^0) was admitted to the intensive care unit with dyspnoea, cough and fever. Patient was drowsy with tachycardia, tachypnoea, blood pressure (BP) 90/66 mm Hg and saturation (SpO_2) 86%. On auscultation bibasilar inspiratory crackles were heard. There were no signs of congestive cardiac failure. Arterial blood gas (ABG) showed pH of 7.32, pCO_2 45 mm Hg, pO_2 50 mm Hg and haematocrit 40%. Chest X-ray showed severe chest deformity and haziness [Figure 1]. Patient was intubated and mechanically ventilated without much difficulty. Initial ventilator settings were volume control ventilation, tidal volume 400 ml, FiO2 1.0, positive-end expiratory pressure 8 mm Hg and frequency 20/min. ABG sample taken after an hour showed pO_2 250 mm Hg, pCO_2 40 mm Hg, SpO_2 100% and FiO_2 was hence subsequently reduced to 0.5 while maintaining adequate saturation. On 2nd day ventilation was changed to the synchronized intermittent mandatory ventilation mode with pressure support of 12 cm H_2O, but he became visibly agitated, developed tachypnoea and his heart rate rose to 160/min. On auscultation bilateral crackles were still present with lot of secretions. Infusion of midazolam 2 mg/h was started to sedate the patient. Injection paracetamol 1 g 8 hourly was started for analgesia. Fluids were given to increase central venous pressure from 2 to 6 cm H_20, which settled his HR to 130/min and BP 106/70 mm Hg. On day 3, secretions reduced, but reducing the midazolam infusion lead to tachypnoea and HR of 164/min. We then started patient on injection dexmedetomidine (DEX). A loading dose 1 mcg/kg for 10 min was given followed by 0.2 mcg/kg/h maintenance infusion. Injection midazolam was stopped and no haemodynamic compromise was observed.

After 18 h of starting DEX, patient was successfully breathing on continuous positive airway pressure and subsequently T-piece. Injection DEX was stopped. ABG after half an hour was good, vitals were stable and patient was no longer agitated and hence he was extubated.

The primary goal in patients presenting with ARF in severe kyphoscoliosis is correction of arterial hypoxemia.

Our patient was intubated considering his mental condition[2] and he developed agitation requiring

sedation to decrease excessive central respiratory drive and improve patient-ventilator synchrony. The recent revised guidelines for pain, agitation and delirium in the critically-ill suggest the use of propofol or DEX instead of benzodiazepines (BZP) for agitation, but also summarised that the current literature supports only modest differences in outcomes with BZP-based versus non-BZP-based sedation (3). BZP remain important for managing agitation in intensive care unit (ICU) patients, especially for treating anxiety, seizures and alcohol or BZP withdrawal.[3] Thus injection midazolam was started as it is easily available in our setup but could not be tapered successfully and so we used DEX next.

DEX is a highly selective alpha-2 agonist. A major advantage is that it is associated with minimal respiratory depression[4] thus, allowing continuous sedation during the entire weaning process until extubation. DEX also inhibited the haemodynamic stress response during weaning from mechanical ventilation in our patient. It also helps eliminate emergence agitation when sedation is being tapered. Lack of addictive properties, cumulative effects or clinical signs of withdrawal, make DEX an attractive potential alternative to current ICU sedation regimens.[5]

We did not observe any bradycardia or hypotension with DEX. It could be because of minimum infusion dose that was used for maintenance and normovolemia. The sedative and analgesic property of DEX helped us wean the patient successfully. Thus, we were able to use DEX safely and effectively in a condition where patient cooperation was just not possible and respiratory depression at the time of extubation could be dangerous due to severe restrictive lung disorder.

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