Cardiac arrest after bolus of epidural catheter in a 9 year old boy

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After participating in the PBLD the learner will be able to:

1. Understand the potential complications of an epidural catheter
2. Know common safety guidelines when performing regional anesthesia
3. Make a differential diagnosis in case of cardiac arrest after regional anesthesia
4. Better understand the effects of local anesthetics on cardiac electrophysiology

A 9 year old boy with Pfeiffer syndrome comes to the operating room for take down of a colostomy done immediately after birth for anal atresia.

1. What different options are available to manage postoperative pain in this patient?

Consent was obtained to place an epidural catheter under general anesthesia before starting the surgical procedure. After induction of general anesthesia and intubation the patient was placed into the lateral decubitus position for placement of an epidural catheter. A 20 G multi-orifice catheter was placed under sterile conditions at the L3±L4 level using an 18 G Tuohy needle. The epidural space was identified by a loss of resistance technique at the 1st attempt, and the catheter was advanced 5 cm into the epidural space without difficulty. Aspiration for blood and cerebrospinal fluid was negative as well a test dose of lidocaine 1% 2 ml with epinephrine 1:200 000 produced no effects.

1. What is the most effective way of ruling out the intravascular placement of an epidural catheter in an anesthetized patient?
2. How do we recognize an intravascular placement of an epidural catheter?

The patient was then placed in the supine position. While the surgeons were preparing the abdomen, the patient received bupivacaine 0.25% 3 ml with epinephrine 1:200 000 epidurally, after negative aspiration for blood. We had planned to give boluses to a total dose of 10 ml of this local anesthetic. One minute after the 1st dose of bupivacaine, dysrhythmias were noticed on the ECG.

1. What cardiac changes should we expect after an intravascular injection of a high dose local anesthetic? Why?
The patient's heart rate remained stable (74±78 beats min⁻¹), as did the arterial blood pressure (systolic 90±95 mm Hg, diastolic 40 mm Hg). Because of the rhythm abnormalities, we decided to wait before administering the full dose of epidural bupivacaine. Approximately 3 minutes later and before surgical incision, the heart rhythm suddenly changed into ventricular tachycardia and a few seconds later into ventricular fibrillation and cardiac arrest.

1. **What should be the treatment of a cardiac arrest in a similar scenario?**

After resuscitation measures, the heart rate rose to 180 beats minute and the arterial blood pressure to 150/100 mm Hg. A few seconds later, the patient had a second episode of ventricular fibrillation that resolved after brief chest compressions. The heart rate stabilized around 100 beats minutes, and arterial pressure stabilized with systolic values around 90 mm Hg.

1. **What would you do next?**

Blood samples were taken for measurement of plasma bupivacaine concentration. Arterial blood gas was normal, with the exception of hypokalemia (potassium 2.8 mmol litre⁻¹), probably because of the recent administration of bicarbonate. The patient was then extubated and taken to the cardiac intensive care unit. Before removal, we again aspirated the epidural catheter with a smaller syringe, and at this time we noticed a free return of blood. Repeated ECG in the first 24 h after the cardiac arrest revealed sinus rhythm and no evidence of Wolff-Parkinson-White syndrome. The QT interval appeared borderline normal, but the corrected QT was constantly increased.

1. **Would you do further investigations at this point? If so what would you check?**

The plasma concentration of bupivacaine 2 min after the cardiac arrest was 0.48 μg ml⁻¹

1. **What caused the cardiac arrest in this patient?**
DISCUSSION

Intraoperative cardiac arrest is a rare event. The most recent survey on anesthesia-related cardiac arrest in children (POCA Registry) (1) reports an incidence of 1.4 per 10,000 cases. Thirty-two per cent of these episodes are secondary to cardiovascular problems and only 3% are attributable to arrhythmias. The reported incidence of arrhythmias after inadvertent systemic injection of local anesthetic following intravascular migration of an epidural catheter is only 0.4% (2). It is well known that the methods commonly used to detect systemic injection of local anesthetic, such as aspiration and return of blood, and evaluation of the heart rate and T-wave changes after a test dose, can fail (3-4), as documented in this case report. Fisher and colleagues (5) showed that, even after combining detection of changes in T-wave amplitude (increase by 25%) with any alteration in the rhythm, it is possible to miss 3% of inadvertent systemic injections. We do not know why the initial test dose did not result in any significant hemodynamic or ECG changes in our patient. There is a possibility that the initial dose of epinephrine (10 μg), although within the recommended range in children (0.25±0.5 μg kg⁻¹) (5) was too low to trigger any significant change. It is also possible that the epidural catheter migrated after the patient was repositioned on the operating table. Bupivacaine-induced arrhythmias include PR and QT interval prolongation, QRS widening, arterio-venous block, ventricular tachycardia and ventricular fibrillation. We observed most of these electrocardiographic changes in our patient. Because of the short interval between the appearance of arrhythmias and the initial bolus of bupivacaine, we immediately suspected an intravascular injection of the local anesthetic and did not administer further doses of bupivacaine. However, the bupivacaine concentration immediately after cardiac arrest was within normal limits. These plasma concentrations were significantly lower than those associated with neuro- and cardio toxicity in adults (6). The patient received only bupivacaine 0.25 mg kg⁻¹, and several studies in healthy adults have shown that the i.v. administration of higher doses of bupivacaine (30±45 mg) over a short period (5±10 min) is well tolerated and followed by minimal cardiovascular effects (7-8). The subsequent discovery of a prolonged QT interval in this patient could explain the cardiac events. They could have been triggered by the small dose of epinephrine (15 μg given in conjunction with bupivacaine) after the epidural catheter had probably migrated intravascular. Intraoperative torsade de pointes have been described in a child with LQTS after injection of a small dose (4 ml) of epinephrine (1:100,000) by infiltration for hemostasis (9). Arrhythmias in patients with LQTS are often triggered by physical exertion, emotional events or situations characterized by a high sympathetic discharge. Epinephrine is one of the drugs that should be avoided in patients with LQTS, and the treatment of this syndrome is mostly based on anti-adrenergic agents, such as b-blockers. We suspect that the accidental intravascular injection of epinephrine, which was combined with bupivacaine, after migration of the epidural catheter, may have triggered the arrhythmia. Long QT syndrome is a congenital or acquired disorder characterized by abnormal prolongation of ventricular repolarization, measured as lengthening of the QT interval on any of the 12 ECG leads (10). Other ECG abnormalities include bradycardia, increased QT dispersion and T-wave alterations. These abnormalities cause predisposition to ventricular tachyarrhythmia, such as polymorphic ventricular tachycardia and ventricular fibrillation. Acquired QT prolongation is most often attributable to the administration of drugs or electrolyte imbalance. There are two
known congenital forms of LQTS, the Romano-Ward syndrome (autosomal dominant trait) and the Jervell and Lange-Nielsen syndrome (autosomal recessive inheritance, associated with congenital deafness). The pathophysiology of cardiac arrhythmias in these patients is related to impairment of outward potassium currents or to defective sodium channel activation. Both mechanisms result in reduced outward current during repolarization, with secondary prolongation of cardiac action potentials and lengthening of the QT interval. A screening ECG in every child undergoing general anesthesia seems inappropriate, given the rarity of LQTS and the high incidence of a normal ECG at rest in patients with this congenital disorder. However, because of the potential for intravascular injection of the mixture of local anesthetic and epinephrine during any regional anesthesia technique, anesthetists should consider the risks involved in performing central and peripheral nerve blocks in patients with known LQTS or a family history of LQTS. If it is decided to use a regional technique, anesthetists should probably use a solution containing plain anesthetic rather than a combination of local anesthetic and epinephrine. In conclusion, this case report confirms the difficulty of recognizing the intravascular migration of an epidural catheter. Other reasons besides local anesthetic toxicity may be responsible for intraoperative cardiac arrest, and a full cardiac evaluation should be obtained in such cases.

REFERENCES

9) Richardson MG, Roark GL, Helfaer MA. Intraoperative epinephrine-induced torsades de pointes in a child with long QT syndrome. Anesthesiology 1992; 76: 647±9