

Inadvertent infusion of potassium chloride via an epidural catheter

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Abstract : A 73-year old man underwent a segmental liver resection for a solitary liver metastasis from a rectal carcinoma. On post-operative day one, an accidental potassium chloride infusion (total 29 mmol or 1135 mg of KCl) was given via the epidural catheter. Within a few hours this resulted in pruritus, progressive muscle spasms, decreased consciousness and vegetative symptoms such as tachycardia and hypertension. Subsequently respiratory insufficiency developed, necessitating intubation and ventilation of the patient with admission to the Intensive Care Unit. The patient received a single dose of 40 mg of dexamethasone intravenously to prevent or decrease possible myelum edema, and 100 ml.h⁻¹ of NaCl 0.9% infusion over the epidural catheter for several hours. The patient made a complete recovery, was extubated successfully six hours after ICU-admission and discharged home free of symptoms.

Key words : Anesthesia ; regional ; epidural ; complications ; potassium.

INTRODUCTION

In recent years the use of epidural catheters for peri-operative analgesia and anaesthesia has increased steadily. Despite the fact that responsible personnel is trained in using epidural catheters, inappropriate infusions administered via an epidural catheter are reported incidentally.

In this case report we discuss symptoms and therapy of inadvertent epidural administration of potassium chloride, and give recommendations to prevent situations as described.

CASE

A 73-year old man (height 1.59 m, weight 58 kg, BMI 23, ASA score 2) underwent a segmental liver resection for a solitary liver metastasis from a rectal carcinoma. Eleven months earlier he was diagnosed with a fixed rectal carcinoma, for which he received a deviating colostomy and combined radiotherapy and chemotherapy. Four months later

he underwent a low anterior resection in another hospital with a colostomy and intra-operative radiotherapy. Post-operatively the patient had three brief cardiac arrests, for which he needed resuscitation. He recovered without further sequelae. Besides his oncological history, he was treated for arterial hypertension with atenolol, and benign prostate hyperplasia with tamsulosine.

As agreed pre-operatively, at the start of the segmental liver resection an epidural catheter was instituted at Th4-Th5, through which a continuous infusion of 8 ml/hr of bupivacaine 0.125% and sufentanil 1 µg/ml was administered, after a test dose of 4 ml of bupivacaine 0.25% plus adrenaline 1/200,000. Post-operatively, he was admitted to the Medium Care Unit. The following morning at 7 AM, the time of the nursing shift change, the patient was found in a confusing, unresponsive state. Checking the pumps, the nurse discovered that the syringe for intravenous potassium chloride infusion and the one for the epidural analgesia administration had been switched. The epidural potassium chloride infusion was immediately stopped. At that time, the patient had been administered a total of 29 mmol of 7.45% potassium chloride epidurally (at a rate of 5 mmol.h⁻¹ = 5 ml.h⁻¹). The patient showed an altered mental state, and made uncontrollable, scratching movements over his thorax and arms. His vital signs at that moment were normal : peripheral oxygen saturation 99%

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(one l.min⁻¹ oxygen administered via nasal prongs), blood pressure 140/55 mm Hg, heart rate 85 bpm. The serum potassium, measured one hour earlier (four hours after the inadvertent epidural potassium chloride infusion) was 4,2 mmol/l (reference : 3,5-5,0 mmol/l). His blood pressure and heart rate increased the following minutes to 225/100 mm Hg and 130 bpm respectively. His legs showed a marked marble state and felt cold on palpation. He developed progressive muscle spasms of his legs and lower back. His mental state altered to unconsciousness. An episode of bradycardia (heart rate 55 bpm) and hypotension (measured non-invasively of 85/45 mm Hg) developed, followed shortly by a snoring respiration, necessitating intubation and monitoring of the patient at the Intensive Care Unit. Subsequently performed laboratory tests showed the following results (normal values between brackets) : hemoglobin 6.1 mmol/l (8.5-11.0) ; C-reactive protein (CRP) : 76 mg/l (< 8) ; leucocytes : 14.1×10^9 (4.0-10.0) ; thrombocytes : 319×10^9 (150-400) ; Na : 140 mmol/l (135-145) ; K : 3.6 mmol/l (3.5-5.0) ; Cl : 110 mmol/l (100-105) ureum : 3.5 mmol/l (3.0-6.3) ; creatinine : 96 μ mol/l (50-105) ; albumin : 21.9 g/l (35-50). An arterial blood gas analysis showed a severe metabolic acidosis : pH : 6.96 ; PO₂ : 172 mmHg (65-95) ; PCO₂ : 35 mmHg (35-45) ; bicarbonate : 8 mmol/l (21-28) ; base excess : -23 mmol/l (-2-2) ; arterial oxygen saturation : 98 % ; corrected anion-gap : 31.1 mmol/l. Serum lactate was 17.8 mmol/l (0.63-2.44). An electrocardiogram was run, showing sinus rhythm, with a partial LBBB and first grade AV-block.

After discovering the error, prompted by the disturbing symptoms, and because of lacking knowledge about this problem, a literature search was undertaken (www.ncbi.nlm.nih.gov). Based on the case report by KULKA *et al.* (5), our patient was treated with a single intravenous dose of dexamethasone 40 mg in an attempt to prevent or decrease any potential myelum edema. An hourly epidural infusion of 100 ml of NaCl 0.9% was started and given for eventually six hours, to dilute the hypertonic potassium-solution, possibly still present in the epidural space. The blood gas analysis (metabolic acidosis) was interpreted as being the result of intensive muscle spasms. To correct the severe metabolic acidosis (pH 6.96), an IV bolus dose of 100 ml of sodium bicarbonate 8.4% was administered, which improved the arterial blood gas analysis two hours later to a pH 7.23, PCO₂ 34 mmHg and bicarbonate 13 mmol/l. Serum lactate content decreased from 17.8 to 2.03 mmol/l.

The patient progressively regained consciousness. The marble aspect of his lower extremities diminished rapidly within a few hours. The patient was able to be extubated successfully six hours after admission to the Intensive Care Unit and made a complete recovery.

DISCUSSION

Epidural administration of local anaesthetics, with or without opioids, is a method of pain relief that is increasingly used in the peri-operative and (chronic) pain relief setting. In most Dutch hospitals a combination of two drugs is used, e.g. bupivacaine 0.125% and sufentanyl 1 μ g/ml. Wrongful solutions may be administered accidentally via epidural catheters, even by experienced staff nurses. Errors like in this case can be caused by similarity between ampoules (considered a "problem inherent to the equipment"), lack of attention, lack of correct storing of similar ampoules in different places, and fatigue of the health care staff (11). Few case reports have been published concerning inadvertent infusion of potassium chloride via an epidural catheter, used in the peri-operative or ambulant pain relief setting (1, 5-14).

Our patient showed, after the epidural administration of a total of 29 mmol KCl 7.45% (1 mmol = 39.1 mg ; in total 1135 mg), signs and symptoms of toxicity : pruritus, agitation, muscle spasms, hypertension, tachycardia, a diminished mental state and respiratory insufficiency.

One of the first signs was pruritus, a consistent finding in accordance to the literature (5, 7, 9 ; see table 1), although pruritus is not a obligatory symptom (8, 10). Striking progressive muscle spasms of the lower extremities and lower back may have been due to the hyperosmolaric character of the potassium infusion (7.45% = 1752 mosmol/l), or to the directly irritating effect of the KCl-solution in the epidural space. In the published case reports, no uniform explanation of the aforementioned symptoms is provided. Furthermore, symptoms of hyperactivity (sympathetic vegetative symptoms, muscle spasms) (7) and hypoactivity (paralysis) (5) are being explained using the same physiological principles.

Literature does not allow a uniform physiological explanation. Increased extracellular potassium concentration at the myelum level can induce changes in resting potential, depolarization and repolarisation, resulting in sensory (pruritus) and motor (muscle spasms) symptoms. Painful

Table 1

Reported cases of inadvertent infusion of potassium chloride via an epidural catheter

Author	level	epidural administration	KCl dose (mg)	start of symptoms	pruritus	ventilation	epidural therapy afterwards	i.v. corticosteroids	recovery time
KULKA (5)	L2-L3	7,45% perfusion	2680	1 hour	yes	no	99 ml NaCl 0,9%.h ⁻¹	40 mg dexamethasone	12 h
LITZ 1 (7)	Th8-Th9	7,45% perfusion	2830	some hours	yes	no	no	no	3 h
LITZ 2 (7)	Th7-Th8	7,45% perfusion	745	?	no	no	no	no	6 h
DO NASCIMENTO (9)	L3-L4	9,55% bolus	960	30 min.	yes	yes ; pulmonary edema	no	200 mg hydrocortisone	24 h
LIU 1 (6)	Th12-L1	15% bolus	1500	some min.	no	yes ; exhaustion	100 mg methylprednisolone	10 mg dexamethasone	14 h
LIU 2 (6)	Th11-Th12	15% bolus	1500	some min.	no	yes ; exhaustion	100 mg methylprednisolone	10 mg dexamethasone	18 h
PEDUTO (10)	L3-L4	15% bolus	450	some min.	no	no	no	2 g hydrocortisone	4 h
VERCAUTEREN (14)	?	7,45% bolus	370	instantly	no	no	no	no	2 h
TESSLER (13)	L3-L4	15% bolus	1500	2 hours	no	no	no	10 mg dexamethasone	± 24 h
LIN (6)	?	0,2% perfusion	1500	10 min.	no	no	10 ml NaCl 0,9% one bolus	no	5,5 h
SHANKER (12)	?	11,25% bolus	2250						incomplete
VAN DEN BERG (1)	Th11-Th12	7,45% perfusion	895	2.5 hours	no	no	80 mg triamcinolone-acetonide	8 mg dexamethasone 1 g methylprednisolone	12 h
Our case report	Th4-Th5	7,45% perfusion	1135	some hours	yes	yes ; respiratory depression	100 ml NaCl 0,9%/.h ⁻¹	40 mg dexamethasone	6 h

? : information not available.

sensations are described in body parts caudally to the introduced epidural catheter (5, 7, 8, 10). LIU notes that painful sensations seem to adequately reflect the true position of the catheter tip, which can migrate caudally or cranially after insertion (8). Our patient did not complain about pain, possibly because of his agitation and altered mental state. However, pain is not an obligatory symptom, and can be diminished or absent, when potassium chloride is inadvertently used as a solution for an epidurally administered analgesic, and not as a pure potassium chloride solution, as in our patient (9, 14).

Studies have shown that cranial spread of epidurally injected morphine takes place in a rapid active flow after some hours of slow passive flow (2), which explains the vegetative sympathetic symptoms that have been reported in all previous patients. Possibly a further cranial spread of potassium can induce loss of consciousness and ventilatory depression, as was the case in our patient. The patients described in two other reports that needed mechanical ventilation, developed ventilatory insufficiency based on pulmonary edema (9) and exhaustion (8 ; see table 1).

The period of hypotension and bradycardia may be due to an overactive parasympathetic nervous system as a reaction to potassium at cervical level, although hypoxia following ventilatory depression cannot be ruled out as the pulse-oximeter was continuously being dislodged from the patients' finger due to his movements at that precise moment. This potential complication of potassium chloride infusion in the epidural space has not been reported up till now, nor has there been any allusion made in the literature on the extensive marbled skin on the lower extremities as seen here. The latter cannot be explained by a high arterial blood pressure and raised superior caval vein-venous oxygen saturation (SvO₂ 85%) as a sign of decreased tissue perfusion due to a decreased cardiac output. We believe this cutis marmorata is rather a sign of severe shunting due to extensive peripheral vasoconstriction, induced by a raised sympathetic nervous system activity due to direct stimulation at the spinal level. Soon after the induction of adequate therapy, the marbled skin disappeared.

We explained the severe metabolic acidosis (pH 6.96 ; lactate 17.8 mmol/l) by anaerobic

metabolism during the temporary but intense muscle spasms at the lower extremities and lower back. After IV administration of 50 mg rocuronium prior to intubation, the muscle spasms disappeared. They did not reappear after the effect of rocuronium wore off. A few hours after initiating IV dexamethasone therapy and administration of 100 ml.h⁻¹ NaCl 0.9% via the epidural catheter, the lactate concentration resumed to normal levels (2.03 mmol/l).

There is scientific evidence that high dose of intravenous corticosteroids can improve and reduce post-traumatic spinal cord injury (4). Steroids are potent lipid peroxidation inhibitors, an effect which is believed to be the primary therapeutic action in spinal cord injury (3, 4). No direct evidence exists that administering corticosteroids after potassium infusion via an epidural catheter has any positive prognostic effect. Liu assumes a beneficial effect of administering epidural corticosteroids in alleviating spinal cord edema and irritation (8). Others sufficed to give IV infusion of corticosteroids (5, 9, 10, 13). In accordance to the report by KULKA *et al.* (5), our patient received 40 mg of IV dexamethasone (see table 1).

Administering NaCl 0.9% via the epidural space is questioned by some authors (6, 7, 9, 13), as neuroplegia can occur even at a low dose of potassium, and diluting the epidural space content may result in raising the level of blockade (6).

In total our patient received 1135 mg of potassium in the epidural space, which is about comparable to the amounts (1500 mg) reported by LIU *et al.* (8) and TESSLER *et al.* (13). PEDUTO *et al.* supposed a correlation between the total amount of administered potassium and the lag time to neurological recovery (10). Meanwhile other reports rejected this presumption (5,7).

Based on the report by SHANKER *et al.* (12), it can not be ruled out that the concentration of the administered potassium solution has an effect on the subsequent recovery. SHANKER *et al.* were the only ones to report a patient, who did not recover completely after an inadvertent infusion of potassium chloride solution via an epidural catheter. The patient was paraplegic after an infusion of KCl 11.25%-solution (2250 mg), up till his death six months later (12). On the other hand LIU *et al.* (8) and TESSLER *et al.* (13) reported on patients receiving even higher doses of potassium chloride solutions (15%), who recovered without sequelae. The influence of the administered concentration on the final recovery thereby remains illusive. VAN DEN BERG *et al.* (1) in their report suggested that administering potassium chloride solutions by a perfusor

possibly is less harmful and leads to quicker recovery than a bolus administration.

Different measures to reduce the risks of inadvertent administration of infusion substances via an epidural catheter have been proposed. Double checking by another nurse, using color codes and different colors for different solutions, the use of larger syringes (requiring less changes, visible differences with smaller syringes for intravenous use) and specific connections have all been suggested and implemented to various degrees, although no study has proven its efficacy. We believe that the solution to the problem is to produce a specific epidural delivery system, different from the intravenous delivery system, containing lines, syringes, connecting tubes, etcetera. A comparable system already exists for feeding tube material.

CONCLUSION

Inadvertent infusion of a potassium chloride solution via an epidural catheter is accompanied by a complex of symptoms, in which vegetative sympathetic symptoms as hypertension, tachycardia and vasoconstriction predominate. Besides these symptoms, signs of pruritus, sweating, muscle spasms and even loss of consciousness and respiratory insufficiency have been reported.

Treatment may consist of intravenous and/or epidural administration of corticosteroids to diminish or prevent spinal cord edema and/or an epidural saline infusion to dilute the potassium in the spinal canal. Whether this administration speeds up the recovery time remains unproven. However, complete recovery seems to be the rule, rather than the exception.

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