

Original Article

Acute subdural hematomas in shunted normal-pressure hydrocephalus patients – Management options and literature review: A case-based series

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Abstract

Background: Ventriculoperitoneal shunting (VPS) is considered a risk factor for developing subdural hematomas (SDH). Treating cases of acute SDH (aSDH) in shunted normal-pressure hydrocephalus (NPH) patients can be challenging, and data in this field are scarce. We report our experience treating shunted NPH patients presenting with aSDH.

Methods: Eight patients, aged 73 ± 6 years, with a history of VPS for NPH, hospitalized because of aSDH were included in this study. We retrospectively analyzed data regarding patients' clinical and radiological presentation, hospitalization course, the use of antithrombotics, and response to different treatment regimens.

Results: Four patients had pure aSDH, three had acute on chronic SDH, and one had subacute SDH. Patients presented with GCS 13–15 and various neurological signs, mainly confusion and unsteady gait. Two cases improved following resetting of their programmable shunt valve to its maximal pressure setting. Six cases improved after evacuation of the hematomas, five of them were operated a few days after initially resetting of the valve pressure. Three patients were discharged home, whereas five were referred to rehabilitation. Extended Glasgow Outcome Scale scores at discharge and during long-term follow-up were 5 and 7, respectively.

Conclusions: Treatment of patients with VPS for NPH, presenting with aSDH, may differ according to the neurological status, imaging, and clinical course. Treatment options include restricting shunt function, hematoma evacuation, or both.

Key Words: Acute subdural hematoma, hydrocephalus, shunt, treatment

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INTRODUCTION

Normal-pressure hydrocephalus (NPH) is characterized by the Hakim–Adams triad, including gait disturbances, memory decline, and urinary incontinence. Ventriculoperitoneal shunts (VPS) are commonly used for treatment of NPH.^[4,9,14] Cerebrospinal fluid (CSF) shunting is considered a risk factor for subdural hematomas (SDH), which may develop among 2–17% of patients with CSF shunts.^[2,7,15] It is thought that the decrease in size of the drained ventricles results in stretching of bridging veins in the subdural space, making them prone for bleeding following even minor traumas. Additionally, a shunt can decrease the size of the ventricles, allowing more space for the developing hematoma while avoiding a potential “tamponade” effect, thus creating a more favorable milieu for bleeds to expand. Factors that have been associated with SDH in VPS patients include the valve-type of the shunt system and the use of antiplatelet medications (APM) or anticoagulants.^[2,7,13] Lower valve settings predispose patients to overdrainage, increasing the risk for subdural hygromas and hematomas.^[12] In addition, patients with higher lumbar opening pressures before shunt insertion were found to have increased risk for SDH, probably resulting from a relatively stronger tendency toward overdrainage.^[12]

Managing cases of acute SDH (aSDH) in shunted NPH patients can be challenging because of the need for simultaneously addressing not only the hematoma itself but also the draining function of the shunt. The literature regarding handling of aSDH in patients with shunts is scarce and mainly based on case reports or small series.^[5,8,11] A recent paper described the treatment of 12 patients with ventriculoperitoneal and lumbo-peritoneal shunts who suffered from aSDH. Its main conclusion was that in cases that primarily underwent evacuation of the hematoma, treating the shunt by raising the valve pressure threshold or ligating it possibly reduced the rate of bleeding recurrence. On the other hand, patients initially treated only by intervening in their shunt systems (without treating the hematoma itself) occasionally needed a later evacuation of the hematoma.^[10]

As the data regarding this topic are limited, we chose to present our experience in treating NPH patients with VPS, who suffered from aSDH. An analysis and comparison of the different treatment options are discussed.

MATERIALS AND METHODS

We retrospectively reviewed the hospital records of adult (>18 years old) NPH patients with VPS, who suffered from aSDH, that were hospitalized in the Department of Neurosurgery at the Tel Aviv Medical

Center between the years 2010 and 2016. We included only primary NPH patients. Patients with acute on chronic or subacute bleeds were included too.

We did not include cases who presented with purely chronic bleeds or hypodense subdural effusions. Patients with intraparenchymal hematomas were also not included.

1. Eight patients with acute, acute on chronic, or subacute SDH were identified and analyzed for the following details: patients' age, gender, cause of hydrocephalus, and use of APM or anticoagulant medications prior to injury
2. Circumstances and timing of head injury, duration between symptom onset and admission, Glasgow Coma Scale (GCS), and neurological deficits on admission and during hospitalization were documented. The maximal width of the hematomas and midline shift (MLS) were measured on admission and during follow-up
3. Treatments and their timing during the hospitalization were recorded, including changing of the shunt's valve pressure setting, shunt ligation or externalization, the addition of shunt assist devices, and surgical procedures for evacuation of the hematomas
4. Outcome included discharge destination (home, rehabilitation, or nursing care) and Extended Glasgow Outcome Scale (GOSE) at discharge and follow-up.^[16] We also recorded the development of recurrent NPH-related symptoms, the need for additional shunt related surgeries, and the need for additional hematoma related surgeries.

The study was performed following an Institutional Review Board approval (0705-16-TLV) and in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki. For this type of retrospective study, informed consent was waived. The authors have no potential conflicts of interest to disclose. This study did not receive any specific grants from funding agencies in the public, commercial, or not-for-public sectors.

Statistics

This is an observational study, focusing on a small patients group; thus, no statistical analyses were done. Data are presented in a descriptive manner, and numerical data are presented as mean \pm standard deviation.

RESULTS

Patient background

Over a period of 7 years (2010–2016), 210 patients received a VPS for the diagnosis of NPH at our center. During the same period, eight of these patients were hospitalized with acute or subacute SDH. The average

Table 1: Individual patient's data

	Age, gender	Valve-type and pressure setting	Hemorrhage type, side	Hematoma width and MLS (mm) at presentation	Clinical presentation: GCS, neurological condition	Use of anticoagulation/ APM; treatment with procoagulant factors or PLTs	Intervention (days from admission)	Discharge destination and GOSE
1	85, M	Medos 150 mmH ₂ O	ASDH, Rt	3; 0	13, Confusion	Clopidogrel; PLT	Day 1: resetting valve pressure to 200 mmH ₂ O	Rehabilitation GOSE 5
2	64, M	Medos 180 mmH ₂ O	Acute on CSDH, Rt	13; 6	15, Lt arm pronation	No; no	Day 1: resetting valve pressure to 200 mmH ₂ O Day 5: burr hole, shunt assisted device	Home GOSE 7
3	74, M	Medos 200 mmH ₂ O	Acute on CSDH, Lt	30; 13	14, Confusion	No; no	Day 1: burr hole, shunt ligation	Home GOSE 7
4	71, M	Medos 150 mmH ₂ O	ASDH, Rt	15; 5	15, Lt facialis and hemiparesis	Clexane, thrombocytopenia; PLT	Day 1: resetting valve pressure to 200 mmH ₂ O	Rehabilitation, GOSE 5
5	68, M	Strata 1	ASDH, Lt	13; 4	15, Intact	Aspirin; PLT	Day 1: resetting valve pressure to 2.5 Day 12: burr hole	Rehabilitation GOSE 4
6	76, M	Medos 140 mmH ₂ O	Subacute SDH, Lt > Rt	Lt: 28 Rt: 16 MLS: 9	14, Confusion	Aspirin; PLT	Day 1: resetting valve pressure to 200 mmH ₂ O Day 3: bilateral burr holes Day 14: shunt ligation	Rehabilitation GOSE 4
7	75, M	Medos 150 mmH ₂ O	ASDH, Lt	24; 2	15, Unsteadiness	Aspirin; no	Day 1: resetting valve pressure to 200 mmH ₂ O Day 23: burr hole and shunt ligation	Home GOSE 5
8	73, M	Medos 80 mmH ₂ O	Acute on CSDH, Lt	14; 7	13, Confusion	Aspirin; no	Day 1: resetting valve pressure to 200 mmH ₂ O Day 18: burr hole	Rehabilitation GOSE 5

M=Male, Rt=Right, Lt=Left, NPH=Normal-pressure hydrocephalus, CSDH=Chronic subdural hematoma, aSDH=Acute subdural hematoma, MLS=Midline shift, APM=Antiplatelet medications, PLT=Platelets, GCS=Glasgow Coma Scale, GOSE=Extended Glasgow Outcome Scale.

age at presentation was 73 ± 6 years, ranging from 64 to 85 years of age. All patients were male.

Five patients were pretreated with APM, including four with aspirin and one with clopidogrel. APM were discontinued before shunt placement for at least 10 days and resumed within 1 month after discharge. One additional patient who had a history of lymphoma and was treated with enoxaparin also suffered from thrombocytopenia during the traumatic event [Table 1].

Patient history and baseline imaging

All patients had VPS inserted for 14 ± 15 months (range 3–48) prior to their current presentation. Two types of shunt valves were used: Medos programmable valves with siphonguard (Codman Inc.) programed to 80–200 mmH₂O (seven cases) and Strata valve (Medtronic Inc.) programed at 1 (one case). All shunts were located in the right lateral ventricle and were placed via a parieto-occipital entry [Table 1].

Baseline imaging, performed 5 months (range 2–11) prior to the current admission, showed no subdural bleeding. The indication for these CT scans was routine surveillance of shunted NPH patients to document radiological signs of overdrainage. Case 3 had bilateral subdural hygromas and

had insertion of shunt-assisted device (SAD) because of overdrainage syndrome 2 months prior to developing acute on chronic SDH. This patient did not have a new CT scan following the addition of the SAD and prior to admission with the bleed. The other seven cases had no evidence for subdural collections prior to their current admission.

Presentation on admission

Four patients had an aSDH following a fall on the day of admission. Three patients had an acute on chronic SDH, and one had subacute SDH, most of which were associated with recurrent falls over a period of several weeks to months with an acute deterioration just before their admission. GCS on admission ranged from 13 to 15 and the examination was mainly significant for non-focal signs, such as confusion (4) and unsteady gait (1), but also hemiparesis (2) and facial palsy (1).

Hematomas were located on the left (contralateral to the shunt) in five patients (one of which had also a small right bleed). Maximal hematoma width and MLS were 30 and 13 mm, respectively, both of which occurred in an NPH patient with acute on chronic SDH. In cases of left-sided (contralateral) SDH, the size of hematoma and MLS on admission was 22 ± 8 and 7 ± 4 mm, respectively, as compared with 10 ± 6 and 4 ± 3 mm on the right ipsilateral side [Table 1].

Management of clotting disturbances

Three of the five patients who were using APM, as well as one patient with lymphoma and thrombocytopenia that was also using enoxaparin, were given platelets on admission. In all cases, APM and anticoagulants were stopped on admission. Eventually, APM were discontinued permanently in two of the five previously treated patients, whereas it was recommenced in three of them. Enoxaparin treatment was recommenced before discharge for the prophylaxis of venous thrombosis in the case of lymphoma and thrombocytopenia [Table 1].

Management

There were no urgent craniotomies for aSDH. Burr hole (BH) evacuation of SDH was performed in six cases on days 1–23 of admission. BH was performed in all five cases in which the hematoma was contralateral to the shunt, as compared with only one of three cases, in whom the hematoma was ipsilateral to the shunt.

Patients were treated along three different pathways: increasing shunt valve pressure settings without evacuation of the hematoma (two cases), early BH with concurrent shunt adjustments on day of hospitalization (one case), and shunt adjustments followed by late BH (five cases).

Increasing shunt valve pressure setting

Two cases (1 and 4) improved following resetting shunt valves to their maximal pressures. They later developed recurrent NPH symptoms and had their valve pressures decreased. Their MLS and hematoma thickness on admission were no more than 5 and 15 mm, respectively [Table 1].

Early BH with concurrent shunt adjustments

One case (3) with acute on chronic SDH underwent a BH evacuation on the day of admission. This patient suffered from confusion and had a large hematoma of 30 mm and MLS of 13 mm. He showed substantial clinical improvement following BH and shunt ligation. One month later, he developed recurrent NPH-like symptoms necessitating reopening of the shunt system, with no need for any further interventions during a follow-up period of almost 2 years [Table 1].

Shunt adjustments followed by late BH

Five cases (2, 5, 6, 7, 8) had their shunt valve pressures adjusted on admission but underwent BH between days 5 and 23 of admission. The initial size of the hematoma and MLS was relatively large, ranging 13–28 and 2–9 mm, respectively. Three patients had increase in either the hematoma size or MLS before the BH. Three patients had clinical deterioration, whereas two had no improvement under conservative treatment and thus had the hematoma drained. All patients improved upon discharge. Shunt valve pressure settings had to be

later decreased in three cases because of recurrent NPH symptoms [Table 1].

Outcome

Three patients were discharged home and five were referred to rehabilitation. No residual or recurrent hematoma was documented in any of the cases over a median of 10 months (range: 1 month–4 years). All patients showed improvement in their clinical outcome compared to that on admission. GOSE on discharge was 5 and improved to 7 after a few months of follow-up [Table 1].

DISCUSSION

To our knowledge, this article is one of the first to describe management options in shunted NPH patients presenting with aSDH or subacute SDH. In this article, we did not explore or analyze risk factors associated with bleed occurrence, but rather the actual treatment options. The typical patient would be an adult (usually an elderly), often taking anticoagulants or APM, presenting in the context of falls (with or without a clear head injury event), with a nonspecific neurological decline. Patients presenting with mass-effect-related symptoms – such as declined consciousness or focal neurological signs, necessitate an urgent hematoma evacuation with a craniotomy. However, smaller hematomas, or those with no mass effect-related symptoms, may be followed or treated with restriction of the shunt function. Some of these patients may eventually undergo hematoma evacuation at a later stage – possibly with a BH. Regardless to the treatment of the hematoma, restriction of the shunt's function was part of the treatment for all patients. Thus, we recommend restriction of the shunt function by increasing the shunt pressure settings. Whether to externalize the shunt and temporarily close it, ligate the shunt, or drain the hematoma, and the timing of such a procedure, should be based on clinical and radiological findings at presentation and during follow-up.

Reviewing the literature, there are limited data regarding the actual treatment of VPS patients presenting with aSDH, which is not associated with overdrainage. Yet, our decision-making for each individual case was based on previous reports and their relevance to the clinical scenarios.^[5,8] Hoya *et al.* described 2 cases out of a series of 12 that improved after increasing shunt valve pressure settings, with both being considered as having relatively smaller hematomas. It is speculated that raising the valve pressure setting elevates intracranial pressure (ICP) just enough to prevent hematoma expansion but not too severe to induce high ICP signs.^[10] The assumption is that in cases of communicating hydrocephalus as in NPH patients, decreasing ventricular shunt drainage would force CSF to spread into alternative routes,

such as the basal cisterns, thus ameliorating the buildup of intraventricular pressure.^[17] A recent large prospective study among shunted NPH patients showed that the majority of cases with chronic hygromas or SDH in patients with adjustable valves were managed conservatively by readjusting the opening pressures. In comparison, most patients with fixed valves had to undergo surgical interventions, including BH and shunt ligation. Long-term survival was similar among the groups.^[15] It is important to stress that this study did not focus on aSDH, but rather on SDH and hygromas, possibly related to overdrainage.

Interestingly, in our series, BH was more commonly done among patients whose hematomas were contralateral to the side of the shunt and were mostly located on the left hemisphere. Theoretically, patients with hematomas that are ipsilateral to the side of their shunts might benefit clinically from the shunt drainage that lowers the pressure on the side of the hematoma, thus decreasing its impact on brain parenchyma as well as MLS to the contralateral side, provoking less neurological changes. This theory may receive support by the fact that the sizes of SDH and MLS were bigger in the contralateral group, though not estimated statistically because of the small series size. However, it may of course result from the fact that left hemispherical bleeding causes more significant neurological changes by affecting critical functions on the dominant side, thus triggering more active interventions. Determination of the drainage side was not affected by reluctance to operate on the shunted side, but by clinical–radiological considerations.

The influence of long-term use of APM or anticoagulants on the risk of SDH among patients with VPS is a matter of debate. A recent report found a significantly increased rate of SDH among NPH patients who were using aspirin and underwent a VPS.^[2] On the other hand, the chronic use of warfarin was found to be safe in a series of 15 NPH patients who underwent VPS insertion, as the rate of SDH was not higher than in other reports.^[7]

As it is unclear whether APM actually increase the risk of intracranial hematoma expansion,^[6] and as having a shunt confers risk for hematomas, we agree with previous reports and recommend holding antiplatelets on day of admission. We often recommence them later, based on following imaging findings and clinical judgment.^[3] Whether platelets transfusions contribute to the treatment of SDH in general is still controversial;^[1,6] therefore, we believe that further data on their use in our specific population are imperative.

Study limitations

The main limitations of our study are the small series size, as well as the retrospective nature of the study, which hamper the possibility of drawing any statistically based conclusions.

CONCLUSIONS

When treating aSDH in NPH-shunted patients, one should take into account the combined effects of both the hematoma mass effect and the CSF drainage by the shunt. Increasing the shunt valve pressure setting might be effective for some; however, these patients should be followed carefully as most will also require evacuation of their hematomas. Whether and when to evacuate the bleed are based on clinical and radiological findings and dynamics. Last, long-term follow-up is important, as some patients may need to readjust their shunt because of the recurrence of NPH-related symptoms.

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None.

Ethical approval

The study was performed following an Institutional Review Board (IRB) approval (0705-16-TLV) and in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Declaration of patient consent

For this type of retrospective study, informed consent was waived.

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Conflicts of interest

There are no conflicts of interest.

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