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Spinal myoclonus following spinal anesthesia for caesarean section

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ABSTRACT

A primi woman presented with preeclampsia at 39 weeks gestation. Spinal anaesthesia was employed for Caesarean section. A day after delivery she developed twitching of her legs, that was initially thought to be post-partum eclampsia but was subsequently diagnosed as spinal myoclonus. She was treated with diazepam, carbamazepine with improvement over the next 4 days, and discharged home. Her symptoms resolved completely 15 days after the initial event.

Keywords: *Spinal Myoclonus, Spinal anaesthesia, Lower section Caesarean section*

INTRODUCTION

Myoclonus comprises sudden, involuntary contractions of a group of muscles, a single muscle, or part of a muscle. Spinal, focal or segmental myoclonus has features that distinguish it from other forms of more generalized myoclonus. The common causes include tumor, infection, trauma and degenerative processes[1]. Swansonin his extensive report of 67 cases of myoclonus has classified them mainly as segmental and generalized.

CASE REPORT

A 25-year-old primi with no previous or family history of epilepsy, presented to our delivery suite at 39 weeks gestation because of an episode of hypertension and proteinuria over the previous fortnight. On admission, her blood pressure was 140 / 90 mmHg and all other parameters were normal. Due to an abnormal fetal cardiotocograph, a decision was made to deliver her by lower segment Caesarean section. Spinal technique was chosen for anaesthesia. The operation proceeded uneventfully and a live male infant weighing 2.6 kg was delivered. All other surgical findings were unremarkable and routine antibiotic prophylaxis was administered. The following day the patient complained of a severe frontal headache that was not postural, and that resolved with rest and simple analgesics. Her blood pressure was 150/90 mmHg and she was given 10 mg slow release nifedepine orally. Her subsequent recovery was unremarkable, with her blood pressure controlled by10 mg slow release nifedepine orally, twice daily. A day after the delivery, she suddenly developed a tingling sensation in her legs, with some twitching predominantly affecting her both legs. This was immediately followed by headache, occurring every2–4 minutes and lasting up to 10 seconds.

Throughout these events, she was conscious and orientated, though understandably very anxious. Her fundi were normal and reflexes were all present and brisk, and there were no focal neurological signs. Her blood pressure had risen to 180 / 100 mmHg. She was apyrexial and all her blood tests including serum electrolytes, clotting time and uric acid were found to be within normal limits. The major concern at the time was that of postpartum eclampsia, and she was given 10 mg sublingual nifedipine to control the hypertension and 10 mg diazepam i.v., which had no effect on her involuntary movements. She was transferred to the delivery suite for more intensive monitoring and the on-call anaesthetist was contacted. A further 10 mg diazepam was given. In the light of her completely orientated state, with absent focal neurological signs and lack of response to benzodiazepines it was concluded that the fits were

not of post-partum eclampsia. An MRI of the brain and lower thoracic lumbar spine was performed the following morning, and no abnormalities were reported. As the twitching movements were still continuing, we made a clinical diagnosis of spinal myoclonus. She was commenced on 200 mg carbamazepine orally, twice daily, supplemented with 10 mg diazepam. Improvement in the frequency and number of the twitches occurred in her left leg in 2 days and the right leg over next 2 days. She was eventually discharged home a week later.

DISCUSSION

The first recorded case of clinical myoclonus dates back to 1881, when Friedreich described a sporadic case of paramyoclonus multiplex' (myoclonus simplex), consisting of diffuse myoclonus of the trunk and extremities[3]. The prognosis in these patients was clearly better than in those with concomitant seizures and mental changes as described by others[4]. There are a few cases of myoclonus reported in the literature following spinal anaesthesia[5-7]. Fox et al. reported a 57-year-old woman who developed myoclonus of her legs 7 hours after undergo ingcutaneous ureterostomy under spinal anaesthesia with heavy tetracaine in 10% dextrose with epinephrine[6]. Her symptoms diminished considerably with intravenous diazepam but did not disappear completely, though the patient recovered fully within 24 h. Swanson et al. coined the term benign essential myoclonus', reserved for those patients with transient or persistent symptoms without anyneurological features[2]. Drug-induced neurotoxicity is another possibility[8]. In an extensive comparative study, Yamashita et al. observed the effects of intrathecaltetracaine, lidocaine, bupivacaine and ropivacaine, and found the least degree of vacuolation of the dorsal funiculus with ropivacaine[8]. The most striking feature in this case was myoclonus, which was largely limited to the legs. The histological changes confirmed the presence of an inflammatory lesion confined to the grey matter, particularly in the dorsal and lumbar region. Opioid induced myoclonus was studied extensively in animals[9,10]. Our patient developed spinal myoclonus of the both limbs in the puerperium.

Although she had suffered from pre-eclampsia, a diagnosis of eclampsia was ruled out on the basis of a lack of focal neurological signs, a normal orientated state and normal biochemical markers. Viral my elitis or bacterial infections are unlikely as the patient was a pyrexial with a normal white cell count. The cause of her my oclonus therefore remains unsolved. However, the spinal cord during the spinal anesthesia may have resulted in transient sub-acute spinal neuroneitis. There was transient paraesthesiain the legs. It was postulated that the inhibitory effects of the local anesthetic might have led to heightened irritability of the α-motor neurons, leading to myoclonus. This suggests thatthe myoclonus was initiated by spinal cord disease. Electromyography would have been an ideal diagnostic tool but was impractical at thatlevel. Spinal myoclonus is remarkably sensitive to medical therapy with clonazepam, sodium valproate and carbamazepine. Our patient recovered completely from the spinal myoclonus with no residual neurological deficit and hence her prognosis appears to be excellent.

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