CASE REPORT

Severe flaccid paraparesis following spinal anaesthesia: a *sine materia* occurrenceGentian Vyshka,¹ Giuseppe Vacchiano²

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Accepted 18 April 2014

SUMMARY

We report the case of a 45-year-old female patient who underwent spinal anaesthesia for a minor urological intervention, trying to correct her stress urinary incontinence. Hyperbaric bupivacaine was intrathecally injected and the surgical intervention ended uneventfully. Six hours after the operation a severe motor deficit of lower limbs was noted. The situation persisted and neurologists repeatedly checked on the patient; imaging of the lumbar spine was made but no pathological signal was registered. Somatosensory evoked potentials of the lower limbs showed bilaterally delayed latencies of the cortical components, with prolonging of interpeak latencies, suggestive of a suffering of the long somatosensory spinal tracts. Unable initially to walk or stand in erect position with almost no movements in both lower limbs, the patient was admitted in a rehabilitation facility and treated there for 4 months, with her condition substantially improving during this period.

BACKGROUND

The history of spinal anaesthesia has been indelibly and unfortunately impregnated from the ill-fated procedure of 13 October 1947, when Albert Wolley and Cecil Roe became paraplegic in a UK hospital, after that the same anaesthetist injected them the same drug, through the same technique.^{1 2} This notorious case of two patients suing the physician and the Ministry of Health, but unable to receive compensation because an ad hoc theory of 'phenol contamination' and 'invisible cracks' in ampoules convinced the court to declare a non-guilty verdict, has been recently revisited. The 'phenol' theory has been in fact found as technically incorrect, with other explanations offered.³ Apart from aetiological background and hypothesis, the fact is that this complication, and others following, considerably restricted, for some decennia, the use of spinal anaesthesia and probably not only in the UK.²

Lumbar puncture is considered a safe procedure, with very few complications, especially in skilled hands. Nevertheless, there exist a rich casuistic with reports of adverse events, the majority of which are self-limited and benign in nature. This is true for lumbar puncture as a diagnostic procedure, but the situation might become much more complicated when drugs are administered intrathecally, such as in the case of spinal anaesthesia or for other medical reasons. The type of spinal needle might be significant as well, with Whitacre spinal needles presumably causing less frequent post-dural

puncture headache.⁴ Although pregnancy and especially uterine contractions result in increased venous pressure within the epidural venous system, spinal anaesthesia has become a routine procedure in obstetrical practice.⁵ The number of lumbar punctures applied is another technicality of importance, with epidural haematomas reported after numerous punctures and with anticoagulants increasing substantially the risk of such haematomas.⁶ Cerebrospinal fluid cutaneous fistulas have been occasionally reported as well following lumbar puncture.⁷

CASE PRESENTATION

We present the case of a Caucasian woman, aged 45 years, operated in a urological facility, because of stress urinary incontinence. The surgical intervention foresaw the insertion of a mini-sling through a single incision, and the procedure was due under spinal anaesthesia. The spinal anaesthesia was performed with a Whitacre spinal needle gauge 25 (25 G), and 12 mg of Marcaine (Astrazeneca), containing hyperbaric bupivacaine (0.50%), were injected. Anaesthesia was performed from an experienced anaesthesiologist, with the patient in left lateral decubitus, and with its onset registered almost immediately after the spinal injection of hyperbaric bupivacaine in the L5-S1 interspace level. Marcaine used was checked prior to injection for the expiry date, and resulted in order. All precautions for its storage were taken into consideration and no complications were seen in the medical facility related with its use thereafter. A single puncture was made, with clear flow of cerebrospinal fluid seen prior to the injection of bupivacaine. An upper sensory blockade up to the level of 10th thoracic dermatome (T10) was achieved, whereas the lowest level noted was at the fifth sacral dermatome (S5). No paraesthesia or radicular pain was referred from the patient during spinal injection of the anaesthetic drug. The 20 min-long surgical procedure was performed in the lithotomy position in hyperflexion, with the thighs bent back on the abdomen at an angle of 120°, a position described from Delorme *et al*,⁸ and successively considered as optimal for the placement of MONARC slings.

The woman did not refer any serious medical condition in her history. Slightly obese, with a body mass index of 27.5 (her body weight was 74 kg with a height of 164 cm), the patient was rather chronically constipated, and is a heavy smoker with 30 cigarettes daily since more than 15 years. She had an appendectomy at the age of 15 years and at

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To cite: Vyshka G, Vacchiano G. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202071

Figure 1 Normal signal of lumbar spine and paraspinal structures was seen in all sequences (fluid-attenuated inversion recovery, T1 and T2—weighted) of MRI.

the age of 31 years the total asportation of a left breast fibroadenoma. The patient was never subjected to neurological therapies and had no specific symptoms, and excluded gait difficulties or lumbar symptomatology of whatsoever nature, prior to the present episode. The intervention, planned for a 1-day surgery, started at 13:00 and was concluded at 13:20. According to the surgical file the entire procedure was considered 'uneventful'. The patient received as preoperative medication one ampoule of Atropine sulfate (1 mg of active drug, 25 min before intervention). Anaesthesia itself started as well 20 min before the surgical incision, with the patient in stable haemodynamic situation and with a systolic blood pressure of 120 mm Hg and a diastolic value of 80 mm Hg.

Approximately 6 h after the operation was over, an urgent consultancy was requested with the anaesthesiologist and with a neurologist, due to the somnolence of the patient and hyposthenia of lower limbs. At that moment slightly hypotensive blood pressure values were registered, with a systolic pressure of 80 mm Hg and a diastolic pressure of 60 mm Hg. A CT scan of the lumbar spine showed no changes, however corticoids for their antioedematous effect were administered (intravenous dexamethasone 8 mg), with slight improvement of the motor deficit. Further imaging studies followed the second and the fourth day with two consecutive MRI of the lumbar spine; none of those suggested any pathological finding.

The patient was admitted in a neurointensive care unit the next morning after the intervention, for a period of 4 days. Neurosurgical opinion was requested, but the necessity for a lumbar spine surgery was ruled out. Dexamethasone was continued for 1 week after the discharge with the dose tapered to 4 mg before being completely stopped. During all 4 days she was again constantly normotensive. Two weeks after being discharged from the neurointensive care unit, the patient was admitted in a rehabilitative facility, where a physiotherapeutic treatment took place for almost 4 months. On admission at the

rehabilitative facility, a total absence of every motor activity at her right lower limb was registered; otherwise a slight ability to flexion-extension of the toes was seen at her left lower limb. The patient was completely unable to walk or to stand independently. The diagnosis of a flaccid severe paraparesis following spinal anaesthesia, of unknown origin, was made.

INVESTIGATIONS

MRI of the lumbar spine showed no obvious signs of pathology, in all sequences (figure 1).

Owing to the persistence of the severe paraparesis, somatosensory-evoked potentials (SEP) were registered in both lower limbs, with following results (table 1).

The latencies of SEP components evoked over the tibialis posterior nerve were consistently prolonged, and even absent when registered at the cervical region. Abnormal electrical findings were more advanced over the right lower limb. The interpeak latency between the N22 component (registered over the iliac crest ipsilateral to the nerve stimulated) and the P37 component (cortical) suggests an abnormality rostral to the lumbar cord.⁹ Such electrical findings suggested therefore a severe suffering of the somatosensory long spinal tracts, slightly more important in the right side. Owing to obvious lack of pathological changes in two MRI studies, the hypothesis of a neurotoxicity effect of the anaesthetic drug (bupivacaine) was formulated, trying to explain the persistent flaccid paraparesis of the patient.

TREATMENT

After the treatment with corticoids immediately on installation of the paraplegia, the patient was transferred within 2 weeks to a neurorehabilitation facility. The 4-month rehabilitative programme was a complex one, aiming at prevention of the vicious postural sequelae, and towards the enhancement of motor ability and a better coordination of the lower limbs vis-à-vis with the gradual regain of the independent erect standing.

Table 1 Components of SEP, lower limbs

Nerve where SEP elicited	Component registered at the iliac crest	Components registered at C5s-Fz	Components registered at Cz-Fz	Interpeak latency
Right posterior tibialis	N22=31.0 ms	No response	P37=59.90 ms N45=77.90 ms	N22-P37=32.50 ms
Left posterior tibialis	N22=27.4 ms	No response	P37=56.60 ms N45=72.70 ms	N22-P37=28.40 ms

Legend: N22—the second prominent peak representing activity of the dorsal horn of the lumbar spinal cord. P37 and N45—respectively, prominent positivity and negativity waves, registered with a midline central montage (Cz-Fz), corresponding to the cortical generator for the lower limb somatosensory-evoked potentials (SEP). C5s—spine of the fifth cervical vertebra. Cz-Fz—registering scalp midline sites according to the International 10–20 System.⁹ Numbers after the capital letters respective to the evoked responses (such as 22 after N in N22; 37 after P in P37) represent the optimal latency (ie, 22 ms; 37 ms) as registered in standardised conditions, and in healthy persons.
SEP, somatosensory evoked potentials.

Kinesitherapy, psychotherapy and Kabat-Zinn techniques were applied during the period of in-patient treatment.¹⁰ A further programme of home rehabilitation is under way.

OUTCOME AND FOLLOW-UP

On discharge there was an obvious improvement of movements of the lower limbs. However, proprioceptive sensibility deficit persisted. The patient was able to gain the erect position independently, but only with the help of orthopaedic walking sticks. Postural activities were safeguarded and the patient was able to climb the stairs when helped. A secondary depressive syndrome related to the general condition was diagnosed during the first month of the rehabilitation procedures. After psychiatric consultation the patient received supportive psychotherapy in several sessions and no specific antidepressant therapy was deemed necessary.

DISCUSSION

The issue related to the probability of postlumbar puncture paraplegia or paraparesis is a complicated one, not only due to the devastating character of the neurological disability, but also under the medicolegal point of view, due to the litigations that might follow and with the defining of professional responsibilities.

Three main points need to be emphasised:

- ▶ The first issue is related to the traumatic character of the procedure, warranting a cautious position from the treating physician. An informed consent is an indispensable condition prior to performing the lumbar puncture for whatever diagnostic or therapeutic reason. In our case, however, the traumatic hypothesis seems rather impossible, because there were no repeated punctures, and MRI excluded any epidural haematoma following the event.
- ▶ The second issue is related with accidental intrathecal injections of a neurotoxic agent will be suspected, when no other explanation seems rational. There have been reported cases of inadvertent injection of Vinca alkaloids intrathecally; with severe and even fatal consequences.¹¹ Other factors and agents might be suspected, as in the remote claim of Woolley and Roe, when phenol, mineral acid or contaminants of whatever origin might have played an aetiological role.
- ▶ The third possibility, and the most plausible in our case, is the neurotoxicity of drugs administered for the anaesthesia itself. Ever since Bier used cocaine in his first spinal anaesthesia procedure at the end of the 19th century, he clearly noted its toxicity.¹² From that date on, there is a continuous and unrelenting pharmacological research aiming at the synthesis of an effective anaesthetic drug, to be injected intrathecally and free from neurotoxic effect.

Other elements however need to be further scrutinised in all cases, but the situation might still remain a diagnostic conundrum. Hypotension of course will be of diagnostic importance.¹³ This might be true even if hypotensive values are transitorily registered, such as in the case we described above. In our case, a dose of bupivacaine was injected intrathecally. This drug has been imputed to cause neurological deficits when administered for spinal anaesthesia, however practically no drug of the same family makes exception, albeit ropivacaine might be less neurotoxic, when compared with procaine, bupivacaine or levobupivacaine.¹⁴ Following these complications, recent research is focused in molecular mechanisms that might explain the neurotoxicity of this group of anaesthetic drugs.¹⁵ However, worth mentioning is that paraplegia, or permanent lower

extremity weakness following spinal and epidural anaesthesia is an extreme rarity, with sources referring only three cases from a total of 50 000 procedures.¹⁶ In fact, until now casuistics is made from isolated case reports, with unanswered questions in all of these reports.

Learning points

- ▶ Neurological deficit following spinal anaesthesia might have different clinical pictures, from sensory disturbances, to focal myelomalacia, syrinx formation and even paraplegia, the most devastating complication of the procedure.^{17 18}
- ▶ Bupivacaine or other anaesthetic drugs have been imputed as probable aetiological factors causing neurotoxicity when administered for spinal anaesthesia; however, this procedure is considered very safe if performed by skilled specialists.
- ▶ A detailed informed consent is always necessary, with explanation to the patient of all possible complications but also of the advantages of spinal anaesthesia, especially compared to the general anaesthesia, which represents a much more invasive procedure.
- ▶ Pharmacological advances in the field will allow in the next future the synthesis and availability of an anaesthetic free from neurotoxic effects; even actually some drugs of the same family are considered obsolete and replaced by newer generation drugs.

Contributors G.V. wrote the manuscript, reviewed the literature, and raised the theoretical hypothesis related to the case. G.V. followed up with the patient, mentored the writing of the manuscript. Both authors read and approved the final manuscript submitted.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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