Benign intracranial hypertension with spinal and radicular pain

Case report

MARCO BORTOLUZZI, M.D., LEONARDO DI LAURO, M.D., AND GIOVANNI MARINI, M.D.
Neurosurgical Department, Regional General Hospital, Brescia, Italy

A case of benign intracranial hypertension with polyradiculopathy and spinal pain is reported. Radioactive iodinated serum albumin (RISA) cisternography demonstrated the absence of cerebrospinal fluid (CSF) flow into the intracranial cisterns, and gave evidence of CSF absorption through the spinal arachnoid villi.

KEY WORDS - benign intracranial hypertension - cerebrospinal fluid dynamics - RISA cisternography - pseudotumor - pain

The syndrome of benign intracranial hypertension consists of increased intracranial pressure (ICP) with headache, papilledema, and visual disturbances, without impairment of consciousness or neurological localizing signs, except for an occasional sixth nerve palsy. The absence of mass lesions and a normal ventricular system are other peculiar features of this disorder.10,13,19

A case of benign intracranial hypertension, in which the characteristic neurological picture was associated with polyradiculopathy and spinal pain, is reported here. In this patient, abnormalities in the major cerebral venous drainage system and impairment of cerebrospinal fluid (CSF) flow and absorption were demonstrated. Absorption of CSF seemed to occur through the spinal arachnoid villi.

Case Report

This obese 34-year-old woman came to our attention because of uncontrollable back and radicular pain involving the lower limbs, thorax, and cervical area. The pain had persisted for 7 months prior to admission and was associated with a troublesome headache.

History. The clinical history had begun 3 years earlier with a painful “rigidity” of the spine, which progressed to a typical low-back pain. These disturbances were ascribed to a supposed L5-S1 lumbar disc herniation, and the patient was operated on. After disc removal, there was no improvement and the pain continued to worsen. Two years after this operation, the patient underwent another surgical exploration. Bilateral L4-5 laminectomy was performed in the hope of relieving the pain by removing a possible painful scar left by the first operation. At operation, no disc protrusion was seen; the exposed nerve roots as well as the lumbar dural sac were described as very enlarged. The operative scar was reported as normal, and no definite root compression was observed. The dura was not opened because preoperative oil myelography had excluded root cysts or other spinal pathological entities. There was no improvement of symptoms.

For the 5 months prior to admission to our department, the patient was bedridden and unable to walk because of back and leg pain which was intolerable in the standing position. The pain was described as continuous in the back, as though “the spine was being split,” with polyradicular extension to the legs, superficial abdominal wall, thorax, and cervicobrachial area. On the thorax, the pain was “as constrictive as a girdle.” Several episodes of deep abdominal pain without evidence of visceral illness were reported. In the upper cervical area, the pain was referred to as less severe, spreading from the region of the shoulders to the fingers.
Routine laboratory examinations, blood pressure, electrocardiogram, pertechnetate brain scan, and electroencephalogram were all normal. Computerized tomography (CT) of the brain showed no abnormalities, small ventricles, and no edema. No pathological entities were found on CT scanning of the entire cervical and lumbar spine. On air study, an empty sella was observed (Fig. 1), with some enlargement of the callosal and basal cisterns. The ventricles did not fill, and the air entered only partially into the CSF pathways of the brain convexity. No abnormalities of the cisterns in the posterior fossa were seen. Repeat lumbar puncture pressure measurements ranged between 350 and 450 mm Hg. The CSF protein levels were normal. Bilateral carotid angiography demonstrated hypoplasia of the left lateral sinus and a severe obstruction of the right lateral sinus, which seemed to afford the major functional drainage for the longitudinal sinus (Fig. 2). An unusually dilated cervical dural plexus and significant abnormal venous outflow channel were also observed.

Radioactive iodinated serum albumin (RISA) cisternography was performed: human serum albumin (2 mg) labeled with iodine-131 (150 μCi) was injected into the lumbar subarachnoid area. Cisternography demonstrated pathological persistence of the radioactivity over 24 hours throughout the spinal subarachnoid space. There was no flow into the cisterna magna or cerebral subarachnoid spaces. No significant radioactivity in the basal cisterns, brain convexity, or ventricles was detected in the serial 48-hour scans. After

Examination. On admission, slight lower limb weakness with moderate bilateral reduction of deep tendon reflexes was found. There was no Babinski sign, and normal bowel and bladder function was observed. She had impairment of superficial pain, temperature, and tactile sensations bilaterally from T-6 to T-12. The abdominal reflexes were greatly reduced. Jugular compression exacerbated the back and radicular pain. Bilateral papilledema, with visual acuity of 8/10 on the right and 6/10 on the left, and an enlarged blind spot with binasal hemianopsia were found.

**Fig. 1.** Air study showing an empty sella (arrow). There was no ventricular filling.

**Fig. 2.** Carotid angiograms. *Left:* Anteroposterior view showing hypoplasia of the left lateral sinus (arrowheads) and a severe obstruction of the contralateral sinus (arrows). *Right:* Lateral view showing the severe obstruction of the right sinus (arrowheads).
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2 hours, abnormal persistence of radioactivity in the spinal area was observed (Fig. 3). There was evidence of an unusual enlargement of the subarachnoid space below L-2; in the thoracic area, the radionuclide flowed bilaterally for about 2 cm along the spinal roots. This finding suggested delayed clearance occurring only through the root pouches, which were enlarged in the dorsal segment of the spinal cord. A clinical trial for 20 days with dexamethasone (24 mg daily, intramuscularly), 20% mannitol (350 ml daily), furosemide (25 mg orally), and acetazolamide (250 mg every 8 hours) improved the papilledema, but had no effect on the pain.

Operation and Postoperative Course. A lumbar peritoneal shunt with a medium-pressure (5 to 9 mm Hg closing pressure) valve was inserted, resulting in immediate improvement in the spinal pain and headache. On discharge 25 days after surgery, the patient was able to stand and walk. Seven months later, no visual impairment and an almost total resolution of the pain were reported. The patient was able to attend to her housework. Cisternography with 2 mg of human serum albumin labeled with technetium-99m (2 mCi), performed by the lumbar route, demonstrated no abnormal persistence of radioactivity in the spinal canal. No flow toward the cerebral cisterns was detected; the radioactive media followed the channel of the shunt and collected in the peritoneal cavity. This finding proved that the device was functioning.

Discussion

Numerous reports on benign intracranial hypertension have suggested a large variety of factors that may play a role in the etiology of this syndrome; however, the basic nature of this entity is still debated. This syndrome has been ascribed to cerebral edema, increased cerebral blood volume (CBV), and CSF absorption defects. The former hypothesis, proposed by Sahs and Joynt, has not been confirmed by others. The latter two theories have recently received support. Following the idea of Dandy, increased CBV has been demonstrated, suggesting possible venous engorgement. Impairment of the venous drainage system in benign intracranial hypertension has also been reported. The important role of reduced CSF absorption has been shown by Johnston and Patterson. Investigations with a radioactive medium or with lumbar infusion techniques have demonstrated a delayed CSF flow and an impaired CSF absorption across the cerebral arachnoid villi in many cases. Other data in the literature give additional evidence of CSF volume increase. The major objection to the theory of a defect in CSF absorption is the lack of ventricular enlargement.

It seems reasonable to postulate that two different pathogenetic events coexist and may increase both the CSF and brain volume. Normal or even reduced ventricular size may be explained in this way, despite the high ICP leading to an empty sella or bone erosion. In our case, angiographic and cisternographic data suggest that the causative concurrent mechanisms were an increase of CBV due to the venous engorgement and impaired CSF absorption. The RISA cisternogram with showed the radioactive medium only in the spinal subarachnoid spaces, where it persisted for 24 hours, without evidence of significant radioactivity in the brain.

These findings are consistent with the absence of CSF flow toward the cerebral subarachnoid spaces and with altered CSF absorption rates occurring only across the spinal subarachnoid villi. We believe this is the first case in which evidence is given of the primary role that this auxiliary route may assume in CSF drainage when the normal cerebral resorptive routes are obstructed. These data substantiate experimental investigations on the spinal villi. From a clinical standpoint, isotope cisternography may provide an explanation for the lumbar and radicular disturbance.

Lack of CSF flow toward the cerebral cisterns and reduced CSF absorptive capacity lead to a large volume of CSF being trapped in the spinal subarachnoid spaces, causing the dural sac and root pouches to

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stretch and appear very enlarged on RISA cisternography. This phenomenon may account for the polyradicular and back pain, which increased mainly in the standing position, when a further increase in CSF pressure developed in the spinal subarachnoid area.

Another possible associated mechanism may be suggested. Compensatory overfunction could induce hypertrophy of the spinal subarachnoid villi and of the surrounding draining lymphatic ducts and veins, which are all intimately located on the dorsal roots. This abnormal condition may cause root compression or interference with the venous drainage and result in sensory disturbances. This hypothesis may find support in the fact that, after CSF shunting in our patient, the spinal pain immediately disappeared, whereas hypesthesia and radicular pain lasted for months, especially in the thoracic segment where major enlargement of the root pouches suggested more effective CSF absorption.

References


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