

Innovative Insights in Case Reports and Reviews

A Case Report of a Unilateral Sub-Acute Extradural Haematoma Complicating Spinal Anaesthesia: The Value of Cranial Imaging in Persistent Neurological Deficits

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ABSTRACT

Background: Intracranial bleeding, following spinal or neuraxial anaesthesia does not occur very commonly. Rapid intracranial decompression with a gravitational slump of the brain in the intracranial cavity, leads to the snapping of bridging and emissary vessels. This could result in bleeding within the intracranial cavity, especially in the paradural spaces, which recent reports are increasingly recognising.

Patient and Methods: We report the case of a 30-year-old lady, who underwent spinal anaesthesia for an apparently uneventful transabdominal gynaecological procedure, but developed severe progressive generalised headaches 12 days later, which subsequently, became associated with focal neurological deficits, causing a considerable diagnostic dilemma.

Results: Cranial computed tomography (CT) scan revealed a left parieto-temporal subacute extradural haematoma, which was surgically evacuated, resulting in a full neurological recovery.

Conclusion: Patients who undergo spinal anaesthesia for gynaecological and surgical procedures could come down with

post-anaesthetic headaches. Recent reports are identifying complicating intracranial haematomata, in some of these cases of persistent headaches. The reports make a strong case for cranial scans in cases of persistent post-spinal puncture headaches, for definitive diagnoses and treatments.

Keywords: Burr holes, focal deficits, intracranial bleeding, persistent headaches

Introduction

Intracranial bleeding, following spinal or neuraxial anaesthesia does not occur very commonly, but, has been documented in literature as a serious complication of this procedure [1-5]. Martins, et al, reported an intracranial re-bleed, post-spinal anaesthesia, in a pregnant patient, with a previously undiagnosed chronic subdural haematoma [1]. Also, Park, et al, reported a case of acute-on-chronic subdural haematoma resulting from spinal anaesthesia in a patient with undiagnosed chronic subdural hematoma [2].

Iwase, et al, published a case of chronic subdural haematoma (CSDH), presenting after post-dural puncture headaches, complicating a combined spinal and epidural anaesthesia [3]. Remarkably, Bos, et al, had noted that though it is rare, intracranial haematoma collection is a serious complication after neuraxial anaesthesia for obstetric indications, in relatively younger women [4]. Li, et al had, actually, corroborated previous studies, that headaches remain the most common presenting symptoms [5].

In the study by Bisinotto, et al, the reported incidence of intracranial subdural haematoma, following lumbar puncture, is estimated in the range of 1:500,000 to 1:1,000,000 [6]. Some authors, however, insist that the actual incidence is unknown, and may be higher than has been reported in cited publications [2].

In 2010, Amorim et al reported two cases of intracranial haematomata after spinal anaesthesia, and then, analysed them with another 33 cases previously reported in literature [7]. They found that the interval between spinal anaesthesia and onset of symptoms of intracranial haemorrhage ranged from 4 hours to 29 weeks [7]. Headache was present in 73.4% cases, and was, indeed, the main symptom, just as Li, et al, had reported [5,7]. Other features, reported, included altered state of consciousness, vomiting, hemiplegia or hemiparesis, diplopia or paresis of VIth cranial nerve, and speech disorder [7].

Epidural haematoma has been less frequently reported than subdural haematoma, after neuraxial puncture for different indications [8,9]. However, Nagaska, et al, have reported the occurrence of epidural haematoma following spinal anaesthesia, for a caesarian section in a young woman [8]. But, much rarer than the unilateral intracranial haematomata are the bilateral epidural haematomata, which was reported in an adolescent spine surgery, by Li, et al [5].

Obstetric patients often complain of post-partum headaches, most of which are treated without investigation, making aetiological identification rather challenging [10]. But, the works of Li, et al and Amorim, et al, have shown that headaches, remain the first

and most frequent symptom, of intracranial haemorrhage, following spinal anaesthesia, a finding which makes a case for cranial imaging, in moderate to severe, and persisting post-spinal puncture headaches [5,7].

In their work in 2017, Yang and Huang, identified 3 stages in the development of a chronic subdural haematoma. The 1st stage corresponds to the traumatic event, which can be single or multiple, with or without clinical symptoms, leading to haematoma formation, and is the starting point for chronic haematoma development. The 2nd stage is a latency phase when the haematoma develops and slowly grows in volume. The blood clot becomes liquefied by fibrinolytic activity, and a membrane develops on both the dural and arachnoid surfaces, facilitating haematoma encapsulation and the occurrence of micro-haemorrhages due to fragility of newly formed vessels. During this period, patients may remain asymptomatic for weeks to years. However, clinical manifestations occur from the 3rd stage, when there is progressive decompensation of the intracranial capacity, due to the continuous growth of the haematoma capsule [11].

We report the case of a 30-year lady, who underwent spinal anaesthesia for an, apparently, uneventful transabdominal gynaecological procedure, but developed severe generalised headaches, 12-days after, and progressively developed focal neurological deficits, causing a considerable diagnostic dilemma. Cranial computed tomographic (CT) scan revealed a left parieto-temporal subacute extradural haematoma, for which she underwent burr hole evacuation of the haematoma, with resolution of the symptoms. She has remained stable after a 12-month postoperative follow-up period.

Case Report

A 30-year, nulliparous female postgraduate, presented to our service on 27/09/2024 with a 2-day complaint of severe generalized headaches, subtle slurring of speech and weakness of the right half of the body. She had just been discharged from a hospital, 12 days earlier, after undergoing an open transabdominal myomectomy, under spinal anaesthesia, for uterine fibroids, diagnosed during the work-up for primary infertility. She was transfused with 3 units of whole blood during the myomectomy, with no adverse reactions, and had been recuperating, progressively and uneventfully, following release from hospital, until the 12th postoperative day. There were no premorbid diseases outside the indication for surgery.

Clinical examination

Clinical examination revealed mild pallor and low-grade pyrexia (37.6°Celsius). The respiratory rate was 24/minute; pulse 105/

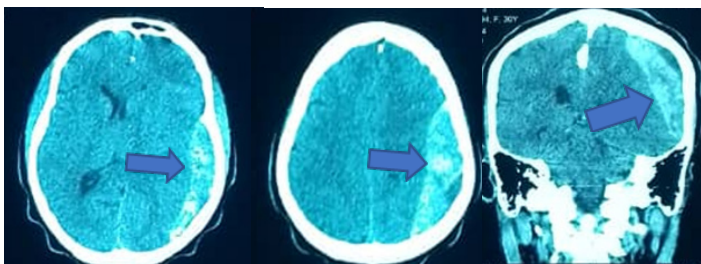
minute; blood pressure 131/96mmHg, SPO₂ 99%. Her Glasgow Coma Score was 14/15, she was confused; pupils were 4mm bilaterally, briskly reactive to direct and consensual light; there was a subtle left VIIth cranial nerve paresis - supranuclear type, expressive aphasia and amnesia for recent (medium term) events. She had normal muscle bulk globally, right hemi-hypotonia, power 2/5 right upper extremity and 4/5 in the right lower extremity, with extensor plantar reflex on the right and areflexia on the left. Tendon stretch reflexes were normal on the left but hyporeflexic on the right hemi-body. There was no meningism.

Laboratory work-up

Haemoglobin 7.7 (normal 12-15.5) g%,
White blood cells 15.5 (4-11) X10⁹/L and
Thrombocytosis 671 (150-400) x10⁹,
Prothrombin time (PT) - 13 (10-15),
International normalized ratio (INR) 0.9 (0.8-1.24), and
Activated partial thromboplastin time (APPT) 28 (23-45) seconds.

Imaging Findings

Cranial computed tomography [CT] scan revealed a large, mixed density, but predominantly hyperdense, left parieto-temporal, intracranial, extra-axial, concavo-convex, space-occupying lesion, adjacent to the endocranium, which was consistent with a possible subacute subdural haematoma. There was midline shift with pressure effect.



Unenhanced cranial CT scan of the patient showing the left parieto-temporal mixed density subacute subdural haematoma in axial and coronal views at different levels

Diagnosis

Subacute subdural haematoma 2^o to spinal anaesthesia.

Treatment

She had left temporal and parietal burr hole evacuation of the haematoma within 48 hours, under local infiltration with 1% lignocaine and 1:100,000 dilution of adrenaline, and intravenous ketamine anaesthesia.

Intraoperative Findings

Intact scalp and calvarium, mixed consistency altered liquid blood mixed with solid clots in the extradural space (not subdural space as was preoperatively diagnosed), 150ml total volume, depressing the underlying brain by 3cm. The brain became spontaneously pulsatile in the immediate post-evacuation period. Blood loss was 80ml, and she was transfused with one unit of fresh whole blood,

intraoperatively. The immediate postoperative condition was stable and satisfactory.

She, progressively, regained full neurological functions and was discharged home on the 10th postoperative day, and has remained symptom-free after 12 months follow-up.

Discussion

Rapid intracranial decompression, following lumbar puncture with cerebrospinal fluid efflux, leads to the snapping of bridging and emissary vessels, from gravity, resulting in bleeding within the intracranial cavity, especially in the paradural spaces. Depending on the intensity of the haemorrhage, manifestation of symptoms may occur within a few hours, or be delayed for several weeks, as reported by Amorim et al, and Yang and Huang. Amorim et al, reported a wide range of 4 hours to 29 weeks latency period, for the manifestation of symptoms. The neurological deficits in our case manifested in less than 2 weeks, which is an early manifestation.

During the development of a chronic subdural haematoma, Yang and Huang, had identified three distinguishable periods - the 1st period corresponds to the trauma, leading to haematoma formation and is the initiating stage for chronic haematoma. The 2nd is a latency phase when the haematoma develops and slowly enlarges in volume, the clot becomes liquefied by fibrinolytic activity and a membrane develops, facilitating haematoma encapsulation. During this period, patients may remain asymptomatic for years. Clinical manifestations occur during the 3rd and final period, when there is progressive decompensation of the intracranial capacity due to the continuous growth of the haematoma capsule.

The manifestation of severe headaches, as the first major symptom in our case, corroborated the findings of other workers, including Li, et al and Amorim, et al, who reported 73.4% incidence of headaches.⁷ The other features in our case were altered levels of consciousness (GCS] 14/15), confusion, left VIIth nerve paresis, expressive aphasia, medium-term events amnesia, and right hemiparesis - in keeping with those by Amorim, et al.

True to the usual rapid resolution of deficits with cases of epidural haematoma, our patient's neurological deficits progressively resolved within 1 week.

We have reported this case to add to the available body of knowledge on the intracranial complications of lumbar puncture for spinal anaesthesia, which do not happen frequently. This rare complication of epidural haematoma, should, also, be borne in mind by clinicians, whenever there are residual, persistent or progressive neurological deficits, following lumbar puncture for regional anaesthesia. Some of the post-spinal puncture headaches which are not usually investigated, could be the result of haematoma collection within the intracranial cavity. This implies that cranial imaging should be part of the routine work-up, once these headaches linger beyond a few days, or progressively worsen in intensity, or become associated with focal neurological deficits.

Conclusion

Patients who undergo spinal anaesthesia for gynaecological and surgical procedures could come down with post-spinal puncture headaches, which should not be ignored. Recent reports are identifying complicating intracranial haematoma, as the cause of some of these persistent headaches. Our report corroborated other published studies and, along with others, make a strong case for cranial imaging in cases of persistent post-spinal puncture headaches, for definitive diagnoses and treatments.

Conflict of Interest and Funding

The author declares no conflict of interest and received no specific funding for this work.

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