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Paraplegia from thoracic epidural anesthesia in a patient with sickle cell disease and normal SaO_2

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SUMMARY

Objectives: In addition of the usual care required when extensive abdominal surgery is performed, certain special precautions are considered necessary in patients with sickle-cell disease. One such case that received thoracic epidural and general anesthesia developed a major neurological deficit as some of these precautions seem to have been forsaken. **Methods:** A 19 year old man with sickle cell disease received epidural and general anesthesia for a cystectomy, lymph node dissection and cholecystectomy lasting 9½ h. He received 2% Lidocaine with 1:200,000 epinephrine in bolus intermittently, for a total of 1,240 mg and 310µg , respectively. SaO_2 ran between 96 and 100%. Upon conclusion of surgery, the epidural catheter was infused with meperidine 0.1% and bupivacaine 0.1% until it was found to have motor and sensory block to L2 level. An MRI revealed an infarct in the conus medularis. **Results:** The patient has remained paraplegic including bowel and bladder dysfunction for over 4 years. **Conclusion:** Though acceptable SaO_2 were present throughout the procedure, the patients temperature fell near 35°C, there was evidence of sickling and hemolysis. No changes have occurred in the neurological deficit of the patient in the last fewer years. Other possible causes are discussed.

Key words: Sickle cell disease, paraplegia, epidural anesthesia, lidocaine, epinephrine.

RESUMEN

Objetivo: *Los pacientes con enfermedad de células falciformes sometidos a cirugía abdominal extensa requieren de cuidados especiales en el transoperatorio. Se reporta el caso de un paciente con enfermedad de células falciformes que desarrolló déficit neurológico después de anestesia combinada general y epidural.* ***Método:*** *Hombre de 19 años con enfermedad de células falciformes que recibió anestesia general y epidural con duración de nueve horas treinta minutos para procedimiento quirúrgico que consistió en cistectomía, disección de ganglios linfáticos y colecistectomía. Recibió lidocaína al 2% con epinefrina 1:200,000 en bolos, hasta alcanzar una dosis total de 1,240 mg y 310 mg respectivamente. La SaO_2 osciló entre 96 y 100%, la temperatura descendió a 35ºC. Al terminar la cirugía se aplicó a través del catéter epidural bupivacaína al 0.1% y mepiridina al 0.1%. El enfermo desarrolló nivel sensitivo y motor a nivel de L2. La resonancia magnética nuclear mostró infarto en el cono medular. El paciente continuó parapléjico con disfunción vesical e intestinal.* ***Conclusiones:*** *A pesar de que la saturación de oxígeno se mantuvo en rangos normales la hipotermia condicionó deformidad eritrocitaria y hemólisis con lesión neurológica irreversible.*

Palabras clave: *Enfermedad de células falciformes, paraplejía, anestesia epidural, lidocaína y epinefrina.*

Major surgical interventions in patients with sickle cell disease present added risks because of the potential complications derived from the anesthetic management. Several factors may precipitate sickling of red cells which then may superimpose morbidity, one of which may be Neurologic deficit if epidural anesthesia was administered. One such case culminated in a catastrophic paresis as described.

CASE REPORT

A 19-year-old black male known to have sickle cell disease (SCD), was admitted to a large county hospital with the diagnosis of ganglioma of the bladder, cholelithiasis, splenic hemangiomas, alcohol and tobacco use; he had had a prior hospitalization for an episode of dehydration and acute sickle cell crisis. He was scheduled to undergo an open cholecystectomy, partial cystectomy and bilateral pelvic lymph node dissection; these procedures were done in a single day in an operation which lasted 9 h 30 min.

An epidural catheter was inserted pre-operatively at the T8-T9 intervertebral space, with the patient in the sitting up position and advanced caudad 4 cm; 10 ml of 2% lidocaine with 1:200,000 epinephrine were injected. Ten minutes later, the blood pressure dropped from 136/50 to 100/60 returning to 120/60 after the administration of a bolus of IV fluids. Fifteen minutes later, general anesthesia was induced with 175 mg of Propofol, 50µg of fentanyl IV, and 8 mg of vecuronium given to facilitate intubation of the trachea and was maintained with isoflurane from 0.5 to 1%, nitrous oxide 1 l/min and oxygen 0.7 l/min. The FiO_2 was kept between .3 and .34, the body temperature was recorded to be as low as 35.1ºC. SaO_2 was noted to be between 96 and 100% throughout the procedure, and the end-tidal pCO_2 ranged between 28 and 36 mmHg. A total of 62 ml of 2% lidocaine (1,240 mg) were given in intermittent bolus of 9 ml at variable intervals, including 310µg of epinephrine (1:200,000). The EBL was 400 ml, the urine output was recorded as 1,200 ml plus what was lost in the operative field, while 12,000 ml of Ringer's lactate were given intravenously. The patient's trachea was extubated at the end of the procedure and he was sent to the ICU for observation.

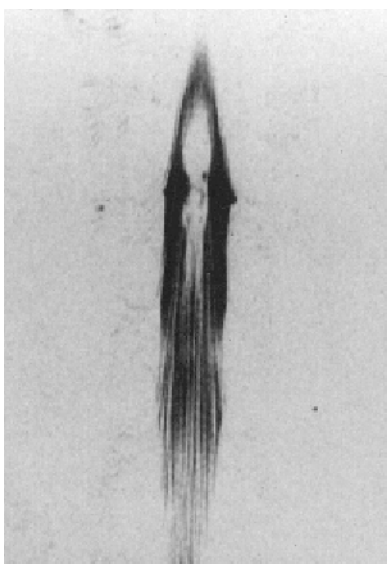

Figure 1. MRI of the conus medullaris and the cauda equina depicting an area of infarct on the right side extending towards the center.

The epidural catheter was left in place and connected to a PCA pump infusing meperidine 0.1% and bupivacaine 0.1% at 8 ml/h for postoperative analgesia. The next day, he was noted to have complete motor and sensory deficit below L_2. SaO_2 readings were "in the upper 80's" in spite of receiving 4 l/min of supplemental O_2 by facemask. Chest X-ray showed bilateral pleural effusion and pulmonary edema; the Hct was 27%, with evidence of hemolysis and sickling at blood microscopy. A CAT scan was taken which showed "no evidence of epidural hematoma, abscess or tumor." The epidural infusion of analgesics was discontinued. On the fourth postoperative day, an MRI revealed "spinal cord infarct in the lower cord (conus region). Suspect infarct secondary to sickling event in the perioperative period" (Figure 1). Three weeks later, the pleural effusions were aspirated. On the 44th post-op day, the patient was sent to a spine clinic for rehabilitation. There has been no change in the complete sensory and motor deficit of the patient who continues to require bowel and bladder care, and has remained paraplegic four years later.

DISCUSSION

Sickle cell disease is characterized by the presence of hemoglobin S that forms a semisolid gel during hypoxemia and/or acidosis due to reversible polymerization that deforms the red cells in the shape of a sickle[1]. The usual triggering factors are PaO_2 below 40 mmHg or a PH between 6.5 to 7 that initiate sickling within 2 minutes. The degree of the morphological deformity is directly related to the rate of deoxygenation, the level of 2-3 DPG and the mean corpuscular hemoglobin index[2]. When erythrocytes sickle, they become rigid and more readily adhere to the vascular epithelium obstructing the capillary lumen provoking distal ischemia and infarct, then those re-entering the circulation, hemolize[1]. The administration of anesthesia in patients with SDC requires certain precautions to avoid compromise of the oxygen delivery to hypoxia sensitive sites[2]. Specifically in SCD, adequate hemoglobin oxygenation is primordial to prevent "sickling" or the alteration of erythrocyte morphology that would impede their adequate passage through microcapillary vascular beds, being the CNS (cerebral and spinal cord), coronary, renal and enteric the most susceptible[3,4].

In this patient, after induction of anesthesia, the FiO_2 was maintained only at around 0.3, the SaO_2 readings remained

near 100%, but the temperature fell to near 35ºC and hypocarbia was allowed to develop during surgery, both of which may produce vasoconstriction inspite of the near 100% SaO_2 readings that gave the anesthesia team a false sense of security, since tissue desoxygenation apparently occurred as consequence of reduced arteriolar blood flow to the spinal cord[5]. There are several factors involved that might have compromised the arterial blood supply to the distal end of the spinal cord. Bilateral lymph node pelvic dissection might have interrupted the blood supply provided by the radicular arteries as Usubiaga et al[6] reported in two cases under epidural anesthesia that developed permanent neurological deficits after prevertebral surgery.

High concentration of lidocaine has been shown to produce irreversible conduction loss in frog nerves[7]; the substantial dose of 1,240 mg in 9 h might have remained in contact with nerve roots and conus medularis longer than usual, as the patient was slightly hypothermic and by the addition of epinephrine (310 µg), administered into the epidural space over the course of 9½ hours of surgery, both could have exacerbated the precapillary constriction further depriving the conus medularis and the cauda equina of oxygen carrying erythrocytes[8]. This selective ischemia is feasible under epidural anesthesia when epinephrine reduces the absorption of lidocaine[8,9], not only promoting ischemia from a reduced blood flow[3,6,9] but also by potentiating axonal degeneration as commercially available solutions of epinephrine contain sodium bisulfite, a known neurolytic substance[10,11].

Vasoconstriction might have also contributed to the infarct by way of the one single vessel that gives arterial blood supply to the most lower portion of the cord, ensuing in the conus medularis and cauda equina receiving only half of their oxygen supply from the unpredictable branching of the Adamkiewicz artery[12] and the other half from the CSF[13], rendering them more susceptible to ischemia when vasoconstriction occurs.

Although Sakura et al[14] suggested that a "deeper block" may be obtained when 1:200,000 epinephrine is added to lidocaine, the rationale for its use in continuous epidural anesthesia is questionable as the presence of a catheter allows for repeated injection. Specifically, the use of epinephrine, by any route, has been thought to be contraindicated in patients with SCD authors[1-3,15,16].

Since there was evidence of hemolysis and sickling in this patient's blood, it is probable that a reduced blood flow produced an infarct in a cold, vasoconstricted arterial vascular bed. A prompt Neurology consult and the appropriate (MRI) imaging study might have revealed the precise diagnosis, at the time, when a therapeutic intervention during the "window of opportunity" might resulted in a more favorable outcome[17].

The causes that might have contributed to produce paraplegia and loss of neural control of the bladder and bowel in a young patient with SCD under epidural and general anesthesia are discussed with emphasis in avoiding the use of epinephrine, lidocaine (as potentially neurotoxic agent) and hypothermia in this group of patients even when SaO_2 appears to be within normal range.

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