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Rebound intracranial hypertension after noninvasive treatment of intracranial hypotension: Case report and literature review

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Abstract

Intracranial hypotension is a clinical syndrome characterized by orthostatic headache and low cerebrospinal fluid pressure. Noninvasive management is the usual first line treatment. Epidural blood patch is the treatment of choice if noninvasive treatments are ineffective. Cases with rebound intracranial hypertension after epidural blood patch treatment have been reported in the medical literature previously. We report here three patients with rebound intracranial hypertension who were treated noninvasively for intracranial hypotension. This phenomenon has not been reported previously. The underlying cause of intracranial hypotension was epidural anesthesia in the first, lumbar disc surgery in the second patient, and idiopathic in the third patient. They had been treated either with bed rest or with medical treatment not requiring epidural blood patch. After a short remission the patients were seen with a different headache pattern. They all had papilledema on examination. Automated perimetry revealed bilateral blind spot enlargement in Patient 1 and peripheral constriction in Patient 2. Cranial MRI and MRV in all three patients were normal. All the patients recovered very quickly with acetazolamide 1.5 or 2gm/day.

In conclusion, rebound intracranial hypertension should be kept in mind in patients with intracranial hypotension who developed changes in the headache pattern, had new symptoms of nausea, vomiting, blurred or double vision during follow-up. Rebound intracranial hypertension can develop after conservative treatment of intracranial hypotension.

Keywords: Intracranial hypotension; orthostatic headache; rebound intracranial hypertension

INTRODUCTION

Intracranial hypotension is a clinical syndrome characterized by orthostatic (postural) headache, low cerebrospinal fluid (CSF) pressure and abnormal cranial magnetic resonance imaging (MRI). It may occur spontaneously (primary) or due to lumbar puncture, myelography, severe trauma or postoperative dural tear (secondary).[1,2] Noninvasive medical management including bed rest, oral hydration, caffeine or theophylline intake is the usual first line treatment. Epidural blood patch is the treatment of choice if noninvasive treatments are ineffective.[3,4] In the literature, there are cases of rebound intracranial hypertension (RIH) after epidural blood patch treatment.[6-11] However, no cases of RIH in intracranial hypotension that improved spontaneously or by medical treatment have been previously reported. In this case report, three cases of RIH following intracranial hypotension, treated conservatively are presented.

CASE REPORTS

Patient 1

A 50-year-old man was admitted to our clinic because of an increase in headache severity and change in its quality which became worse in the morning, associated with pulsatile tinnitus and transient visual obscurations for the last two months.

He had a history of hip surgery with epidural anesthesia performed six months ago. This was followed by three months of postural headache for which he was seen by neurologists and treated conservatively by bed rest, analgesics and caffeine as intracranial hypotension. There was a short period of near complete relief followed by the

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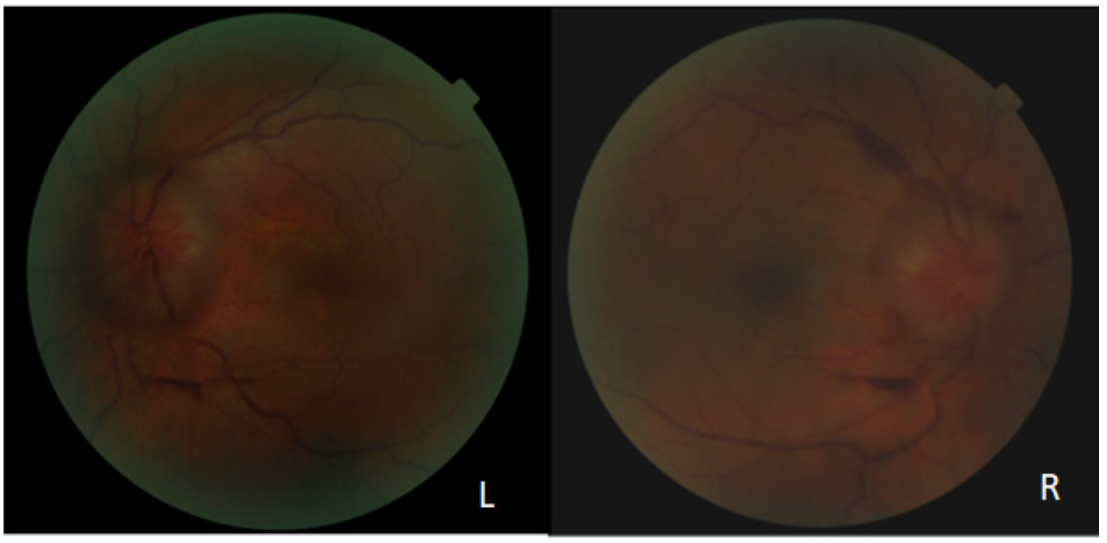

Figure 1. Fundus photographs patient 1 showing bilateral papilledema with hemorrhages.

new headache for which he was admitted.

Neurological examination showed normal visual acuity both sides. Bilateral papilledema was seen on fundoscopy (Figure 1). Blind spot enlargement was present on automated perimetry (Figure 2). Routine blood tests and blood biochemistry was normal. Other than enlargement of the optic nerve sheaths and posterior scleral flattening (Figure 3), cranial MRI was normal. MR venography was also normal. RIH was diagnosed complicating intracranial hypotension. Acetazolamide 1.5gm/d was initiated. The symptoms and signs resolved in three months.

Patient 2

A 49-year-old woman was admitted to our clinic with severe headache more prominent on awakening, progressive visual blurring and diplopia that developed over the last month.

She had history of lumbar disc surgery over four months ago. At the first day after the operation she began to complain of severe headache that was aggravated by standing. CSF leak was detected and she was treated with bed rest, theophylline and analgesics which resulted in symptom resolution for about three months. This was followed by the

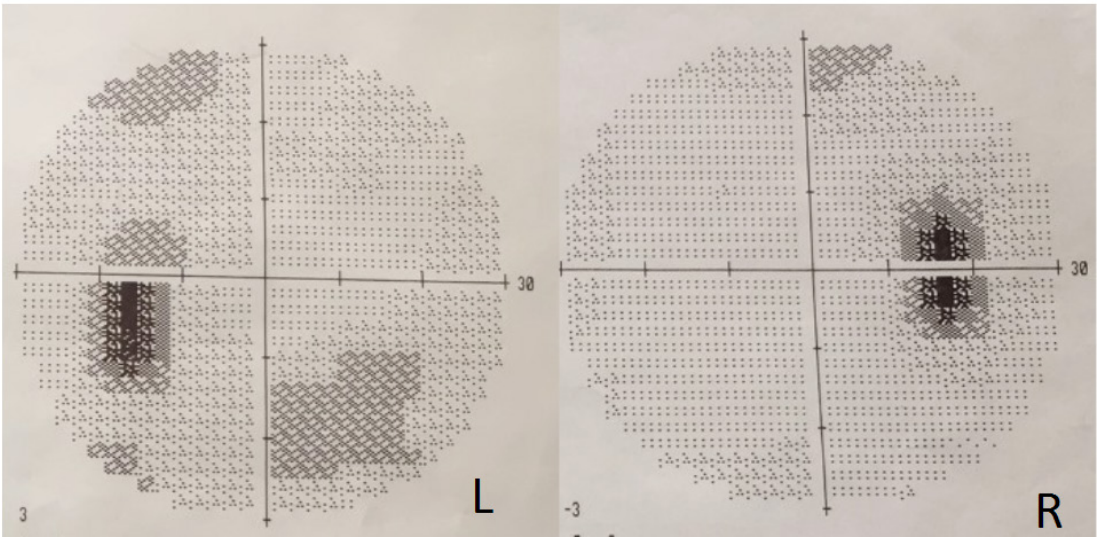

Figure 2. Visual field testing of patient 1 showing bilateral enlarged blind spots (Humphrey automated perimetry, central 30-2 threshold SITA Fast program)

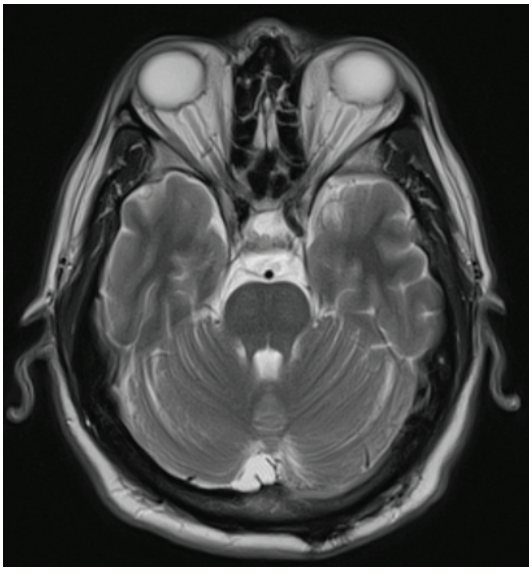

Figure 3. T-2 weighted axial MR image of patient 1 showing enlargement of the optic nerve sheaths and posterior scleral flattening on behalf of intracranial hypertension.

new headache that triggered the recent admission.

On examination the visual acuity was 8/10 on the right and 9/10 on the left eye. Papilledema was noted bilaterally on fundoscopy. There was esodeviation of both eyes with bilateral abduction paresis. Automated perimetry revealed peripheral constriction of the visual fields on both sides. Routine blood tests and blood biochemistry was normal. Cranial MRI and MR venography were also normal. RIH complicating intracranial hypotension was diagnosed, and acetazolamide 2gm/d was initiated. Her symptoms and signs resolved completely after two months.

Patient 3

This 18-year-old woman was admitted to our clinic because of headache aggravated by lying down with tinnitus and transient visual obscurations for the last two weeks.

She had headache four months ago that was aggravated by getting up. The cranial MRI performed at that time showed diffuse pachymeningeal enhancement and the lumbar puncture showed 60 mmH20 of CSF pressure (Figure 4). As an underlying cause was not present, she was diagnosed as idiopathic intracranial hypotension and was treated conservatively with bed rest. The symptoms resolved in approximately a month, but reappeared a few weeks later with a different quality that triggered the current admission.

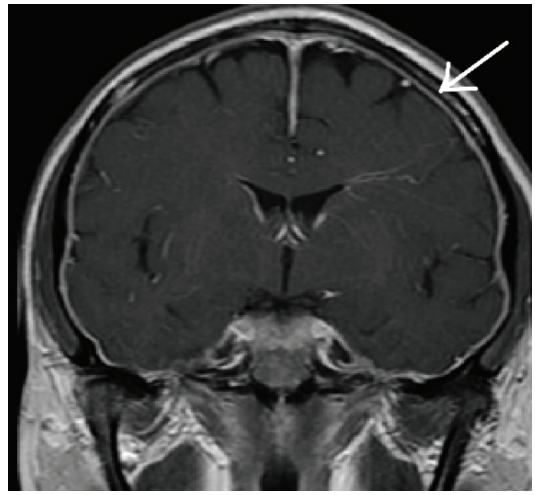

Figure 4. T-1 weighted coronal MR image with gadolinium of patient 3 at the initial headache period showing diffuse pachymeningeal enhancement on behalf of intracranial hypotension

On neurological examination, she had normal visual acuity on both eyes with papilledema on fundoscopy. Automated perimetry revealed normal visual fields. Routine blood tests and blood biochemistry was normal. Cranial MRI and MRV were also normal. RIH complicating intracranial hypotension was diagnosed. She was started on 1.5gm/d acetazolamide and was symptom free after two months.

DISCUSSION

Intracranial hypotension may occur spontaneously, or may develop secondary to lumbar puncture, myelography, severe trauma or dural rupture after surgery. Headache is the most common symptom. It is typically postural, increases with standing and decreases with supine posture. Orthostatic headache is caused by compensatory vasodilation of the dural sinuses and meningeal blood vessels and by traction or distortion of various anchoring pain-sensitive structures of the brain due to downward displacement of the brain from reduced hydrostatic force.[14,15] In addition to headache, nausea, vomiting, neck pain, vertigo, diplopia, hearing disorders, tinnitus can also occur.[12,13] Traction or compression of the cranial nerves is suspected to be the mechanism.

MRI findings include diffuse pachymeningeal enhancement, descent of the brain, subdural fluid collections, typically hygromas, infrequently hematomas are features supportive of the diagnosis.[3]

CSF opening pressure is typically low. Protein concentration may be normal or high with values up to 100 mg/dL, and a lymphocytic pleocytosis of up to 40 cells/mm^2. CT myelography is thought to be the most useful investigation to localize the site of the CSF leak.[3]

Noninvasive treatment consisting of bed rest, hydration, caffeine or theophylline is the first line therapy. Intravenous or oral caffeine and theophylline cause arterial contraction by blocking adenosine receptors. It result in clinical improvement by reducing intracranial blood volume and venous fullness.[16,17] Epidural blood patch is the treatment of choice if noninvasive treatments fail. Epidural blood patch is an effective treatment and is by injecting autologous blood into the epidural space.[18]

Cases of RIH after epidural blood patch treatment for intracranial hypotension has been previously been reported.[6-11] Change in the location and pattern of headache are symptoms suggestive of the diagnosis. The headache that become worse with recumbent position, that localized in the periorbital area and early morning headache are characteristic features of intracranial hypertension.[11] Nausea, vomiting, blurred vision and diplopia are other features that suggest the development of RIH.

The mechanism underlying RIH development is unknown. In patients treated with epidural patch, the patching material was proposed to increase intracranial pressure though the increase was not proportional with the volume of the material used.[9]

One potential explanation could be the distention of the intracranial venous structures to compensate for decreased CSF volume which failed to reverse immediately after repair of the CSF leak. However, this explanation can only help to understand the early RIH.

Upregulation of CSF production or disrupted CSF reabsorption during the CSF leak for compensation can be the mechanism of delayed RIH. Subsequent cessation of the leak may cause increased intracranial pressure for variable periods until a normal balance between production and resorption of CSF is established.

Another possible mechanism is that long-term CSF leakage causes degeneration in the CSF resorption mechanism. The balance between CSF production and resorption may subsequently be impaired and RIH may occur until sufficient function is achieved in the resorption mechanisms.[9]

Acetolazomide, a carbonic anhydrase enzyme inhibitor, was used for the treatment of RIH in the cases described in the literature. Acetazolamide disrupts sodium-hydrogen ion exchange in choroid plexuses and decreases CSF production and intracranial pressure.[19]

Our three patients represent RIH developing after resolution of intracranial hypotension either spontaneously or by noninvasive treatment. This has not been reported previously. Change in the headache pattern contributed to the diagnosis. Associated papilledema was the hallmark of intracranial hypertension which was florid in Patient 1. In addition, bilateral esodeviation of the eyes with abduction paresis was present in Patient 2. Clinical features of intracranial hypertension developing after weeks in these patients may be due to upregulation of production and failure of resorption of CSF during the period of leak which stabilized within a short time by using acetazolamide that inhibits CSF production.

In conclusion, RIH can develop as a complication after intracranial hypotension either treated conservatively or with epidural blood patch. Changes in the headache pattern, development of new nausea, vomiting, blurred or double visions are the key features of the intracranial hypertension.

DISCLOSURE

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Conflicts of interest: None

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