



Neurological Complication After Spinal Anaesthesia: A Case Report

Fagbohun Omolola^{1,*}, Opara Chukwuma²

¹Department of Anaesthesia, Lagos State University College of Medicine and Teaching Hospital, Ikeja, Nigeria

²Department of Obstetrics and Gynaecology, Alimosho General Hospital, Igando, Nigeria

Email address:

lolaifagbohun@yahoo.com (F. Omolola)

*Corresponding author

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Abstract: Background: Neurological complications following spinal anaesthesia are rare and often transient. It is difficult to ascertain the aetiology in most cases although needle trauma and local anaesthetic toxicity has been implicated. Patient often present with abnormal sensation, reduced power in the affected limb, sensory impairment and unilateral movement disorders of the affected limb. It usually resolves within a short interval without any definitive treatment. However presence of a neurological symptom post sub arachnoid block could herald a disastrous neurological complication. We present the case of a 35 year old booked (G2P1) lady who had emergency caesarean section under spinal anaesthesia and developed foot drop and weakness in the left lower limb post surgery. We report this case to increase awareness on rare neurological complications such as foot drop following spinal anaesthesia which is hardly documented as a known complication of sub arachnoid block in our environment.

Keywords: Caesarean Section, Spinal Anaesthesia, Neurological Complications, Foot Drop

1. Introduction

Neurologic complications following neuroaxial blocks are rare with an incidence of 1 in 10,000 cases [1, 2] The most common complication is a reversible neuropathy due to direct needle/catheter trauma or intra neural injection of a local anaesthetic agent presenting as a radiculopathy involving a single spinal nerve root. [3] In a 2 year study by Scott and Tunstall neurological disorders were reported in eight out of 14,856 deliveries with spinal anaesthesia (0.054%), all of which were transient. [4] In another study, neurological disorders were reported in 24 cases, with a prevalence of 0.06% after spinal anaesthesia [5]. The aetiology of neurological deficit post spinal anaesthesia is usually unknown but foot drop has been associated with needle injury to the lumbosacral trunk and local anaesthetic toxicity. The symptoms of this neurological disorder include paraesthesia, sensory impairment and unilateral movement disorders of the affected limb. [6].

2. Case Report

Our patient was a 35 year old booked (G2P1) lady who was scheduled for emergency caesarean section on account of fetal macrosomia and postdatism. She weighed 65kg with a height of 1.8 meters. She was classified as American Society of Anesthesiologists physical status 1. The pre-anesthetic review was essentially normal and patient had no neurological deficit prior to surgery. All the investigations done including a full blood count, random blood sugar, electrolyte urea and creatinine and urinalysis were within normal limits.

Spinal anaesthesia was established at the first attempt with ease in the sitting position with a 25-gauge quincke spinal anaesthesia needle. The needle was introduced at the L3-L4 inter-space with a free back flow of clear cerebrospinal fluid observed. 2.8ml of 0.5% heavy bupivacaine was injected and patient was placed in the supine position. A sensory block level of about T6 was achieved. Patient did not complain of pain or abnormal sensation at the time of establishing spinal

anesthesia. She was hemodynamically stable intra-operatively and had about 2 liters of 0.9% normal saline infused. The estimated blood loss was about 800mls. She was delivered of a life female baby weighing about 3.2kg. Patient was able to move her lower limbs about 3 hours after surgery. However, about 12 hours after the cesarean section and 9 hours from recovering, the patient was noticed to have a high stepping gait and she complained of inability to move her left foot properly with an associated tingling sensation. On examination, power in the right lower limb was 5/5 and there was no sensory or motor deficit in the right lower limb while power in the left lower limb was about 3-4/5 and patient experienced difficulty dorsiflexing and plantar flexing the left foot while sensation remained intact. Lumbar magnetic resonance imaging (MRI) and electromyography (EMG) were requested for to identify site of lesion and possible pathology but both were not done as patient had financial constraints. Intravenous methylprednisolone was prescribed at a total dose of 500mg daily for 3 days along with vitamin B complex. By about 48 hours after surgery, patient had regained full power and sensation in the upper part of the left lower limb and the foot drop had improved considerably. The foot drop gradually resolved and was almost not noticeable by the 4th day post surgery when she was discharged. Patient returned for clinic visit a week after discharge without deficit in function of the left lower limb. Neurological examination was normal.

3. Discussion

Neurological complications following spinal anesthesia are rare and transient, with a prevalence of about 3.5%. [3] The aetiology of these complications are largely unknown but direct needle trauma, cauda equine syndrome and local anesthetic induced neuro toxicity have been suggested. [6] Several studies have suggested that patients who experience paresthesia or pain during spinal needle placement or patients in whom repeated attempts were made to locate spinal space are likely candidates for neurological complications after spinal anaesthesia. [7, 8] Needle size, type of needle and local anesthetic dose has been implicated in the development of neurological complications [7, 9]. None of the above aetiologies was likely to be responsible for the neurological complication in our patient. Although a size 25 gauge quincke spinal anaesthesia needle was used, caution was taken to gently introduce the needle to reduce the risk of trauma. The patient did not complain of pain or abnormal sensation which could indicate parasthesia while spinal anaesthesia was being established and the spinal space was easily located at the first attempt which precludes trauma from multiple attempts. Intense and long term hemodynamic changes are among the factors, which cause spinal cord ischemia and spinal artery thrombosis that could lead to neurological complications. [7, 10] In the index case, there were no specific hemodynamic changes observed during surgery or spinal blockade. Placing the patient in the lateral position for a prolonged period has also been

implicated as a cause of neuropathy. [9, 11] In the index report, spinal anaesthesia was established in the sitting position and the process lasted less than 10 minutes while surgery lasted less than an hour in the supine position with a left lateral tilt, hence patient was not in the lateral position for an unduly long duration that could have predisposed a nerve to injury. The onset of neurological complication and the rate and time of recovery from these complications and the extent of neurological damage vary among patient and are dependent on multiple factors. In a study by Auroy et al on 103,000 patients with spinal blockade, all neurological problems started within 48 hours, and recovery took from 2 days to 3 months. [5] In our case, the complication was detected about 9 hours after recovery from spinal anesthesia and the patient recovered fully within 10 days of the spinal anaesthesia.

When neurological deficit occurs after spinal anaesthesia, close differential diagnosis such as haematoma, nerve damage, space occupying lesions and others should be excluded. [8] MRI studies could have helped in identifying the pathology, the level of the lesion and in excluding close differentials but our patient could not afford the cost of the studies. Neurological deficit is usually treated with steroids and vitamin B6 [10, 12].

4. Conclusion

We report a rare incident of a 35 year old booked (G2P1) patient who exhibited neurological deficit after spinal anaesthesia. Neurological deficit after spinal anaesthesia is a rare and usually transient complication after spinal anaesthesia which should be promptly investigated and treated as its presence may herald worse neurological complications if ignored.

Declaration of Interest

There is no conflict of interest.

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