Accidental tenfold overdose of propofol in a 6-months old infant undergoing elective craniosynostosis repair
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Introduction: Craniosynostosis is related to the premature closure of skull sutures in infants. Corrective procedures are performed in young infants and represent major surgery with unavoidable, extensive blood loss, particularly if multiple sutures are involved (1, 2). We report on a 6-month old male infant undergoing elective craniosynostosis repair who accidentally received a tenfold propofol dose over an intraoperative period of 4 h.

Case Report: A 6-month old, 6700 g otherwise healthy male infant with trigonocephalus was scheduled for elective craniosynostosis repair. After induction with fentanyl, thiopentone and cisatracurium anesthesia was maintained using an undiluted continuous propofol (1%) infusion. A dose of 30 mg/h propofol (4.47 mg/kg/h) was intended, however, 30 ml/h, corresponding to 300 mg/h (= 44.7 mg/kg/h) was accidentally programmed into the perfusor and infused unremarked. One hour after induction a continuous IV remifentanil infusion was started at 0.35 µg/kg/min. Two hours after induction surgery began. Thus far, the infant had received 44.7 mg/kg/h propofol for two hours and 0.35 µg/kg/min remifentanil for one hour with invasive systolic blood pressures (IBP) above 70 mm Hg, heart rate (HR) between 100 - 115/min and a central venous pressure (CVP) of 4 cm H₂O. Volume loss was replaced guided by CVP with a target of 6 – 8 cm H₂O. The initial hemoglobin concentration was 9.3 mg/dL. Erythrocyte concentrates (EC) and fresh frozen plasma (FFP) were administered continuously, supplemented by 300 mg calcium. HR remained stable at 100/min with IBP between 60-70mm Hg; CVP was 6 cm H₂O. Despite IV administration of 50 µg atropine twice, no increase in HR was observed. 50 min after surgery began systemic IBP dropped to 40 mm Hg, HR remained at 100/min with a CVP increase to 12 cm H₂O. After the IV administration of adrenaline 1 µg, systolic IBP increased to 90 mm Hg and HR to 140/min. Dopamine and dobutamine were administered continuously. Hemoglobin was 11.1 mg/dL. Thus far the child had received 700 mL EC and 450 mL FFP. Systolic IBP was stabilized at 100 mmHg, HR at 125/min and CVP at 7 cm H₂O. Dopamine and dobutamine infusions could both be reduced. Four hours after beginning the propofol infusion, the overdosage was recognized and the propofol infusion stopped. Anesthesia was maintained with remifentanil at 0.24 µg/kg/min and IV midazolam boli. Infusion of EC and FFP was continued and CVP kept at 8 – 9 cm H₂O. Dopamine and dobutamine infusions were further reduced and 30 min later discontinued. Hemoglobin was 10.9 mg/dL with no signs of metabolic acidosis. Throughout the procedure the child had received 300 mL of crystalloid infusion, 900 mL of EC and 715 mL of FFP. Urine output was 240 mL. Postoperatively, the infant was transferred to the ICU where ECG and echocardiography showed no evidence of cardiac damage or dysfunction. 24 h after admission the infant was extubated uneventfully. Postoperatively, all laboratory tests were within the normal range except for an intermittent increase in creatinine kinase (101 U/L) and lactate dehydrogenase (336 U/L). On postoperative day 3 the child was transferred to the general paediatric ward and discharged home after 14 days without clinical evidence of neurological or cardiac deficits.

Discussion: Propofol has been used widely for induction and maintenance of anesthesia, and to sedate critically ill children receiving mechanical ventilation (3,4) The propofol infusion syndrome in children receiving long-term propofol sedation is associated with rhabdomyolysis, refractory lactic acidemia, bradyarrhythmia, lipaemic plasma and even myocardial failure (5-7). The intermittent postoperative increase in lactate dehydrogenase and creatinine kinase in our infant was most likely due to surgical trauma. Despite the propofol overdosage for 4 h our infant neither developed lactic academia nor lipaemic plasma. The myocardial failure observed in our patient cannot solely be accounted for by the propofol overdose. Other mechanisms such as increased intracranial pressure, venous air embolism, and extensive blood loss are possible factors which could have contributed to the sudden hypotension. Estimating an approximate blood volume of 570 mL in this infant, the blood volume was exchanged 2.0 times during intraoperative transfusions. We, therefore, cannot rule out the possibility that a short hypovolaemic episode contributed to the cardiac decompensation, especially since the catecholamine infusions could be reduced before the propofol overdosage was noticed. Whether a single factor or multiple mechanisms lead to the observed myocardial failure, therefore, remains unclear.

Refs: