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# Systemic Toxicity And Statisf actory Analgesia Following Epidural Injection

EPİDURAL ENJEKSİYON SONRASI ORTAYA ÇIKAN SİSTEMİK TOKSİK REAKSİYON VE YETERLİ EPİDURAL ANALJEZİ

Zeynep ESENER

Ondokuzmayıs Üniversitesi Tıp Fakültesi Anesteziyoloji Anabilim Dalı, Samsun

#### 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 transurethral resection of prostatic carcinoma, has been presented and possible causes have been discussed. In this case rapid absorbtion 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 aimedat 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

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### 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.

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#### ÖZET

Transuretral rezeksiyon için tek doz epidural anestezi uvgulanmasını takiben siddetli sistemik toksik reaksiyon gelişen prostat karsinomalı 60 yaşında bir hasta sunuldu ve ohsı nedenler tartışıldı. Toksik reaksiyonun tedavisinden sonra hastada aynı zamanda yeterli epidural analjezi ve motor blok geliştiği izlendi. Bu olguda, zaten damardan zengin olan epidural alana enjeksivon vapılmış olmasına ek olarak muhtemelen işlem sırasında lasere olan ve/veya bölgede mevcut metastazlar nedeniyle genişlemiş olan venlerden lokal anesteziğin hızla ve büyük miktarda sistemik dolaşıma geçmesi bu komplikasyonun ortaya çıkmasına yol açmış olabilir. Konvülsiyon olmaksızın ortava cıkan toksik reaksivonlar total spinal blokla karıştırılabilmektedir. Ancak vital fonksiyonların devamını sağlamaya yönelik destekleyici tedavi her iki durumda da yeterli olmaktadır. Endişe verici bir komplikasyon olmasına karşın, sistemik toksik reaksiyon hastanın başlangıçtan itibaren yakından izlenmesi, gerekli önlemlerin alınması ve terapötik girişimlerle yeterli şekilde tedavi edilebilmektedir.

Anahtar kelimeler: Anestezi yöntemleri, epidural anestezi, kornplikasyonlar, sistemik toksisite, damar zedelenmesi

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#### CASE REPORT

A 60 year old, 68 kg, 172 cm and reasonably well nourished male patient was admitted to hospital for transurethral resection of prostatic carcinoma causing urinary obstruction. On physical examination he had a blood pressure of 140/90 torr, a pulse rate of 80 beats per min., a respiratory rate of 14 per min, and the temperature was 36.7 C orally. He had a normal chest x-ray, but pelvic graphy showed soft tissue invasion by carcinoma and osteoblastic metastases in pelvic bones and head of femur. There were octal 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 intravenous 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 difficulty 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. Increased intestinal peristalsis and incontinence were noticed. There was no sign of Claude-Bernhard-Horner syndrom. In fact pupils were dilated after the spontaneous respiration stopped until blood pressure was restored. Hypotension was treated with fluid loading and vasopressor injection, twice 12.5 mg ephedrine and urgent digitalisation was started with Cedilanid 0.8 mg. Sixty minutes later, spontaneous respiration started and 10 minutes after that he could not tolerate the endotracheal tube and was extubated. At this time he was conscious and cooperative. A T6 level of sensory anaesthesia and motor blockade of lower extremities were observed Operation was postponed to follow the recovery of the patient from the effects of the local anaesthetic. On transfer to recovery room ECG showed sinus rythm at a rate of 90 beats per min. and the patient was fully conscious, alert and cooperative. The duration of the block to complete recovery of motor and sensory function was 180 minutes and there was no residual effect of the event.

#### DISCUSSION

Serious complications of epidural anaesthesia in-

elude systemic toxic reactions to local anaesthetic drug, total spinal or massive extradural block (1,4,6). Acute systemic toxicity may result from rapid absorbtion in a vascular area or from accidental intravenous injection through an epidural needle or catheter (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 reported 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.

Prilocaine (Citanest) is known to have rapid onset, 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 convulsions 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 stimulation and secondly depression following overstimulation. 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 artifical ventilation may have prevented cerebral hypoxia and perhaps occurence of convulsions.

Return of spontaneous respiration and consciousness with 60 and 70 minutes respectively took longer than reported by Ryan (11) (2 minutes) and Moore

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(8) (30-45 minutes). 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 absorbtion 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 occurence of systemic toxic reaction.

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