Systemic Toxicity And Satisfactory Analgesia Following Epidural Injection

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SUMMARY

A 60-year-old male patient who had severe acute systemic toxic reaction as well as satisfactory epidural analgesia and motor blockade in the lower extremities following a single dose epidural anaesthesia for transuretral resection of prostatic carcinoma, has been presented and possible causes have been discussed. In this case rapid absorption of the local anaesthetic in an area rich in blood vessel, lacerated veins and distention of epidural veins due to the secondaries in surrounding tissues may have contributed to the occurrence of systemic toxic reaction. Systemic toxic reaction without convulsions may be confused with total spinal block, but the initial supportive treatment aimed at maintaining vital functions is identical in both situations. Although an alarming complication, systemic toxic reaction following epidural injection can be diagnosed and treated satisfactorily with close observation of the patient and taking the necessary precautions and therapeutic measures.

Key words: Anaesthetic techniques; epidural anaesthesia, complications, vessel puncture, systemic toxicity

INTRODUCTION

One of the serious complications associated with epidural anaesthesia is accidental intravascular injection through a Tuohy needle or epidural catheter which may give rise to systemic toxicity of the local anaesthetic injected or incomplete analgesia (3, 7, 11, 13). We report a case in which acute systemic toxicity as well as satisfactory analgesia and motor blockade resulted following a single dose epidural injection.
sue invasion by carcinoma and osteoblastic metastases in pelvic bones and head of femur. There were oc-
tal nodal extrasystols and left atrial dilatation in
Single dose epidural anaesthesia was planned for the procedure and the patient was premedicated with atropine 0.5 mg and diazepam 10 mg. An intra-
venous infusion of dextrose in saline was started and blood pressure and pulse rate were monitored. In the right lateral position a 17G Tuohy needle was
inserted through L 34 space and epidural space was identified using hanging drop sign. Before the epidural space was entered blood was seen coming through the hub of needle. But it disappeared when the needle was rotated and advanced further. After careful aspiration, 20 ml of 1.5 per cent Prilocaine (Citanest) was injected slowly. The patient had diff-
iculty in speaking and breathing as soon as he was turned on his back, but was fully conscious. Hundred per cent oxygen was given with a face mask, there
was no cyanosis or convulsions. Blood pressure was 120/80 torr and pulse rate 90 per min. A total spinal block was thought possible at this stage. Five minutes later respiration stopped altogether and the patient could be ventilated easily. Ten minutes later he lost consciousness. Meantime frequent blood pressure and pulse rate recordings did not show any change. The patient was relaxed as if given a muscle relaxant. To 
facilitate ventilation trachea was intubated and there was no reaction to either laryngoscopy or the tracheal tube. Thirty minutes after the injection of local anaesthetic, blood pressure started to fall and pulse rate increased. The lowest systolic blood pressure recorded was 70 torr and the highest pulse rate 150 beats per min. with occasional ventricular beats. In-
creased intestinal peristalsis and incontinence were noticed. There was no sign of Claude-Bernhard-Horner syndrome. In fact pupils were dilated after the
insertion of local anaesthetic and that ventilation and sup-
port of circulation are vital elements in recussitating patients with systemic toxicity. In our case early 
recognition of the complication and establishment of a definite diagnosis is not essential at the beginning since the initial therapy is identical regardless of the cause.
Prilocaine (Citanest) is known to have rapid on-
set, good spread and a wide therapeutic index (12), and the dose of 300 mg used in this patient is smaller than the recommended dosage (9). Unusual in our case was the systemic toxic reaction without con-
volutions as well as satisfactory regional analgesia and motor blockade as determined when the patient regained consciousness. Establishment of sensory anaesthesia at T6 level excludes the possibility of a massive extradural or total spinal block. Inadvertent intravascular injection of the entire 20 ml was also not possible due to the fact that satisfactory analgesia and motor blockade resulted following the injection and lasted three hours.
Basically the signs and symptoms of a systemic reaction due to the high blood level of the local anaesthetic drug are firstly central nervous system stimulat-
ion and secondly depression following over-
stimulation. Nevertheless sometimes toxic reactions do not follow this pattern. In 14 major reactions attributed to pontocaine there was no early sign of stimulation, instead they showed drowsiness, shallow respiration, loss of consciousness and apnea (8) which is a similar course seen in our patient. De Jong et al. (5) report that well ventilated animals can survive at least twice the convulsant dose of any amide local anaesthetic and that ventilation and support of circulation are vital elements in recussitating patients with systemic toxicity. In our case early recognition of the complication and establishment of artificial ventilation may have prevented cerebral hypoxia and perhaps occurrence of convulsions.
Return of spontaneous respiration and conscious-
ness with 60 and 70 minutes respectively took longer than reported by Ryan (11) (2 minutes) and Moore

DISCUSSION

Serious complications of epidural anaesthesia in-
clude systemic toxic reactions to local anaesthetic
drug, total spinal or massive extradural block (1,4,6).
Acute systemic toxicity may result from rapid absorrption in a vascular area or from accidental intra-
venous injection through an epidural needle or cath-
ether (10,11). Lacerated or open veins have also been
suggested as a cause of failure or generalized reaction with epidural anaesthesia (7). Incidence of either
direct vessel puncture with Tuohy needle or of
venous cannulation with the catheter has been re-
ported as 1.7 (4) and 1 per cent (2, 15). It is of course difficult to say, whether, when blood is seen dripping from the hub of the needle the point has in fact reached the extradural space or has merely hit a blood vessel on the way. Systemic toxic reactions to local anaesthetic drugs characterized by respiratory and circulatory depression without convulsions may be confused with a high or total spinal anaesthesia. But the establishment of a definite diagnosis is not essential at the beginning since the initial therapy is identical regardless of the cause.
Steinhaus and Howland (14) state that suppression of reflexes by intravenous lidocaine is striking. Using the endotracheal tube as a predictable and effective cough stimulus they found that cough reflex can be completely suppressed. In our case also there was no reaction to either the endotracheal intubation or the movement of the tube for at least 50 minutes.

Late changes in blood pressure may have been due to the direct effect of the local anaesthetic agent on blood vessels which responded to vasopressor therapy and fluid loading.

In conclusion: Systemic toxic reaction to local anaesthetic drug as well as satisfactory analgesia and motor blockade may result following epidural anaesthesia. In the case presented here rapid absorption in an area rich in blood vessel, lacerated veins and distention of epidural veins due to the secondaries in surrounding tissues may have all contributed to the occurrence of systemic toxic reaction.

REFERENCES