Unusual adverse effect of dexmedetomidine and its management

Sir,

Dexmedetomidine, a drug which paved the way into anaesthesia for Intensive Care Unit sedation, has put firm stay into every segment of anaesthetist's armamentarium. We report an unusual adverse effect of dexmedetomidine, its timely recognition and successful management.

A 45-year-old American Society of Anaesthesiologists physical status class 2 female with the difficult airway was scheduled for mandibulectomy and free flap cover for carcinoma of the buccal mucosa. Awake fibreoptic bronchoscope-guided intubation was accomplished safely with airway nerve blocks and dexmedetomidine infusion of 0.5 μg/kg/h started 30 min before intubation. Subsequently, general anaesthesia was induced and maintained with propofol infusion, vecuronium and morphine. Dexmedetomidine infusion at 0.3 μg/kg/ rate was continued.

The patient's urine output was adequate till 9 h into the procedure and thereafter began to increase up to 400 ml/h. The intraoperative input–output fluid chart was matching until the 9th h. The serum and urine sodium were 154 and 16 mmol/L, respectively, at that time. The random blood sugar was 134 mg/dl and accidental or perioperative usage of diuretic was also ruled out. We suspected a drug-related side effect, and we cross-checked the diuretic side effect of used drugs. Based on the previous sparse data, we decided to stop dexmedetomidine infusion and wait for spontaneous resolution. Since haemodynamics were stable, we continued the surgery. Till then, calculated losses were replaced with colloid and desmopressin intranasal spray was also arranged for the failure of the spontaneous resolution of polyuria. After 4 h, there was a spontaneous reduction in urine volume and all laboratory parameters returned to normal. Rest of the postoperative course of the patient was uneventful.

The usual causes of intraoperative polyuria are hyperglycaemia, diuretic administration, injudicious fluid administration and the side effect of some drugs. In our case, we ruled out all possibilities after meticulous workup. Evidence search on the internet revealed few reported cases of polyuria after dexmedetomidine use.

Greening *et al.* reported similar diuresis with dexmedetomidine infusion (at 0.5 μg/kg/h) at the

4th hour with the haemodynamic disturbance, which resolved within 2 h after discontinuation.[1] Pratt *et al*. reported that diuresis occurred with the high maintenance dose of dexmedetomidine.[2] Others have reported an intraoperative diuresis following a bolus dose of dexmedetomidine (at 1 μg/kg/h) without maintenance infusion.[3]

Our case was peculiar, as the diuresis manifested after 8 h with even low-dose infusion as compared to previous reports (at 0.3 μg/kg/h rate following 0.5 μg/kg/h loading dose over 30 min) and it resolved within 4 h of discontinuation without any haemodynamic disturbance.

Dexmedetomidine inhibits the release as well as end receptor action of vasopressin. This mechanism of diuresis tests the validity of desmopressin as a rescue drug in the scenario.[4]

During the polyuric phase, we replaced urine losses with colloids. Colloids were preferred in this situation based on the premise that with the increase in oncotic pressure, there will be a reduction in glomerular filtration. Since we are uncertain about the vasopressin responsiveness, we decided to target the glomerulus rather than the collecting duct.[5]

Therefore, we concluded that diuretic side effect of dexmedetomidine should be considered in the differential diagnosis in any patient receiving dexmedetomidine and presenting with polyuria. Prompt discontinuation of dexmedetomidine may result in spontaneous resolution of polyuria.

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Conflicts of interest
There are no conflicts of interest.

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