



CASE REPORT

Idiopathic intracranial hypertension occurred after spinal surgery: report of two rare cases and systematic review of the literature

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Abstract

Background Idiopathic intracranial hypertension (IIH) is a relatively rare syndrome of increased intracranial pressure of unknown etiology. It is characterized by cerebrospinal fluid (CSF) opening pressure more than 250 mmH₂O, with normal cranial imaging and CSF content. IIH occurred after spinal surgery is extremely rare.

Methods We present two IIH cases occurred after spinal surgery and conduct a systematic review of articles reporting IIH occurred after spinal surgery.

Results The first patient underwent a posterior decompression and fixation for cervical fractures. IIH symptoms appeared 3 days postoperatively and gradually resolved with appropriate medication. The second patient underwent posterior spinal fusion with segmental instrumentation for congenital scoliosis. IIH symptoms appeared 5 days postoperatively and the patient died due to the irreversible intracranial hypertension although underwent intensive care and treatment. The literature review revealed that there were only five cases of IIH occurred after spinal surgery reported till date.

Conclusions IIH occurred after spinal surgery is relatively rare; the diagnosis is based upon exclusion of other diseases. IIH should be kept in mind in patients underwent spinal surgery as it could develop into irreversible intracranial hypertension.

Keywords Idiopathic intracranial hypertension · Spine · Surgery

Introduction

Idiopathic intracranial hypertension (IIH), also known as primary pseudotumor cerebri, is a syndrome of unknown etiology that causes increased intracranial pressure [1–3]. It is a condition of increased intracranial pressure without a space-occupying lesion, hydrocephalus and intracranial hemorrhage [4, 5]. The classic symptoms and signs of IIH are headache, pulsatile tinnitus, transient visual obscurations, blurred vision, diplopia and papilloedema. And the result of test is characterized by normal neuroimaging and cerebral spinal fluid (CSF) content [1]. In 1893, IIH was firstly described by Quincke as “serous meningitis” in patients who had increased intracranial pressure without a brain tumor [6]. Over the last 120 years, this relatively rare disease has acquired many different names, such as serous meningitis [6], pseudotumor cerebri [7], benign intracranial hypertension, primary intracranial hypertension and IIH [1, 8, 9]. The term “idiopathic intracranial hypertension” was firstly proposed by Corbett et al. [9], although the exact etiology and pathophysiology currently remains unclear. However, a large number of risk factors have been identified to be associated with IIH, such as obesity, recent weight gain, polycystic ovarian syndrome, and thin children [1, 10].

The diagnostic characteristics of IIH were initially enumerated by Dandy et al. [11] in 1937 and were later formulated into a set of diagnostic criteria by Smith [7]. Finally, Friedman et al. updated the criteria in 2002 [8] and 2013 [1]. According to Friedman et al., the criteria for diagnosing IIH should include (1) papilledema; (2) normal

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neurologic examination except for cranial nerve abnormalities; (3) neuroimaging: normal brain parenchyma without evidence of hydrocephalus, mass, or structural lesion and no abnormal meningeal enhancement on MRI, with and without gadolinium, for typical patients (female and obese), and magnetic resonance venography for others; if MRI is unavailable or contraindicated, contrast-enhanced CT may be used; (4) increased lumbar puncture opening pressure ($\geq 250 \text{ mmH}_2\text{O}$ CSF in adults and $\geq 280 \text{ mmH}_2\text{O}$ in children) in a properly performed lumbar puncture; (5) normal CSF composition [1].

It is reported that the incidence of IIH in developed countries is about 0.9/100,000 persons and 3.5/100,000 in 15- to 44-year-old females [12, 13]. And the incidence is increasing in parallel with the current worldwide epidemic of obesity [14, 15]. Although considerable literature has been published concerning IIH occurred during pregnancy [16, 17] and childhood [18, 19], there are few studies reported IIH occurred after spinal surgery.

In this study, we present two cases of IIH occurred after spinal surgery and conduct a systematic review to determine the overall incidence, diagnosis, management and the prognosis of IIH occurred after spinal surgery. The most important purpose of this report and systematic review is to raise the awareness that headache, pulsatile tinnitus, vomiting and visual impairment after spinal surgery can represent IIH, an extremely rare but potentially devastating condition.

Case report

Case 1: IIH after spinal trauma surgery

This 58-year-old female patient presented with progressively headaches, frequent vomiting, and blurring vision in both eyes on day 3 after a spinal trauma surgery. One week earlier, she developed C4 and C5 fractures with spinal cord injury (ASIA-C) after a 50 kg wood crushed her neck. The CT scan showed right inferior articular process fracture of C4 and the right pedicle and laminar fractures of C5 (Fig. 1a, b). The MRI showed spinal cord swelling and high signal intensity in T2-weighted imagine at C4–6 levels (Fig. 1c, d). She underwent a posterior decompressive bilateral laminectomy and instrumented arthrodesis from C4 to C6. The operation went smoothly and there was no intraoperative complication; however, IIH symptoms developed 3 days postoperatively. The medical history was uncomplicated with mild hypertension for 5 years.

On general physical examination, all vital signs were within normal limits except the systolic blood pressure was 147 mmHg and BMI was 31.22 kg/m^2 . Visual acuity and visual field were both normal and the second through

twelfth cranial nerves were all normal. On specialized examination, the patient had 2/5 strength in left biceps, wrist extensors, triceps, 1/5 strength in the finger flexors and left lower extremity muscles. Sensation was grossly intact to light touch throughout all extremities, and thermal and pinprick hypoesthesia was observed below C5. Rectal tone and contraction were normal, so this patient was classified as C5 ASIA Grade C tetraplegia. And ophthalmologic funduscopic examination conducted by a senior ophthalmologist disclosed bilateral papilledema.

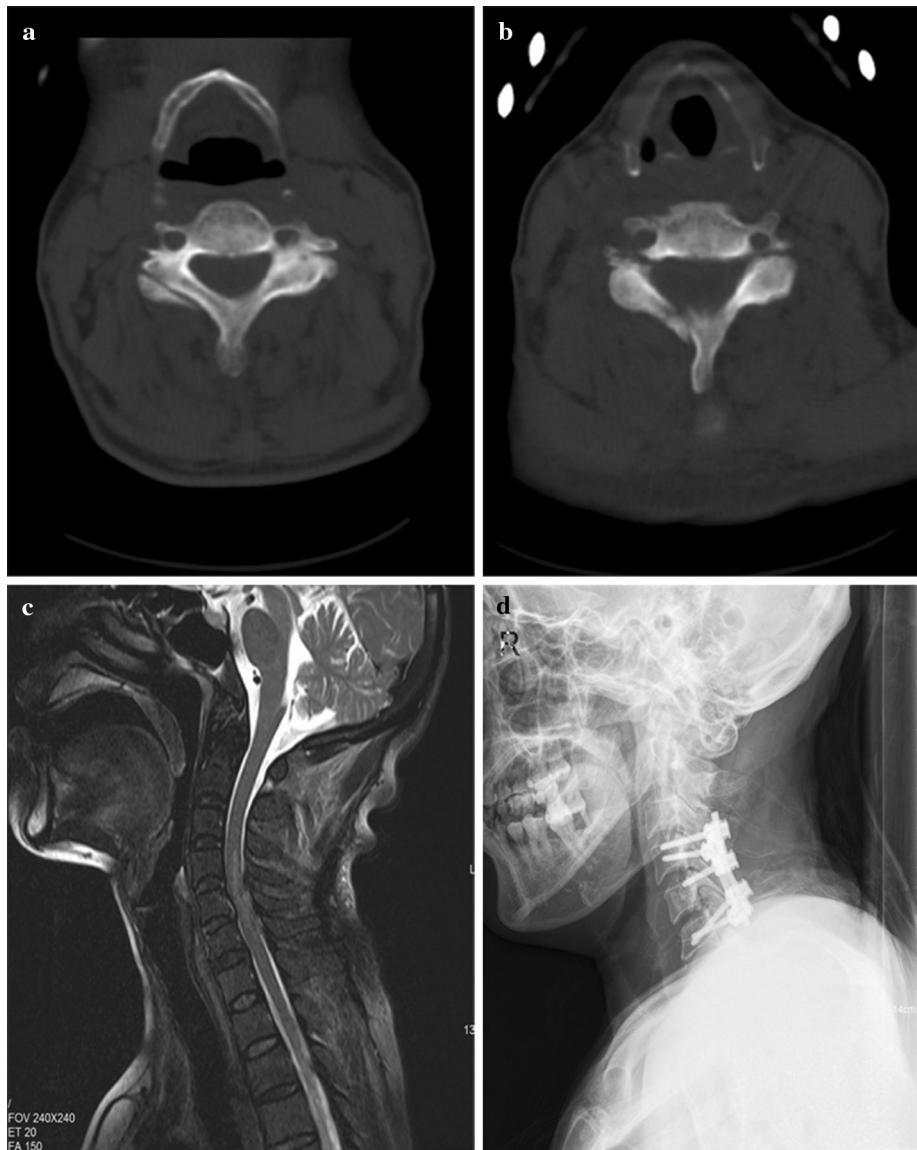
CT scan and MRI of the head were taken, but the result showed no space-occupying lesion, hydrocephalus and intracranial hemorrhage (Fig. 2a, b). Magnetic resonance venography was taken and no cerebral venous sinus thrombosis was found (Fig. 2c, d). A lumbar puncture was finally completed, and the opening pressure was found to be $290 \text{ mmH}_2\text{O}$. After removing 20 ml of CSF, the patient's CSF opening pressure decreased to $210 \text{ mmH}_2\text{O}$, and her headache transient resolved. The CSF analysis showed the glucose was 39 mg/dl, protein was 36 mg/dl, and chloride was 122 mEq/l and no cell. Stains for fungi, acid-fast bacilli and spirochetes as well as culture for bacteria and fungi were all negative. A diagnosis of IIH was made and acetazolamide (500 mg twice daily) was prescribed.

Two weeks after the surgery, the patient's condition improved from ASIA Grade C to ASIA Grade D. However, symptomatic headache still persisted, another lumbar puncture was performed and CSF opening pressure was $260 \text{ mmH}_2\text{O}$. The treatment with acetazolamide was continued and weight loss was recommended. Three weeks after the surgery, she had just slight headache and acetazolamide was reduced to 250 mg twice daily. At 3 months postoperatively, the patient's BMI decreased to 29.32 kg/m^2 and the IIH symptoms completely resolved. The ophthalmologic funduscopic examination also showed no papilledema. So, acetazolamide was discontinued, and the patient remained free of IIH symptoms at the 6-month follow-up visit (Figs. 3, 4).

Case 2: IIH after spinal deformity surgery

This 20-year-old girl presented with severe headache, pulsatile tinnitus, nausea, projectile vomiting and blurred vision in both eyes on day 5 after a spinal deformity surgery (Fig. 3). She underwent posterior spinal fusion with segmental instrumentation from T2 to L3 for congenital scoliosis 5 days before IIH onset. The intraoperative blood loss was 2 l, and she received 3 U of autologous blood and 6 U allogeneic blood transfusion. Her medical history is significant for congenital scoliosis for 20 years, mild respiratory dysfunction for 6 years and interventional therapy for congenital patent ductus arteriosus at 1 year of age.

Fig. 1 Case 1. The initial CT scan showing the horizontal images of the C4 (a) and C5 (b) fractures. T2-weighted MRI of sagittal plane showing spinal cord swelling and high signal intensity at C4–6 levels (c). The sagittal X-radiography showing a posterior decompressive bilateral laminectomy and instrumented arthrodesis from C4 to C6 (d)



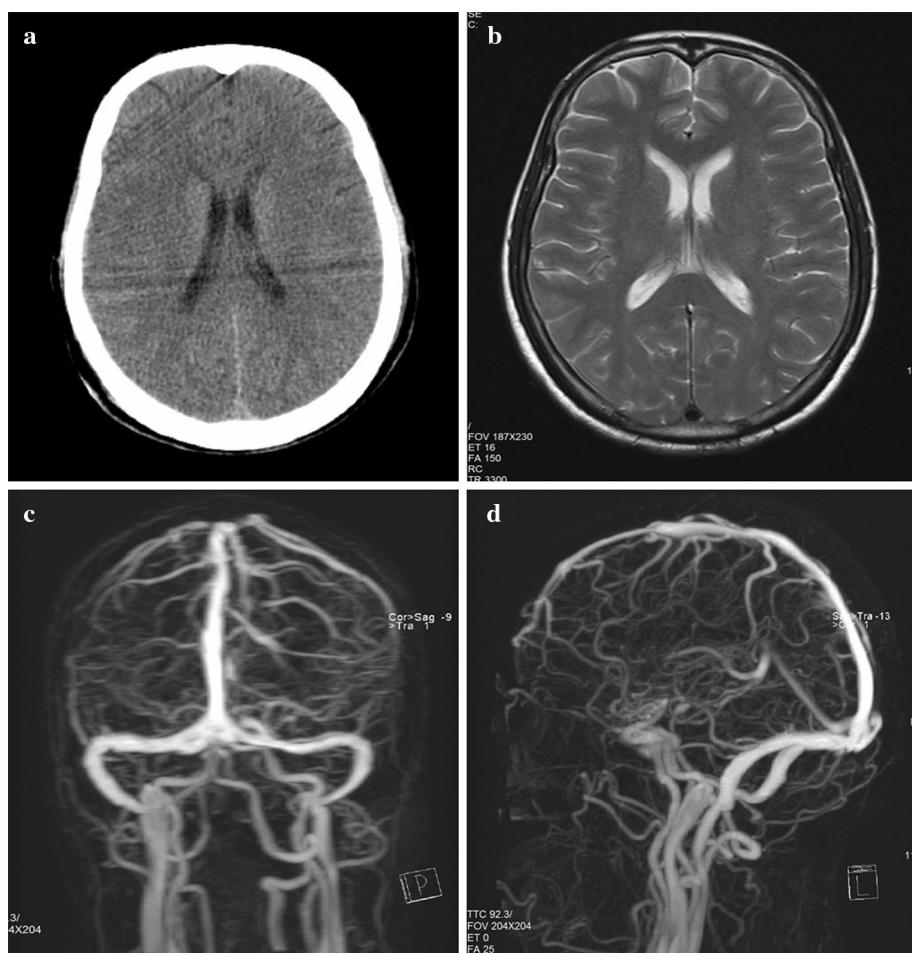
Postoperatively, she was started on methylprednisolone (40 mg twice daily) for 3 days as the strengths of bilateral quadriceps were grade 4 of 5. On general physical examination, she was afebrile and had no focal physical signs of infection and all vital signs were normal. On specialized examination, the second through the twelfth cranial nerves were normal and the strengths were graded 4/5 in bilateral quadriceps. The ophthalmologic examination by a senior ophthalmologist revealed bilateral severe papilledema with a 6/10 visual acuity loss in left eye and 8/10 in right eye.

CT scan and MRI of the head were unremarkable (Fig. 4a, b). Magnetic resonance venography demonstrated that the intracranial venous sinuses were also patent (Fig. 4c, d). Lumbar puncture showed the CSF opening pressure of 300 mmH₂O. After removing 15 ml of CSF, the intracranial pressure decreased to 235 mmH₂O and her

headache transient relieved after received 100 ml 20 % mannitol. The CSF analysis showed normal glucose and protein concentration, as well as cytology and cell counts. A diagnosis of IIH was established and the patient was given 500 mg acetazolamide and 100 ml 20 % mannitol twice daily.

One day later, another lumbar puncture showed the opening pressure was still 280 mmH₂O. So 40 mg furosemide twice daily combined with potassium was added to the medication regimen. Two day later, the patient reported that she had explosive and intense headache, and she had a dark screen over the visual field of the left eye. Another 125 ml 20 % mannitol was given; however, she began vomiting, seizing and lost her consciousness after half an hour. Tracheal intubation and mechanical ventilation was performed immediately. CT scan of the head still

Fig. 2 Case 1. Cranial CT scan (a), MRI (b) and magnetic resonance venography (c, d) images. The CT scan and MRI of sagittal plane showed no space-occupying lesion, hydrocephalus and intracranial hemorrhage. No cerebral venous sinus thrombosis was found on magnetic resonance venography image



showed no space-occupying lesion, hydrocephalus and intracranial hemorrhage. Unfortunately, MRI could not be done as the patient cannot wean from mechanical ventilation and contrast-enhanced CT venography failed because the contrast medium could not diffuse into the brain due to the extremely elevated intracranial hypertension. Four hours after the patient lost consciousness, her breath and heartbeat stopped due to the irreversible intracranial hypertension.

Systematic review of the literature

A complete PUBMED search for original articles reporting IIH occurred after spinal surgery was conducted. Inclusion criteria included (1) articles in English; (2) article that reported at least one case of IIH occurred after spinal surgery; (3) the article must have included demographic details, diagnostic method, management and follow-up. Exclusion criteria included (1) secondary intracranial hypertension; (2) comment article; (3) article not mentioned the data on demographic details, diagnostic method,

management and follow-up. Using a combination of the search terms “Idiopathic intracranial hypertension”, “IIH”, “primary pseudotumor cerebri”, “benign intracranial hypertension”, “pseudotumor cerebri”, “serous meningitis”, “spinal operation”, “spinal surgery”, and “spinal surgical procedure”, a total of 219 primary articles were identified as of September 1, 2015. Further relevant articles were identified by scanning their abstracts and titles. Two reviewers further independently examined the full texts and reached a consensus on those to be included. After excluding articles based on title and abstract, only three papers detailing five cases of IIH occurred after spinal surgery were included. And these five cases could be subdivided into two different categories: IIH occurred after spinal trauma surgery and IIH occurred after spinal deformity surgery.

In 1995, Daftari et al. presented an IIH case occurred after an occipitocervical arthrodesis with immobilization in a halo vest for an odontoid fracture [20]. This patient was a thin young woman and she developed IIH after three operations for the nonunion of odontoid. The patient was treated by 500 mg acetazolamide twice a day. Eight

Fig. 3 Case 2. Preoperative anteroposterior (left) and lateral (right) X-ray showing congenital scoliosis



months after the operation, her cervical radiograph showed the consolidation of the fracture and the headache and papilledema completely resolved. In 2004, Sussman presented a similar IIH case who had a suboptimal surgical stabilization for a traumatic C5–7 burst fractures with C5 spinal cord injury (ASIA-A) [21]. This 26-year-old male and obese ($\text{BMI } 32 \text{ kg/m}^2$) patient underwent an anterior cervical corpectomy (C5–7) and fusion and developed IIH 12 days postoperatively. IIH symptoms gradually resolved after acetazolamide (500 mg twice daily) and a posterior arthrodesis and fusion. On day 53 after the diagnosis of IIH, the patient's symptoms had resolved and an ophthalmologic examination showed no papilledema.

There are also previously reported IIH cases occurred after spinal deformity surgery. In 2011, Kunes et al. reported three adolescent patients who underwent uncomplicated segmental spinal instrumentation for pediatric spinal deformity correction and subsequently developed IIH [22]. This case series included two 13-year-old female patients of scoliosis and one 16-year-old male patient of Scheuermann kyphosis. These three patients were all full recovery after a regimen of acetazolamide for 1–2 months. However, papilledema persisted postoperatively in the patient with Scheuermann kyphosis. The authors identified the common variables in these three patients were adolescent age, spinal deformity, overweight, symptom onset

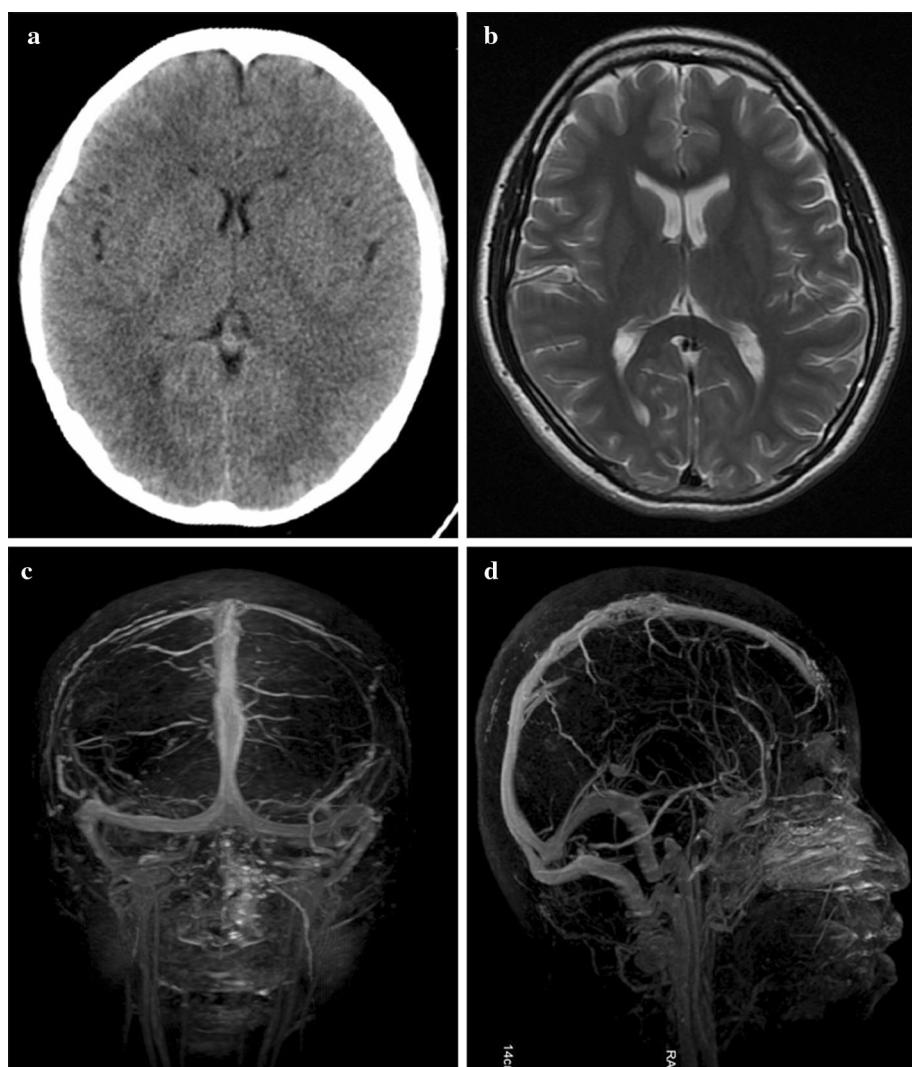
within 2 weeks postoperatively, significant intraoperative blood loss, and intraoperative use of antifibrinolytics.

Discussion

Over the last few years, many articles have been published on IIH, especially about the diagnosis and treatment of IIH. However, IIH occurred after spinal surgery is extremely rare. In this report, we present two IIH cases occurred after spinal surgery, including one case occurred after spinal trauma surgery and another case occurred after spinal deformity surgery. These two patients both presented with clearly definable IIH based on the criteria set forth by Friedman et al. [1]. Then we conducted a literature review to gain an overview of the incidence, diagnosis, management and the prognosis of IIH occurred after spinal surgery. The result showed that there are only five reported cases of IIH occurred after spinal surgery. These five cases could also be subdivided into: IIH occurred after spinal trauma surgery and IIH occurred after spinal deformity surgery. To the best of our knowledge, this is the first literature review focusing on IIH occurred after spinal surgery.

Although, IIH has been reported for more than 100 years, the pathogenesis of IIH is still not well

Fig. 4 Case 2. Cranial CT scan (a), MRI (b) and magnetic resonance venography (c, d) images. The CT scan and MRI of sagittal plane showed no space-occupying lesion, hydrocephalus and intracranial hemorrhage. The intracranial venous sinuses were patent on magnetic resonance venography image



understood. Over the years, there are several theories proposed to explain the pathogenesis of IIH, such as abnormal CSF homeostasis, excessive CSF production, reduced CSF absorption, increased cerebral blood flow, intracellular and extracellular edema and CSF outflow obstruction increasing the cerebral venous pressure [23–26]. Obesity, recent weight gain, female gender, polycystic ovarian syndrome, and thin children were reported to be the risk factors for IIH [1, 10, 27]. The first case in this report is obese and female, and the second case is female and had a recent weight gain in last 2 months, which may put them at increased risk for IIH. However, IIH could also occur in thin adult patient, such as the case in the report of Daftari et al. [20]. The pathogenesis of IIH occurred after spinal surgery is still unclear. Sussman et al. proposed a new theory to explain IIH occurred after spinal trauma surgery: after spinal trauma, the spinal ligamentous and soft tissue are injured, spinal cord is swelling, and retro-pulsed bony fragments could contribute to the extrinsic

compression of the vertebral plexus; Then, the spinal fixation surgery further increases the existing soft tissue, ligament and spinal cord edema; Finally, the outflow of vertebral venous plexus is obstructed, which leads to the increase of venous sinus pressure and the onset of IIH [21]. Similarly, Kunes et al. proposed a hypothesis of IIH occurred after spinal deformity surgery: Distortion or displacement of vertebral venous plexus and/or the hyperfibrinogenemia resulting from the use of antifibrinolytics during spinal deformity surgery hinder the cranial venous return, which increase the intracranial pressure and lead to the development of IIH [22]. However, there is no direct and consistent evidence currently available to support these hypotheses and more experiments should be carried out to validate these hypotheses.

The manifested symptoms of IIH include headache, pulsatile tinnitus and papilledema. It may also include visual disturbances and diplopia, with the latter being secondary to sixth cranial nerve palsy [28, 29]. As the

classic symptoms of IIH just reflect generalized intracranial hypertension, the diagnosis of this rare disease is made after the exclusion of potential conditions that could lead to increased intracranial hypertension. Over the years, many sets of diagnostic criteria of IIH have been proposed [1, 7, 8]. In summary, the diagnostic evaluation should include an ophthalmologic funduscopic examination by an ophthalmologist, as papilledema is considered as a hallmark of IIH [1]. If papilledema is confirmed, cranial imaging should be performed immediately to exclude secondary causes of increased intracranial pressure. Once intracranial mass lesions, obstructive hydrocephalus and venous outflow obstruction have been excluded, a lumbar puncture should be performed to confirm increased intracranial pressure and rule out inflammatory or malignant pathology. Obviously, the diagnosis of the five cases in literature review and our two cases were diagnosed of IIH according to the process mentioned above.

The primary goal of IIH treatment is to prevent or arrest progressive visual loss, relieve headache and other symptoms of elevated intracranial pressure [3, 5]. The treatment should be carried out in collaboration with an ophthalmologist as the chief hazard of IIH is permanent visual loss due to papilledema. Lumbar puncture is not only establishing diagnosis but also could be used to reduce the net volume of CSF and then the intracranial pressure [9, 30]. Acetazolamide, which decreases the production of CSF, is used as the first-line drug for IIH treatment [31, 32]. Currently, there was no recommended schedule of acetazolamide, Thurtell et al. proposed a reasonable starting dose is 500 mg twice daily, gradually titrating up to a maximum of 4 g daily in twice-daily doses [4]. In another study, Skau et al. recommended the initial dose should be 250 mg twice a day and the dose gradually increases to 1000–1250 mg daily [24]. If acetazolamide fails or is not tolerated, diuretics combined with potassium is generally considered the second agent of choice [33, 34]. A short-term corticosteroids can also be considered in acute severe cases in which high-dose corticosteroid therapy (1 mg/kg, maximum 36 mg betamethasone) might be a good option to improve severe headache and rapid visual deterioration [35, 36]. Surgical intervention is considered for the patients who do not respond well to medical management or for those who have severe papilledema with vision loss [4, 37]. Surgical procedures used for the treatment of IIH include CSF shunting, subtemporal decompression, venous sinus stenting, and optic nerve fenestration [37, 38].

In the literature, IIH is known as a benign disease as it generally has a favorable long term outcome with prompt diagnosis and management. The headache, diplopia and papilloedema can be completely cured. However, vision loss tends to be permanent once the retinal nerve fiber layer loss and retinal ganglion cell atrophy develops [39, 40]. It

is reported that visual impairment progressively worsened in 10 % of patients who have received prompt intervention [41]. Another study reported 24.6 % IIH patients became blind in one or both eyes during a 5- to 41-year follow-up [42]. The five cases of IIH occurred after spinal surgery in the literature all had a good long term prognosis. However, there was no dead case of IIH reported. In our cases, the IIH patient occurred after spinal deformity died due to the irreversible intracranial hypertension although she underwent intensive care and treatment. So IIH occurred after spinal surgery can also be a lethal disorder, which the physicians should be aware.

Conclusion

We present two cases of IIH occurred after spine surgery. And a systematic review revealed that five cases of IIH occurred after spine surgery has been published in the English literature. IIH occurred after spinal surgery is a very uncommon condition that should be recognized by neurosurgeons and orthopedic surgeons, because prompt diagnosis and medical therapy can lead to a satisfactory clinical outcome. Also, IIH occurred after spinal surgery can be a lethal disorder, which the physicians should be aware.

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Compliance with ethical standards

Conflict of interest None of the authors has any potential conflict of interest.

References

- Friedman DI, Liu GT, Digre KB (2013) Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. *Neurology* 81:1159–1165. doi:[10.1212/WNL.0b013e3182a55f17](https://doi.org/10.1212/WNL.0b013e3182a55f17)
- Shin RK, Balcer LJ (2002) Idiopathic intracranial hypertension. *Curr Treat Options Neurol* 4:297–305
- Binder DK, Horton JC, Lawton MT, McDermott MW (2004) Idiopathic intracranial hypertension. *Neurosurgery* 54:538–551 (discussion 551–552)
- Thurtell MJ, Wall M (2013) Idiopathic intracranial hypertension (pseudotumor cerebri): recognition, treatment, and ongoing management. *Curr Treat Options Neurol* 15:1–12. doi:[10.1007/s11940-012-0207-4](https://doi.org/10.1007/s11940-012-0207-4)
- Bidot S, Bruce BB (2015) Update on the diagnosis and treatment of idiopathic intracranial hypertension. *Semin Neurol* 35:527–538. doi:[10.1055/s-0035-1563569](https://doi.org/10.1055/s-0035-1563569)
- Quincke H (1893) Meningitis serosa. *Sammel Klin Vortr* 67:1
- Smith JL (1985) Whence pseudotumor cerebri? *J Clin Neuropthalmol* 5:55–56

8. Friedman DI, Jacobson DM (2002) Diagnostic criteria for idiopathic intracranial hypertension. *Neurology* 59:1492–1495
9. Corbett JJ, Thompson HS (1989) The rational management of idiopathic intracranial hypertension. *Arch Neurol* 46:1049–1051
10. Brazis PW (2007) Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension. *Am J Ophthalmol* 143:683–684. doi:[10.1016/j.ajo.2007.01.001](https://doi.org/10.1016/j.ajo.2007.01.001)
11. Dandy WE (1937) Intracranial pressure without brain tumor: diagnosis and treatment. *Ann Surg* 106:492–513
12. Durcan FJ, Corbett JJ, Wall M (1988) The incidence of pseudotumor cerebri. Population studies in Iowa and Louisiana. *Arch Neurol* 45:875–877
13. Craig JJ, Mulholland DA, Gibson JM (2001) Idiopathic intracranial hypertension; incidence, presenting features and outcome in Northern Ireland (1991–1995). *Ulster Med J* 70:31–35
14. Chen J, Wall M (2014) Epidemiology and risk factors for idiopathic intracranial hypertension. *Int Ophthalmol Clin* 54:1–11. doi:[10.1097/IIO.0b013e3182aabf11](https://doi.org/10.1097/IIO.0b013e3182aabf11)
15. McCluskey G, Mulholland DA, McCarron P, McCarron MO (2015) Idiopathic Intracranial Hypertension in the Northwest of Northern Ireland: epidemiology and clinical management. *Neuroepidemiology* 45:34–39. doi:[10.1159/000435919](https://doi.org/10.1159/000435919)
16. Kesler A, Kupferminc M (2013) Idiopathic intracranial hypertension and pregnancy. *Clin Obstet Gynecol* 56:389–396. doi:[10.1097/GF.0b013e31828f2701](https://doi.org/10.1097/GF.0b013e31828f2701)
17. Falardeau J, Lobb BM, Golden S, Maxfield SD, Tanne E (2013) The use of acetazolamide during pregnancy in intracranial hypertension patients. *J Neuroophthalmol* 33:9–12. doi:[10.1097/WNO.0b013e3182594001](https://doi.org/10.1097/WNO.0b013e3182594001)
18. Krishnakumar D, Pickard JD, Czosnyka Z, Allen L, Parker A (2014) Idiopathic intracranial hypertension in childhood: pitfalls in diagnosis. *Dev Med Child Neurol* 56:749–755. doi:[10.1111/dmcn.12475](https://doi.org/10.1111/dmcn.12475)
19. Salpietro V, Chimentz R, Arrigo T, Ruggieri M (2013) Pediatric idiopathic intracranial hypertension and extreme childhood obesity: a role for weight gain. *J Pediatr* 162:1084. doi:[10.1016/j.jpeds.2013.01.048](https://doi.org/10.1016/j.jpeds.2013.01.048)
20. Daftari TK, Heller JG, Newman NJ (1995) Pseudotumor cerebri after occipitocervical arthrodesis and immobilization in a halo vest. A case report. *J Bone Joint Surg Am* 77:455–458
21. Sussman WI, Shaw E (2014) Intracranial hypertension after spinal cord injury and suboptimal cervical fusion. *PM R* 6:199–202. doi:[10.1016/j.pmrj.2013.08.595](https://doi.org/10.1016/j.pmrj.2013.08.595)
22. Kunes J, Thompson GH, Manjila S, Poe-Kochert C, Cohen AR (2011) Idiopathic intracranial hypertension following spinal deformity surgery in children. *Neurosurg Focus* 31:E20. doi:[10.3171/2011.7.FOCUS11160](https://doi.org/10.3171/2011.7.FOCUS11160)
23. De Simone R, Ranieri A, Bonavita V (2010) Advancement in idiopathic intracranial hypertension pathogenesis: focus on sinus venous stenosis. *Neurol Sci* 31(Suppl 1):S33–S39. doi:[10.1007/s10072-010-0271-z](https://doi.org/10.1007/s10072-010-0271-z)
24. Skau M, Brennum J, Gjerris F, Jensen R (2006) What is new about idiopathic intracranial hypertension? An updated review of mechanism and treatment. *Cephalgia* 26:384–399. doi:[10.1111/j.1468-2982.2005.01055.x](https://doi.org/10.1111/j.1468-2982.2005.01055.x)
25. Nedelmann M, Kaps M, Mueller-Forell W (2009) Venous obstruction and jugular valve insufficiency in idiopathic intracranial hypertension. *J Neurol* 256:964–969. doi:[10.1007/s00415-009-5056-z](https://doi.org/10.1007/s00415-009-5056-z)
26. Biouss V, Bruce BB, Newman NJ (2012) Update on the pathophysiology and management of idiopathic intracranial hypertension. *J Neurol Neurosurg Psychiatry* 83:488–494. doi:[10.1136/jnnp-2011-302029](https://doi.org/10.1136/jnnp-2011-302029)
27. Friesner D, Rosenman R, Lobb BM, Tanne E (2011) Idiopathic intracranial hypertension in the USA: the role of obesity in establishing prevalence and healthcare costs. *Obes Rev* 12:e372–e380. doi:[10.1111/j.1467-789X.2010.00799.x](https://doi.org/10.1111/j.1467-789X.2010.00799.x)
28. Patton N, Beatty S, Lloyd IC (2000) Bilateral sixth and fourth cranial nerve palsies in idiopathic intracranial hypertension. *J R Soc Med* 93:80–81
29. Cinciripini GS, Donahue S, Borchert MS (1999) Idiopathic intracranial hypertension in prepubertal pediatric patients: characteristics, treatment, and outcome. *Am J Ophthalmol* 127:178–182
30. Tang RA, Dorotheo EU, Schiffman JS, Bahrami HM (2004) Medical and surgical management of idiopathic intracranial hypertension in pregnancy. *Curr Neurol Neurosci Rep* 4:398–409
31. Supuran CT (2015) Acetazolamide for the treatment of idiopathic intracranial hypertension. *Expert Rev Neurother* 15:851–856. doi:[10.1586/14737175.2015.1066675](https://doi.org/10.1586/14737175.2015.1066675)
32. Johnson LN, Krohel GB, Madsen RW, March GA Jr (1998) The role of weight loss and acetazolamide in the treatment of idiopathic intracranial hypertension (pseudotumor cerebri). *Ophthalmology* 105:2313–2317. doi:[10.1016/S0161-6420\(98\)91234-9](https://doi.org/10.1016/S0161-6420(98)91234-9)
33. Victorio MC, Rothner AD (2013) Diagnosis and treatment of idiopathic intracranial hypertension (IIH) in children and adolescents. *Curr Neurol Neurosci Rep* 13:336. doi:[10.1007/s11910-012-0336-x](https://doi.org/10.1007/s11910-012-0336-x)
34. Kosmorsky GS (2014) Idiopathic intracranial hypertension: pseudotumor cerebri. *Headache* 54:389–393. doi:[10.1111/head.12284](https://doi.org/10.1111/head.12284)
35. El-Saadany WF, Farhoud A, Zidan I (2012) Lumboperitoneal shunt for idiopathic intracranial hypertension: patients' selection and outcome. *Neurosurg Rev* 35:239–243. doi:[10.1007/s10143-011-0350-5](https://doi.org/10.1007/s10143-011-0350-5) (discussion 243–244)
36. Rogers DL (2014) A review of pediatric idiopathic intracranial hypertension. *Pediatr Clin N Am* 61:579–590. doi:[10.1016/j.pcl.2014.03.004](https://doi.org/10.1016/j.pcl.2014.03.004)
37. Uretsky S (2009) Surgical interventions for idiopathic intracranial hypertension. *Curr Opin Ophthalmol* 20:451–455. doi:[10.1097/ICU.0b013e3283313c1c](https://doi.org/10.1097/ICU.0b013e3283313c1c)
38. Spitzke A, Malik A, Lee AG (2014) Surgical and endovascular interventions in idiopathic intracranial hypertension. *Curr Opin Neurol* 27:69–74. doi:[10.1097/WCO.0000000000000049](https://doi.org/10.1097/WCO.0000000000000049)
39. Rizzo JL, Lam KV, Wall M, Wilson MD, Keltner JL (2015) Perimetry, retinal nerve fiber layer thickness and papilledema grade after cerebrospinal fluid shunting in patients with idiopathic intracranial hypertension. *J Neuroophthalmol* 35:22–25. doi:[10.1097/WNO.0000000000000181](https://doi.org/10.1097/WNO.0000000000000181)
40. Dhungana S, Sharrack B, Woodroffe N (2010) Idiopathic intracranial hypertension. *Acta Neurol Scand* 121:71–82. doi:[10.1111/j.1600-0404.2009.01172.x](https://doi.org/10.1111/j.1600-0404.2009.01172.x)
41. Wall M, George D (1991) Idiopathic intracranial hypertension. A prospective study of 50 patients. *Brain* 114(Pt 1A):155–180
42. Corbett JJ, Savino PJ, Thompson HS, Kansu T, Schatz NJ, Orr LS, Hopson D (1982) Visual loss in pseudotumor cerebri. Follow-up of 57 patients from five to 41 years and a profile of 14 patients with permanent severe visual loss. *Arch Neurol* 39:461–474