International Surgery Journal
Stolevski V et al. Int Surg J. 2024 Dec;11(12):2121-2125
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20243558

Case Report

Isolated expressive aphasia caused by acute subdural hematoma: case report and literature review

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Received: 25 October 2024
Accepted: 18 November 2024

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ABSTRACT

The occurrence of aphasia as an isolated and dominant clinical feature of acute subdural hematoma (aSDH) has been rarely mentioned in the available literature. Being the most common focal intracranial lesion, subdural hematomas (SDH) pose a clinical challenge, especially when presented with unorthodox clinical features. We report a case of a patient with a traumatic aSDH, presenting with intense frontal headaches and normal neurological examination. On the 3rd postadmission day, he developed expressive dysphasia which progressed to aphasia, with neurological examination findings of an upper motor neuron lesion (UMNL), without hematoma expansion, verified via head CT (computed tomography). The patient underwent decompressive craniotomy and hematoma evacuation. Early speech improvements were noted immediately postoperatively, and at hospital discharge the patient had no evident clinical features of speech disorder and neurological deficits or tests suggesting UMNL. Speech disorders in the setting of an aSDH need to be further investigated and potentially considered as an indication for surgical management, especially in rare instances where they present as an isolated clinical feature and conservative treatment is initially considered.

Keywords: Aphasia, Acute subdural hematoma, Craniotomy, Case report

INTRODUCTION

Neurogenic language disorder in the form of isolated dysphasia/aphasia as the dominant clinical symptom and sign of acute subdural hematoma has seldom been reported and investigated in clinical practice, thus its clinical significance in deciding the most adequate type of treatment is of great importance. Aphasia/dysphasia represents an impairment in language production and/or comprehension due to an acquired brain lesion.[1]Acute subdural hematomas are the most common focal intracranial lesion being reported even as nearly half of all intracranial hemorrhages.[2,3] Having an overall mortality rate of 66% and 19% functional recovery, the most adequate treatment of aSDH still presents a challenge, especially when patients present with unusual clinical features, as in our case, with isolated expressive aphasia.[4] This scientific paper aims to summarize the rarity of isolated language disorders caused by aSDH and suggest the possibility for potential modifications in the treatment guidelines regarding this clinical entity.

CASE REPORT

A 64-year-old male patient presented to the neurosurgery department, with a 2-day history of intense frontal headache (unresponsive to over-the-counter analgesics) following a minor head trauma to the left temporoparietal region. The neurological examination we performed revealed no abnormalities. The patient had undergone conservative treatment for left segmental pulmonary thromboembolism (5 years ago) and is subsequently receiving oral anticoagulant therapy (vitamin K antagonist - acenocoumarol).

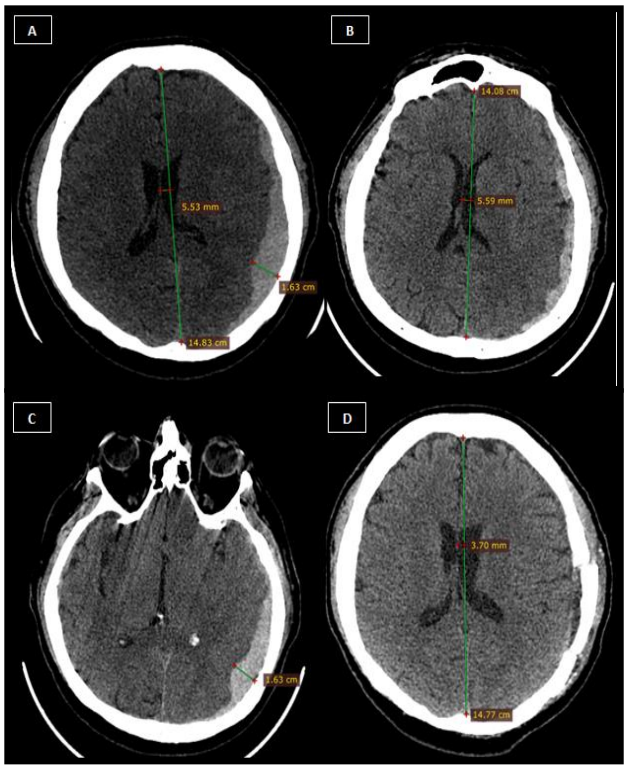

Figure 1: Head CT scan. (A) axial head CT scan image showing a left-sided acute temporoparietal subdural hematoma with 1.63 cm thickness and 5.53 mm midline shift – admission CT scan. (B) axial head CT scan image showing a progression of the midline shift to 5.59 mm – CT scan right before surgery. (C) axial head CT scan image showing a left-sided acute temporoparietal subdural hematoma without further hematoma expansion – CT scan right before surgery. (D) axial head CT scan image after craniotomy, with reduced midline shift to 3.70 mm and without any residual hematoma – 7 days postoperative CT scan.

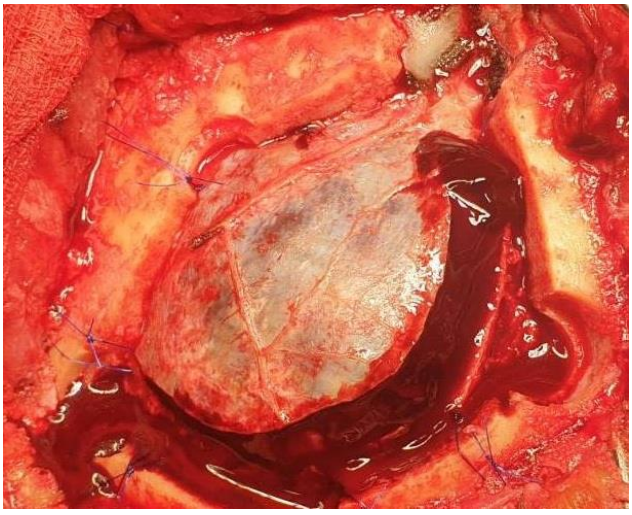

Figure 2: Intraoperative findings. Acute subdural hematoma visualized after temporoparietal craniotomy and dural opening.

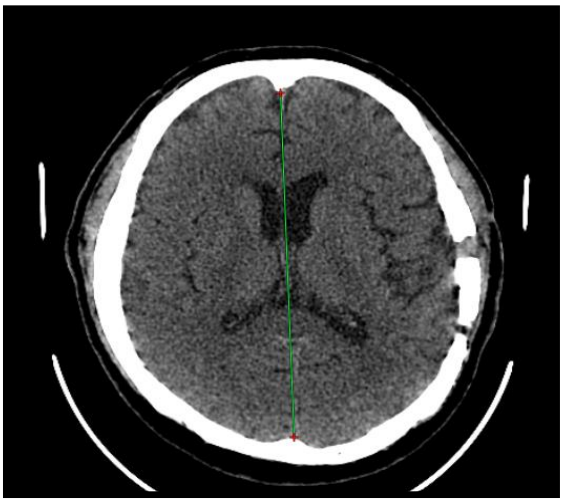

Figure 3: Head CT scan – 7 months postoperative. Axial image showing no midline shift or residual hematoma/hygroma or any type of intracranial collection or lesion.

Furthermore, he is receiving antihypertensive (angiotensin II receptor blocker – losartan), diuretic (loop diuretic – furosemide), and antilipid (statin - rosuvastatin) therapy, and has chronic venous insufficiency and varicose veins of his lower extremities for more than 15 years. We performed a head CT and a left-sided temporoparietal acute subdural hematoma was revealed, with a thickness of 16.3 mm and midline shift of 5.53 mm (Figure 1A). The patient was immediately admitted to the intensive care unit and was put under constant non-invasive monitoring, with normal vital parameters and normal neurological examination in the following 2 days, and control head CT findings identical to the initial one.

Furthermore, anticoagulant reversal was immediately performed with vitamin K and fresh frozen plasma in adequate doses, considering his past medical history. The results of the hemostasis screening test, coagulation profile, and international normalized ratio (INR) were normal and within reference ranges. On the 3rd postadmission day, he developed expressive dysphasia which progressed to aphasia in no more than 90 minutes. In addition to the expressive aphasia, the neurological examination revealed a positive Babinski reflex on the right foot and absent superficial abdominal reflexes on the right side, suggesting UMNL, without any additional neurological deficits.

A control head CT revealed a slight increase in the midline shift (5.59 mm) (Figure 1B, 1C), with identical clot thickness as the initial CT. Due to the rapid worsening of the patient's speech abilities and neurological tests, and the increase in midline shift on head CT, we immediately changed our treatment plan and the patient underwent a left temporoparietal craniotomy and hematoma evacuation (Figure 2). Early speech improvements were noted immediately postoperatively after early extubation, presented as expressive dysphasia,

with no new or added neurological deficits. In the following 2 days the dysphasia diminished completely and the patient had no evident clinical features of any speech disorder. He was fully mobilized on the 3rd postoperative day and low-molecular-weight-heparin thromboprophylaxis was initiated, in adequate doses, considering his medical history. The patient was discharged on the 5th postoperative day in a stable condition, with normal vital parameters, no neurological deficits or tests/signs/reflexes suggesting UMNL or speech disorders, and adequate operative wound healing. A control head CT was performed 6 days after discharge and revealed a reduced midline shift (3.70 mm), without residual hematoma (Figure 1 D). Regular follow-ups and continuation of oral anticoagulant therapy were recommended under transfusion medicine specialist supervision. During our regular monthly follow-ups, the patient did not report any changes or problems, and the neurological examinations were normal. A control head CT was performed after 7 months, showing no presence of a midline shift or residual hematoma/hygroma (Figure 3). Further follow-ups are adequately scheduled.

DISCUSSION

Regarding the etiology of aSDH, numerous causes are mentioned such as head trauma, coagulopathy/medical anticoagulation (sometimes without a history of trauma), thrombolysis, postsurgical, intracranial hypotension, shaken baby syndrome, spontaneous and cocaine-related.[5-9] The two most common causes of acute SDH are accumulation around parenchymal laceration and tearing of surface or bridging vessels from cerebral acceleration-deceleration during violent head motion.[7] In addition to the neurological examination, the head CT is a gold standard for diagnosing aSDH.[10]

Regarding surgical management, Bullock et al, emphasized the indications and timing of surgery, and also the adequate surgical methods, although various reasons such as poor or unfavorable initial medical status, past medical history, family wishes and patient's will can affect our decision making process in order to initially choose a conservative approach, especially in patients with normal neurological examination findings.[11] In our extensive literature review, only 3 cases of aSDH associated with isolated neurogenic language disorder were identified, as summarized in Table 1.[12-14] All patients were males, and the average age was 67.6 years (range, 55-86 years). Intracranial decompression was achieved by performing craniotomy and hematoma evacuation in all cases, with addition of aneurysm excision in one of the cases as reported by Vajramani et al. (no craniectomies were performed).[14] As emphasized by Aoki et al and Vajramani et al, the speech recuperation phase lasted 3 months and 24 days to full speech recovery, respectively.[12,14] Furthermore, Mishriki et al. did not report the recovery interval, but they reported full speech recovery, nonetheless.[13] The time intervals of neurogenic language disorder recovery associated with aSDH are variant, as our literature review has presented. Comparing our findings with rapid full speech recovery while considering the full recovery in all known cases, we present the potential need to contemplate the significance of language/speech disorders (especially isolated) in the setting of aSDH surgical treatment. In addition, the necessity for adequate postoperative rehabilitation with patient-specific speech and language therapy should be evaluated and accordingly applied to obtain ultimate outcomes. The role of the neurological examination is of great significance in promptly defining the best treatment option, as presented in our case. As our patient developed positive right-sided Babinski reflex and absent right-sided superficial abdominal reflexes, in addition to the expressive dysphasia progressing to aphasia, there was a high suspicion of a UMNL lesion related to hematoma expansion or brain edema.[15] Intracranial decompression was immediately considered based on the abnormal neurological examination and previous CT scan findings, even before performing a control head CT (findings of a slight increase in midline shift versus the previous CT scan–0.06 mm).

The speech disturbance resulted in early consideration of intracranial decompression by craniotomy and hematoma evacuation, with the consequent complete resolution of symptoms postoperatively, confirming the aSDH as the etiology of the expressive aphasia. Nevertheless, numerous cases in the available literature show conservative treatment with spontaneous resolution of aSDH and good/fair outcomes.[16-21] Still, outcomes and prognosis differ based on the specific case/patient, and most importantly, the aforementioned cases of aSDH were not associated with isolated speech disorders, which ultimately is our main topic of discussion due to its rarity and specificity. Neurogenic language disorders manifested in the setting of an aSDH need to be further investigated and reported, as well as their treatment and prognosis, especially when the initial neurological examination findings are normal and conservative treatment is planned.

Table 1: Articles concerning aphasia caused by aSDH.

Reference	No of cases	Type of aphasia	Age (in years)	Sex	Treatment	Aphasia duration	Aphasia resolution
Aoki et al[12]	1	Amnestic	55	Male	Craniotomy+hematoma evacuation	3 months	Complete
Mishriki et al[13]	1	Nonfluent	86	Male	Craniotomy+hematoma evacuation	-	Complete
Vajramani et al[14]	1	Bilingual	62	Male	Craniotomy+hematoma evacuation+aneurysm excision	24 days	Complete

CONCLUSION

Isolated language and speech disorders associated with an aSDH are a rare clinical scenario, thus pose a challenge in choosing the most adequate treatment method. In the reviewed literature as well as in our case, intracranial decompression via craniotomy and hematoma evacuation resulted in full speech recovery and is the preferred method of treatment, with head CT scan being the gold standard for diagnosis, accompanied by thorough and frequent neurological examinations. The prognosis might be uncertain, as the speech recovery and rehabilitation phase can last from days to months, depending on the specific case. Further investigation regarding isolated neurogenic language disorders caused by aSDH is needed, while considering additional neurological status worsenings, hematoma dimensions and expansion, and midline shift as crucial factors in choosing surgical treatment.

Additional investigations of this subject might lead to potential consideration for inclusion of this rare clinical scenario in the current treatment protocols for surgical management of aSDH, according to the available literature and our case. Such an addition might be substantial for clinicians when choosing the most adequate treatment for such rare scenarios in neurotrauma, especially in patients with poor initial medical status and past medical history where decision making sometimes has to be based on previous experiences, family wishes and patient's will.

The abovementioned cases (including our case) should serve as a reminder for clinicians when conservative treatment is considered, to always keep in mind the risk of potentially permanent language deficits and to contemplate surgical treatment as the best therapeutic option for the specific patient.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Stolevski V, Zdravkov I, Dimovski A. Isolated expressive aphasia caused by acute subdural hematoma: case report and literature review. Int Surg J 2024;11:2121-5.