Prevention and treatment of sevoflurane emergence agitation and delirium in children with dexmedetomidine

Sir,

We read with interest the article "prevention of sevoflurane related emergence agitation (EA) in children undergoing adenotonsillectomy: A comparison of dexmedetomidine and propofol" by Ali and Abdellatif[1] and wish to report a case in which very severe EA in a child responded only to intravenous (i.v.) dexmedetomidine.

A 13-year-old boy who underwent left myringotomy and grommets insertion under sevoflurane anesthesia, with midazolam (1 mg), fentanyl (100 mcg) and propofol (150 mg) induction, suddenly became very agitated and combative in the postanesthesia care unit (PACU), kicking and thrashing around, and showing paranoid delusions and having hallucinations. EA/emergence delirium (ED) was diagnosed. He accidentally removed his i.v. catheter, which was immediately replaced after which a bolus of propofol 30 mg was administered. However, he became even more agitated, kicking and punching medical and nursing staff. He did recognize his mother who was by now extremely alarmed and agitated. He was given i.v. midazolam 4 mg total, morphine 3 mg and further 30 mg boluses of propofol to a total of 150 mg, but without any lasting effect. Subsequently, he was administered i.v. dexmedetomidine 0.4 mcg/kg over about 6 min with almost immediate resolution of the EA/ED. He subsequently emerged from a brief period of restful sleep, calm, alert and orientated without any signs of delirium. He was subsequently discharged after 30 min from the PACU to the ward.

Emergence delirium has been defined as "a disturbance in a child's awareness of and attention to his/her environment with disorientation and perceptual alterations including hypersensitivity to stimuli and hyperactive motor behavior in the immediate postanesthesia period."[2] The most important risk factors are the use of the newer halogenated inhaled anesthetics, such as sevoflurane or desflurane, which have been postulated to alter brain activity by interfering with the balance between neuronal synaptic inhibition and excitation in the central nervous system,[3] and the child's temperament, with children who are more emotional and more impulsive being at increased risk.[4]

This case supports the findings of Ali and Abdellatif[1] that dexmedetomidine, a selective alpha-2 agonist with sedative, analgesic and anxiolytic properties is significantly more effective than propofol 1 mg/kg or placebo in decreasing the incidence and severity of EA/ED when administered intraoperatively to children having surgical operations under sevoflurane anesthesia. It further demonstrates that dexmedetomidine can also treat EA/ED in the PACU. We recommend that it should be used as first line therapy for severe EA in PACU perhaps in combination with small doses of propofol to initially sedate the child.

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