Communicating Hydrocephalus and Vestibular Schwannomas: Etiology, Treatment, and Long-Term Follow-Up

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Abstract

Background Large vestibular schwannomas (VSs) can cause hydrocephalus by obstructing the fourth ventricle. Little is known about the communicating hydrocephalus that is seen with a smaller VS.

Methods The clinicopathological findings and follow up of three patients with communicating hydrocephalus associated with a small VS are presented.

Results Four patients aged 40 to 66 years (mean: 57.7) presented with ataxia, dementia, and urinary incontinence. The VS were 2.0 to 2.4 cm. The cerebrospinal fluid (CSF) protein was elevated in three patients in whom it was measured (1.7 to 6 times normal). The VS was resected in two patients. All of the patients required ventriculoperitoneal shunting (VPS). All of the patients were asymptomatic or improved at follow-up at 9 months to 13 years.

Conclusion Communicating hydrocephalus associated with a VS can occur in younger patients than was previously thought. An elevated CSF protein appears to be important, but other factors may be involved. A shunting procedure is often required to relieve the symptoms of hydrocephalus even if the tumor is resected. Possible etiological causes of communicating hydrocephalus in patients with a small VS are discussed.

Keywords

- vestibular schwannoma
- communicating hydrocephalus

Introduction

Vestibular schwannomas (VS) are benign tumors of the vestibular nerve that are usually diagnosed when they produce unilateral sensorineural hearing loss and tinnitus. VSs can protrude into the cerebellopontine angle (CPA) to compress the cerebellum, the fourth ventricle, the brainstem, and the seventh and fifth cranial nerves. Large VSs can also produce obstructive hydrocephalus by compressing the fourth ventricle

and obstructing the flow of cerebrospinal fluid (CSF). This type of hydrocephalus produces symptoms and signs of intracranial hypertension, such as headache, nausea and vomiting, depressed level of consciousness, and papilledema. In such cases these symptoms often resolve following the resection of the tumor and the resultant decompression of the CSF pathways. Smaller VSs can be associated with communicating, or non-obstructive, hydrocephalus and produce gait ataxia, memory loss, and urinary incontinence.^{1–4} This type of hydrocephalus

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in patients with a small VS may be more common than previously thought.⁵ However, only a handful of cases of patients with communicating hydrocephalus and a VS have been reported in detail, and their long-term treatment outcome is poorly documented. We report four such cases with a follow-up of 1.5 to 13 years and review the pertinent literature on this occurrence.

Materials and Methods

Between 1998 and 2011, 250 patients with a VS were treated at the Montreal Neurological Hospital and Institute by an interdisciplinary team composed of neurosurgeons, otolaryngologists, and radiation oncologists. Three of the 250 patients (1.2%) presented to hospital with symptomatic communicating hydrocephalus, and their cases are reviewed. Patients from our institution previously reported by Bloch and coworkers are not included in this study.² Patients with obstructive hydrocephalus and those with asymptomatic ventricular enlargement were excluded from study.

The pertinent literature published between 1950 to 2011 was retrieved through Google, PUBMED, and Ovid MEDLINE using “vestibular schwannoma,” “acoustic neuroma,” “cerebellopontine angle tumor,” “hydrocephalus,” “communicating,” and “nonobstructive” as search indicators, and was reviewed.

Case Reports: (►Table 1)

Case No. 1

A 62-year-old previously healthy man complained of gait instability and severe memory loss for a few months before admission. Neurological examination revealed decreased hearing in the left ear, a broad-based gait, memory loss, and confusion. Laboratory investigations were noncontributory. A lumbar puncture revealed an elevated CSF protein concentration of 0.87 g/L (normal: 0.20 to 0.50 g/L). Magnetic resonance imaging (MRI) demonstrated widening of the left porus acusticus, an extra-axial, gadolinium-enhancing $1.5 \times 1.6 \times 2.0$ cm lesion of the CPA and communicating hydrocephalus (►Fig. 1A, B). The patient underwent a suboccipital craniectomy and near total resection (►Fig. 1C) of what proved to be a moderately vascular VS. His presenting

complaints did not, however, resolve postoperatively, and repeat computed tomography (CT) imaging showed the persistence of communicating hydrocephalus. A ventriculoperitoneal shunt (VPS) was inserted 1 month after resection of the tumor, which led to complete resolution of his gait instability and reversal of his cognitive decline. His gait and memory function remain normal upon follow-up examination 13 years after surgery. MRI and CT scanning confirmed the absence of further tumor growth and stability of the mildly decreased ventricular size (►Fig. 1D).

Case No. 2

A 45-year-old woman gave a 5-month history of tinnitus, vertigo, decreased hearing, and gait instability with numerous falls. Neurological examination revealed decreased hearing in the left ear and a broad-based, unstable gait with a tendency to fall to the left. Laboratory investigations were unremarkable. MRI demonstrated widening of the left porus acusticus, an enhancing, extra-axial $1.1 \times 1.5 \times 2.1$ cm lesion of the left CPA and communicating hydrocephalus (►Fig. 2A, B). An extraventricular drain (EVD) was inserted preoperatively, and the ventricular CSF protein concentration was 0.40 g/L (normal: 0.05 to 0.15 g/L). A suboccipital craniectomy was performed and gross total resection of the VS was achieved (►Fig. 2C). Hemosiderin, likely the result of previous hemorrhage, was seen upon histopathological examination of the tumor (►Fig. 2E). Recordings of the intracranial pressure from the EVD remained in the normal range through the preoperative and postoperative phase, and it was removed on the first postoperative day. However the patient's gait instability did not improve and CT scanning revealed persistence of the communicating hydrocephalus. A VPS was inserted 22 days after craniotomy, which resulted in complete resolution of her ataxia. She remains asymptomatic 12 years after surgery. Follow-up MRI scans confirmed the absence of tumor recurrence and resolution of the communicating hydrocephalus (►Fig. 2D).

Case No. 3

A 66-year-old woman with a past history of relapsing, secondarily progressive multiple sclerosis suddenly lost hearing from the right ear 6 years before admission. A thick-cut, noninfused T2 and flair MRI performed at the time showed no new demyelinating plaques. There were no obvious lesions

Table 1 Clinical, Demographic, and Radiological Features of Four Patients with Communicating Hydrocephalus from a Vestibular Schwannoma

No. Age sex	Clinical presentation	Size (cm)	CSF protein (g/L)	Treatment	Outcome (years)
^a Case 1: 62 M	Ataxia Dementia	$1.5 \times 1.6 \times 2.0$	0.87	Resection VPS	Asymptomatic (13 years)
Case 2: 45 F	Ataxia	$1.1 \times 1.5 \times 2.1$	0.40	EVD Resection VPS	Asymptomatic (12 years)
Case 3: 66 F	Ataxia Dementia Incontinence	$1.3 \times .2.0 \times 2.4$	0.90	VPS	Improved (9 months)

^aCase 1: lumbar CSF, cases 2 and 3: Ventricular CSF.

Abbreviations: CSF, cerebrospinal fluid; EVD, external ventricular drainage; VPS, ventriculoperitoneal shunt.

Fig. 1 (Case No.1) Axial T1-weighted magnetic resonance imaging (MRI) showing (A) a 2-cm gadolinium-enhancing tumor within the left cerebellopontine angle and (B) communicating hydrocephalus. (C) A postoperative axial MRI confirmed the near total resection of the tumor, but the patient remained symptomatic from communicating hydrocephalus; (D) a computed tomography scan showed persistent ventricular enlargement. His symptoms resolved after ventriculoperitoneal shunting.

in the CPA or in the internal auditory canal. The patient developed gait ataxia, confusion, memory loss, and urinary incontinence 1 month preceding her admission to hospital. There was no history suggesting laterality of her ataxia and no focal signs were apparent on examination. Laboratory investigations were noncontributory. A lumbar puncture revealed a CSF protein concentration of 1.75 g/L (normal: 0.20 to 0.50 g/L). Prolonged lumbar drainage cause a marked improvement in her symptoms. MRI demonstrated widening of the right porus acusticus, a $1.3 \times 2.0 \times 2.4$ cm right CPA tumor, and communicating hydrocephalus (**Fig. 3A–C**). There was no appreciable compression of the fourth ventricle and clinically insignificant local impingement on the brain stem. Because her disabling symptoms were due to communicating hydrocephalus and the tumor had no significant mass effect, the VS was treated expectantly. VPS produced rapid and dramatic improvement in her gait and mild to moderate improvement in her cognitive functions, and she remains stable 1 year postoperatively. She, however, remains incontinent. Ventricular CSF obtained at the time of surgery had a protein concentration of 0.90 g/L (normal 0.05 to 0.15 g/L). A

postoperative MRI scan performed 9 months after surgery showed stability of the tumor and decrease in the ventricular size (**Fig. 3D**).

Discussion

The incidence of hydrocephalus associated with VS ranges from 3.7 to 23.5%, although higher incidences have been reported in smaller series.^{5–12} The occurrence of obstructive hydrocephalus and larger VSs appears straightforward, resulting from simple, mechanical obstruction of the fourth ventricle.^{4,11,12} Less is known about the communicating hydrocephalus that is seen in patients whose VS is too small to obstruct the outflow of CSF from the ventricular system. Only a few VS patients with this form of hydrocephalus have been reported in detail, and questions remain on the etiology of this association, on the treatment options available for patients in which it is present, and on the long-term evolution of treated patients. There are several factors that may be involved in the development of communicating hydrocephalus in patients with a VS. The most widely discussed is a high

Fig. 2 (Case No. 2) (A) Axial T1-weighted magnetic resonance imaging (MRI) demonstrating the presence of a gadolinium-enhancing 2.1-cm left cerebellopontine angle tumor. (B) A coronal T1-weighted MRI without gadolinium infusion also performed preoperatively demonstrates the presence of communicating hydrocephalus. (C) A postoperative T1-weighted MRI performed after the injection of contrast material demonstrating the gross total resection of the tumor and (D) resolution of the communicating hydrocephalus following ventriculoperitoneal shunting. (E) Iron stain of the tumor resected in Case 2. The blue pigment represents hemosiderin. Schwannoma cells are stained pink.

CSF protein concentration.^{2,3,13} However, tumor size, recurrent tumor bleeding, seeding of tumor cells through the CSF, age, and other factors may also play a role.^{4,5,11,14,15}

CSF Protein Concentration

The protein concentration within the CSF is distributed as a gradient: it is lowest in the ventricles, rises as it reaches the cisterns at the base of the brain, and is highest in the lumbar subarachnoid space.^{16,17} The CSF enters the venous system at the level of the arachnoid granulations through transendothelial pores or by pinocytosis across the endothelial cells.

The association of communicating hydrocephalus, an elevated CSF protein concentration, and a small VS was first reported by Gardner and associates in 1954. These authors suggested that the communicating hydrocephalus present in their patients was the result of “partial obstruction of the absorptive areas of the cerebrospinal fluid” by protein molecules secreted by the tumor.¹³ Subsequently, in 1977, Kühne and coworkers reported two similar cases.³ A third patient reported by Kühne et al also had communicating hydrocephalus associated with a small VS, but the protein concentration was “not elevated” in that case.³ Kühne and coworkers therefore concluded that the cause of the hydrocephalus in their cases was not due to an elevated CSF protein concentration.³ Bloch et al revived the topic in 2003 in a report on six patients.² All of their patients had an elevated ventricular CSF protein concentration, and they concluded that this impeded

the absorption of CSF at the level of the arachnoid granulations.²

Although proteins in the CSF may play a key role in the development of communicating hydrocephalus in patients with a small VS, the concentration of protein necessary to produce this is unknown. Varying CSF protein concentrations have been reported in patients with a VS and communicating hydrocephalus. Kühne and collaborators observed that the lumbar CSF protein concentrations were normal, mildly elevated, and less than twice normal in their three patients.³ Bloch and collaborators reported elevated protein concentrations in the ventricular CSF of their six cases² and concluded that a 2.5- to 3-fold increase in the ventricular CSF protein concentration is necessary to produce communicating hydrocephalus in patients with a small VS. Rogg et al have pointed out that in some cases the CSF protein concentrations may be up to 15 times normal.^{2,5} The CSF protein concentration in our patients was 1.7 to 6 times the upper limit of normal, in line with the concentrations reported by Kühne et al and Bloch and coworkers.

A markedly elevated CSF protein concentration could also, conceivably, thicken the CSF enough to impede its flow within the subarachnoid space to such an extent as to produce communicating hydrocephalus, independently of or in combination with a putative interference of CSF absorption at the arachnoid granulation. Such a mechanism appears highly conjectural except, possibly, for the most extreme cases

Fig. 3 (Case No.3) (A) Preoperative axial T1-weighted magnetic resonance imaging and (B & C) plain computed tomography (CT) scan demonstrating the presence of a gadolinium-enhancing right cerebellopontine angle vestibular schwannoma and communicating hydrocephalus. (D) A plain CT scan performed after ventriculoperitoneal shunting showed that the ventricles had decreased in size concomitant with improvement of the patient's symptoms. The vestibular schwannomas was not operated upon and was being managed expectantly.

with the highest CSF protein concentration. Such cases, however, have been reported in association with schwannomas of the cauda equina.¹⁸

None of our patients with communicating hydrocephalus had a purely intracanalicular VS, where the exposure of the tumor to the CSF is limited, and we are not aware of such a patient reported in the literature. This adds weight to the hypothesis that tumor proteins must have access to the CSF to produce communicating hydrocephalus in patients with a VS. This may have been the case in our third patient, who developed unilateral hearing loss from, presumably, a very small intracanalicular tumor 6 years before the onset of clinical and radiographic signs of communicating hydrocephalus associated with a now visible, VS occupying the CPA. Alternately, it might also indicate that intracanalicular tumors are too small to secrete significant amounts of protein into the CSF.

Our third patient is also noteworthy because of the very high protein concentrations present in her CSF, ranging, at different times and from different sites, from 3.5 to 6 times normal. The multiple sclerosis from which she suffered may

have contributed, at least in part, to these high protein concentrations. Finally, as regards this case, the disparity in the protein concentration in the lumbar and ventricular CSF samples bears mentioning. The range of values considered normal for ventricular CSF, which must be obtained via a very invasive procedure that is seldom performed in patients that are not severely ill, may not be as robust as the range of values for the lumbar CSF¹⁹; and this should be taken into account when assessing ventricular protein concentrations.

The presence of a high CSF protein concentration may be necessary for the development of communicating hydrocephalus in patients with a small VS, but it may not be sufficient, in isolation, to cause this condition. If a high CSF protein concentration were sufficient to do so, most, if not all, patients with a VS would develop communicating hydrocephalus because a high CSF protein concentration, sometimes as high as 1 g/L, is a hallmark of this tumor.^{20,21} Thus, the question remains: if an elevated CSF protein concentration is the sole cause of communicating hydrocephalus in patients with a VS, either by sludging of flow or by interfering with

absorption—or both—why don't most patients with this tumor have it, since most have elevated CSF proteins? A prospective study, now underway, may shed light on this question.⁹

Tumor Size

Tumor size plays an important role in the development of hydrocephalus in patients with a VS: tumors larger than 3 cm are associated with obstructive hydrocephalus and communicating hydrocephalus predominates in patients with tumors smaller than 3 cm.^{4–8,11,12,15,22}

It is widely held that the CSF protein concentration in patients with a VS is a function of the size of the tumor,²¹ but this is not the case in patients with communicating hydrocephalus and a small VS: Bloch and coworkers found that the two patients with the smallest tumors in their series had the highest CSF protein concentration, and the patient with the largest tumor had a protein level in the middle range.² Further, Fukuda et al, in a univariate analysis, observed that the size of the tumor and the CSF protein concentration were both related to the presence of hydrocephalus, but only the CSF protein concentration remained as a significant factor in the multiple logistic regression analysis of their data.⁸ Thus, the interplay of the size of the tumor, its intrinsic capacity to secrete proteins and the extent of its projection into the CPA is complex, and the role of these factors in the development of communicating hydrocephalus, in combination or in isolation, requires further elucidation.

Bleeding and Seeding

In addition to the presence of protein-rich CSF in the sub-arachnoid space, it has been suggested that recurrent bleeding from the tumor or seeding of tumor cells within the CSF might interfere with CSF reabsorption in patients with a VS.^{4,14,23} The presence of hemosiderin within our second patient's tumor suggests that her tumor might have bled one or more times previously and that this may have, in turn, contributed to the communicating hydrocephalus by interfering with CSF absorption. The presence of tumor cells within arachnoid granulations has not, to our knowledge, been described in patients with a VS.

CSF Pressure

It has been suggested that obstructive hydrocephalus in patients with a large VS might result from alterations of CSF flow dynamics within the basal cisterns.¹² This seems an unlikely mechanism for the production of communicating hydrocephalus in the presence of a smaller, unilateral tumor, as was the case for our patients and for other patients reported in the literature. Similarly, there seems to be no causal relationship between CSF pressure and communicating hydrocephalus in patients with a VS.²

Age and Other Factors

There are differing opinions on the relationship of age and hydrocephalus of both types in patients with a VS. Some have found that patients with hydrocephalus and a VS are older than patients without hydrocephalus,⁴ but others have been

unable to demonstrate such a correlation.^{5,8,11} Bloch et al, whose patients ranged in age from 71 years to 79 years (mean 76 years), have hypothesized that the association of communicating hydrocephalus and a small VS is a condition of the elderly because older patients would have a lessened ability to adapt to changes in CSF hydrodynamics and would, therefore, be more prone to develop hydrocephalus than younger patients.² Our patients, however, were not elderly. The youngest was 45 years old at the time of diagnosis and the oldest was 66 years (mean 57.7 years). As a group, our patients were almost two decades younger, on average, than the patients reported by Bloch and coworkers.

Gender and the side of the tumor seem to be irrelevant to the development of hydrocephalus in patients with a VS.^{5,8,11}

Association in the Absence of Causation

VSs are common tumors and idiopathic communicating hydrocephalus is a frequently encountered condition. Thus, the possibility that the two may be fortuitously associated cannot be excluded, especially in those patients whose symptoms of communicating hydrocephalus persist despite the resection of the VS. A prospective study may elucidate the nature of the relationship between the two conditions.

Management of Communicating Hydrocephalus in Patients with a Vestibular Schwannoma

The treatment of communicating hydrocephalus in a patient with a VS depends on the clinical presentation, the patient's age, and the presence or absence of medical comorbidities.

A shunting procedure and expectant management of the tumor would seem to be indicated for patients with communicating hydrocephalus whose tumor is small and is otherwise asymptomatic. The tumor can then be treated at a later date if it becomes symptomatic or enlarges. We followed this course in one of our patients, with satisfactory results. Concerns over the possibility of early shunt obstruction from the elevated CSF protein concentration have not materialized to date.

Primary tumor resection would seem to be indicated for younger, otherwise healthy patients with a symptomatic tumor because its resection may reverse the symptoms of communicating hydrocephalus. Primary tumor resection did not, however, alleviate the symptoms in two of our patients and they required a VPS. We recommend that a shunting procedure be performed expediently in patients who remain symptomatic after tumor resection because, by analogy with patients with idiopathic normal pressure hydrocephalus, the results of shunting seem to be worse the longer the patient has been symptomatic. An elevated ventricular protein concentration does not preclude a successful outcome from a shunting procedure.

In one of our cases an EVD was placed preoperatively even if the hydrocephalus was nonobstructive. This seemed prudent at the time to avoid any complications from an unexpected rise in intracranial pressure produced by induction of anesthesia in the presence of a posterior fossa tumor. Conversely, two of our patients did not have an external drain placed preoperatively and their surgery was uneventful.

However, the current consensus of opinion seems to favor the preoperative placement of an EVD in patients with a VS if either communicating or noncommunicating hydrocephalus is present.²⁴

Conclusions

A small VS can sometimes be associated with communicating hydrocephalus, probably from impaired absorption of CSF at the level of the arachnoid granulations or from altered hydrodynamics in the basal cisterns due to an elevated CSF protein concentration. The protein concentration necessary to produce communicating hydrocephalus is unknown but may be lower than previously thought, raising the possibility that other factors, such as bleeding from the tumor, may be involved. The occurrence of communicating hydrocephalus in association with a small VS is not restricted to elderly patients as previously thought. The tumor need not be resected to treat the hydrocephalus and the symptoms of communicating hydrocephalus may not respond to the resection of the tumor. Shunting of CSF is highly successful in such cases; and a very high protein content does not preclude a successful outcome.

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Conflict of Interest

None.

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