

Dexmedetomidine as sedative and analgesic in a patient of sickle cell disease for total hip replacement

Sir,

A 40-year-old, 82 kg male with a fracture of neck of femur was admitted. Airway assessment revealed MPC II. Haemoglobin of 9 g/dl and haematocrit of 26.4% was noted. The peripheral blood smear showed sickle cells. Sickle cell solubility test was positive. The patient had no history of previous hospitalisation, blood transfusion, renal failure, chest pain or any neurologic deficits. Avascular necrosis of bilateral femoral heads and splenic infarcts were noted. Laboratory investigations and chest X-ray were normal. A total hip replacement was planned.

An epidural catheter was introduced at L2-L3 level. Initial bolus of 14 ml of 2% xylocaine with adrenaline after test dose was given to obtain a sensory level of T10 and infusion of 7 ml/hour of 0.5% bupivacaine was used. Adequate oxygenation, hydration and normotension and normothermia were ensured preoperatively and intra-operatively. Intravenous dexmedetomidine, in a bolus of 0.5 µg/kg (40 µg) was infused over 20 min prior to positioning the patient lateral, followed by an infusion of 0.5 µg/kg/hour. Blood loss of 350 ml was managed using warm crystalloids. Intraoperative period was uneventful.

Postoperative pain was managed with an epidural infusion containing 0.2% ropivacaine at 5 ml/hour and dexmedetomidine in a dose of 0.5 µg/kg/hour. No NSAIDs/opioids or sedatives were required in the immediate post-operative period of 24 hours. The pain intensity scale of 3-4 and Ramsay sedation score of 2-3 were charted in the high dependence unit. The epidural catheter was removed after 24 hours and the patient was mobilised. Oral NSAIDs were started. The patient was discharged on the fifth post-operative day without any adverse events.

Sickle cell disease (SCD) is determined by a complex interplay between genetic and environmental factors. The basis of anaesthetic management has traditionally been avoiding the factors known to increase erythrocyte sickling and precipitate the vicious cycle of vaso-occlusive episodes (VOC),

such as volume depletion, hypoxemia, infection, acidosis, hypothermia, hyperthermia, decrease in 2, 3 DPG.^[1] Sickled cells have increased adherence to endothelium leading to micro-vascular infarction and pain, the basis of VOC.^[2] The risk intrinsic to the type of surgery should be considered. Among orthopaedic procedures, hip surgery and hip replacement are associated with a high risk of complications.^[3] Pre-operative examinations should aim to find the risk of SCD-related organ dysfunction.

The main goals of the anaesthetic management in this case were adequate analgesia, early mobilization to prevent stasis and deep vein thrombosis, oxygenation to avoid hypoxic episodes,^[1] adequate hydration and thermoregulation.

Our choice was based on the advantages of dexmedetomidine. It has a rapid onset and offset, predictable mode of metabolism and excretion not requiring end organ-function, no deleterious cardiovascular effect, amnestic effect, no depressant effect on respiration, gas exchange, or pulmonary mechanics.^[4] Infusion maintains unique 'conscious sedation'. Its action at the spinal cord alpha₂ receptors diminishes sympathetic tone and serves as an analgesic at the dorsal horn,^[4] thus, decreasing the requirement of local anaesthetic epidurally. Studies comparing dexmedetomidine with either opioids or other alpha₂ agonist namely clonidine have been documented.^[4] Only one case of its use in acute sickle cell crisis for pain relief has been reported.^[5] Intraoperative dexmedetomidine infusion is effective in prevention of post-anaesthesia shivering,^[6] thereby preventing the increased oxygen demand.

Opioids used for pain relief may cause respiratory depression.^[5] Dexmedetomidine has opioid sparing effect, thus offering an advantage.

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