Non Traumatic Acute Subdural Hematoma Revealing an Intracranial Arteriovenous Malformations: Case Report and Review of Literature

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ABSTRACT

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Intracranial arteriovenous malformation (AVM) is an abnormal connection of blood vessels, arteries and veins without capillary bed or neural parenchyma. This condition is commonly reveqlled by seizures, and also intraparenchy mal, subarachnoid, and intraventricular hemorrhage rhage. Subdural haematoma is rarely associated with a dural arteriovenous malformation. We report a case of a 53 - year - old man who was admitted to the emergency department with sudden loss of consciousness and coma. The brain CT Scan and CT angiography show left frontal intraparenchymal hematoma of 35 X 43 X 25 mm associated with left hemispheric subdural hematoma. The patient completely recovered after decompressive craniotomy followed by the embolization of the nidus.

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I. INTRODUCTION

Subdural hematomas (SDHs) are the most common form of intracranial injuries that occur as a result of head trauma. SDHs result from rupture of the veins traversing the subdural space en route to the venous sinuses of the dura mater. This condition is rarely found to be associated with arteriovenous malformation (AVM). These have been classified angiographically on the basis of their arterial supply into pial, dural, and mixed pial-dural types. The dural type comprises 10 to 15% of intracranial arteriovenous malformations (1,2). Dural arteriovenous fistulae of the anterior cranial fossa are rare (5.8%) but they have an usually high incidence of sudden massive intracranial hemorrhage (62-91%) (3–5). Its association with the ASDH is uncommon. The clinical presentation in most cases is headache and the treatment of dural AVM with cortical venous drainage is aimed at occlusion of the venous drainage or occlusion of all arterial supply, and can be surgical, endovascular, or a combination of both.

We report an unusual case of dural AVM presenting as acute subdural hematoma (ASDH) and discuss the clinical features and treatment options with the aid of a literature review.

II. CASE PRESENTATION

A 53-year-old man with a past medical history of hypothyroidism treated with levothyrox was admitted to the emergency department with sudden loss of consciousness without seizures. The physical examination found a comatose patient with a Glasgow Coma Scale (GCS) of 7/15, he has a stable respiratory rate as well as hemodynamic, the pupillary are equal in size and reactive. His body temperature was 37.5° C, with normal laboratory blood work up. There was no neurological deficit.

The brain CT Scan showed left frontal spontaneously hyperdense lesion measuring about 46 X 33 X 20 mm being the intracerebral hemorrhage (ICH) associated with acute subdural hematoma

(ASDH) of the left convexity about 12 mm of thickness and having mass effect on the homolateral lateral ventricle with the middle line shift up to 8 mm followed by brain herniation under false cerebri (Fig 1 A/). CT angiography confirms the previous findings and evaluates the ICH volume to be 19 cm^3. At the arterial time acquisition there was a central lesion to the intraparenchymal hematoma that was brightly enhanced with some drainage veins toward the superior longitudinal sinus making the suspicion of a possible dural arteriovenous fistula.

The patient was rushed to the operating theater where he underwent surgery for the evacuation of the compressive subdural hematoma and the ICH by the decompressive craniotomy of 14 X 12 cm bone flap. He did well in the post - operative period by regaining consciousness with a GCS of 15/15 without sensitive nor motor palsy (Fig 1 B/).

After dealing successfully with the emergency condition, a brain MRI and cerebral arteriography was done that confirmed the diagnosis of left frontoinsular AVM made of 2.5 cm nidus with the feeding arteries form the frontal branches of the middle cerebral artery and its drainage was mainly through the frontal and cortical veins of the middle cerebral vein into the superior longitudinal sinus (Fig 2). The patient underwent endovascular treatment. The nidus is catheterized with a Marathon microcatheter followed by injection of a total of 1.5 cc of Onyx. A homogeneous and compact filling of the nidus is obtained with the beginning of reflux in the drainage veins. The controls show a total exclusion of the nidus with restoration of venous return (Fig 3). The postoperative period was eventless and the patient was discharged 5 days later. He was doing well at the follow-up appointment of 3 months.

Table 1: Non-traumatic acute subdural hematoma revealing an intracranial arteriovenous malformations

1st author/ year	Age/ Sexe	Clinical presentation	ICH	SDH	AVM	Treatment	Outcome
Ogawa/ 2010 (6)	27/M	Headache	No	Convexity	Convexity	Surgical	Improved
Duffau/ 1999 (7)	64/M	Headache	Frontal	Frontal	ACF	Surgical	Improved
Kitazono/ 2010 (8)	68/M	Headache	Occipital	Occipital	Occipital	Surgical	Improved
Ito/ 1983 (9)	64/M	Unconsciousness	No	Frontal	ACF	Surgical	Improved
Kohyama/ 2009 (10)	60/M	Headache	No	Convexity	Convexity	Endovascular & Surgical	Improved
Kominato/ 2004 (11)	42/F	Unconsciousness & Coma	Temporal	Convexity	Temporal	None	Died
Duffau/ 1999 (7)	64/M	Hemiparesis	Temporal	Temporal	Temporal	Endovascular & Surgery	Died
Halbach/ 1988 (12)	48/F	Headache & Weakness	No	Convexity	Convexity	Endovascular	Improved
Ogawa/2010 (6)	27/M	Headache	Convexity	Convexity	Convexity	Endovascular	Improved
Saito/2014 (13)	56/M	Unconsciousness & Coma	Occipital	Occipital	Occipital	Endovascular & Surgery	Improved
Choi et Cho/2010 (5)	85/M	Unconsciousness	Frontal	Frontal	ACF	Surgical	Improved
Maiuri/ 2001 (4)	59/F	Headache, visual disturbances and loss of memory	No	Parietal	Occipital	Endovascular	Improved
Rengachary/1981 (14)	50/M	Grand mal seizure & Coma	No	Convexity	Temporal	None	Died
Rengachary/1981 (14)	61/M	TBI & Unconsciousness	No	Convexity	ACF	None	Died
Li/2019 (15)	45/M	Headache	No	Convexity	Occipital	Endovascular	Improved
Solis/1977 (16)	48/M	Headache & Severe right facial pain	No	Occipital	Occipital	Surgery	Improved

ACF = anterior cranial fossa, AVM = arteriovenous malformation, SDH = subdural hematoma

III. DISCUSSION

To discuss the clinical features and treatment options, we reviewed 16 cases of nontraumatic acute subdural hematoma revealing intracranial arteriovenous malformations from the previous literature. (table 1). Nine of the 16 cases were presented with headache and 5 with loss of consciousness while only one clinical presentation was seizure. In our present case, the patient never complained of headache but presented with a sudden loss of consciousness and coma. This is obviously secondary to the intracranial hypertension caused by the ICH and the ASDH. Thus, there is no specific clinical presentation for the intracranial AVM in the setting of ASDH. Saito et al. explained the ASDH by the theory of venous ectasias that might have had a fragile venous wall and was suspected of being a rupture point. The hemorrhagic pattern showed that the dominant location was subdural hematoma associated with a small amount of subcortical hematoma. The hemorrhagic point might be the subpial cortical vein draining into the superior sagittal sinus under venous high pressure due to arterial shunt flow. Rupture of the subpial vein might cause both laceration of arachnoid and cortical surfaces. A further hypothesis is that venous high pressure might aggravate cortical reflux and partial venous congestion might cause limited subcortical hemorrhage and simultaneously rupture at the fragile wall of the venous ectasias in the subdural space. Three patients out of the sixteen underwent surgery coupled with endovascular treatment, and six of them benefit from surgery with good outcome, while four had endovascular treatment exactly like the patient of our present case that underwent endovascular treatment with an uneventful postoperative period. Surgical, endovascular, and radiosurgical management of AVMs depends on the size of the nidus and also its location; whether it is located in a functional or not functional brain structure. Our review has shown that a good outcome can be reached by the combination of these therapeutic means.

IV. CONCLUSION

AVMs have various clinical manifestations, including acute subdural hematoma which is a mode of revelation rarely reported in the literature and for which diagnostic and therapeutic management remains of major interest for the patient's vital prognosis. Concerning our patient, initially admitted in critical neurological condition, he underwent surgical treatment combined with early endovascular treatment with full recovery.

Disclosure

The authors did not receive any funding for the preparation of this case report.

This article is an original work that is not being considered or reviewed by any other publication, and has not been published elsewhere in the same or a similar form.

All authors of the manuscript have read and agreed to its content and are accountable for all aspects of the accuracy and integrity of the manuscript.

Informed Consent: The patient gave his informed consent to publish his case.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

Ethics and reporting guidelines

Informed consent and verbal permission were obtained from the patient prior to the submission of this article. Also, this article respects both the Consensus based Clinical Case Reporting Guideline and the Recomme- ndations for the Conducting, Reporting, Editi- ng, and Publication of Scholarly Work in Medical Journals.

REFERENCES

1. Houser OW, Baker Jr HL, Rhoton Jr AL, Okazaki H. Intracranial dural arterioveno- us malformations. Radiology.1972;105(1):55–64.
2. Rosenblum B, Oldfield EH, Doppman JL, Di Chiro G. Spinal arteriovenous malformations: a comparison of dural arteriovenous fistulas and intradural AVM's in 81 patients. Journal of neurosurgery.1987;67 (6):795–802.
3. Halbach VV, Higashida RT, Hieshima GB, Wilson CB, Barnwell SL, Dowd CF. Dural arteriovenous fistulas supplied by ethmoidal arteries. Neurosurgery. 1990;26(5):8 16–23.

4. Maiuri F, Iaconetta G, Sardo L, Briganti F. Dural arteriovenous malformation associated with recurrent subdural haematoma and intracranial hypertension. British journal of neurosurgery. 2001;15(3):273– 6.
5. Choi HJ, Cho CW. Anterior cranial fossa dural arteriovenous fistulae presenting as subdural hematoma. Journal of Korean Neurosurgical Society. 2010;47(2):155.
6. Ogawa K, Oishi M, Mizutani T, Maejima S, Mori T. Dural arteriovenous fistula on the convexity presenting with pure acute subdural hematoma. Acta neurologica belgica. 2010;110(2):190 .
7. Duffau H, Lopes M, Janosevic V, Sichez J-P, Faillot T, Capelle L, et al. Early reble- eding from intracranial dural arteriovenous fistulas: report of 20 cases and review of the literature. Journal of neurosurgery. 1999;90(1):78–84.
8. Kitazono M, Yamane K, Toyota A, Okita S, Kumano K, Hashimoto N. A case of dural arteriovenous fistula associated with subcortical and subdural hemorrhage. No shi- nkei geka Neurological surgery. 2010;38 (8): 757–62.
9. Ito J, Imamura H, Kobayashi K, Tsuchida T, Sato S. Dural arteriovenous malformations of the base of the anterior cranial fossa. Neuroradiology. 1983;24(3):149–54.
10. Kohyama S, Ishihara S, Yamane F, Kanazawa R, Ishihara H. Dural arteriovenous fistula presenting as an acute subdural hemorrhage that subsequently progressed to a chronic subdural hemorrhage: case report. Minimally Invasive Neurosurg ery . 2009;52(01):36–8.
11. Kominato Y, Matsui K, Hata Y, Matsui K, Kuwayama N, Ishizawa S, et al. Acute subdural hematoma due to arteriovenous malformation primarily in dura mater: a case report. Legal Medicine. 2004;6(4):256–60.
12. Halbach VV, Higashida RT, Hieshima GB, Rosenblum M, Cahan L. Treatment of dural arteriovenous malformations involving the superior sagittal sinus. American journal of neuroradiology. 1988;9(2):337– 43.
13. Saito A, Kawaguchi T, Sasaki T, Nishijima M. A case of dural arteriovenous fistula presenting as acute subdural hematoma. Case reports in neurology. 2014;6(1):122– 5.
14. Rengachary SS, Szymanski DC. Subdural hematomas of arterial origin. Neurosurgery .1981;8(2):166–72.
15. Li G, Zhang Y, Zhao J, Zhu X, Yu J, Hou K. Isolated subdural hematoma secondary to Dural arteriovenous fistula: a case report and literature review. BMC neurology.20 19;19(1): 1–6.
16. Solis OJ, Davis KR, Ellis GT. Dural arteriovenous malformation associated with subdural and intracerebral hematoma : a CT scan and angiographic correlation. Computeri zed tomography. 1977;1(2):145 –50.

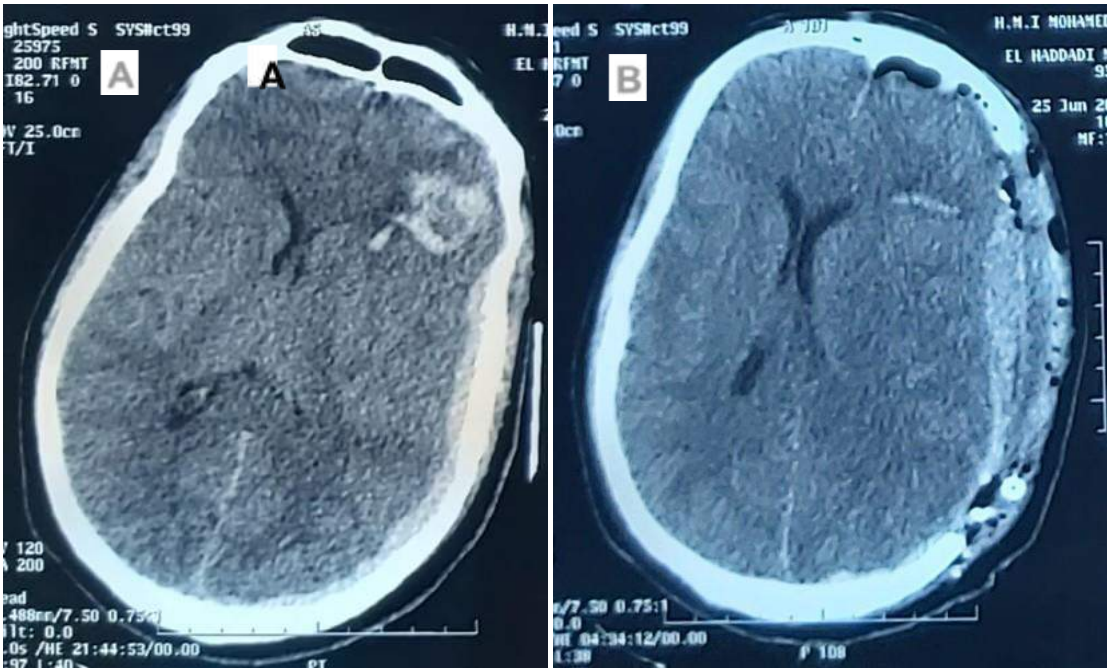

Figure 1: A/ Head CT shows a spontaneous hyperdense ASDH of 12mm thickness on the left hemisphere with 8mm midline shift. Diffuse brain edema. B/ Postoperative axial head CT-Scan after decom- pressive craniotomy and evacuation of the ASDH and also the ICH.

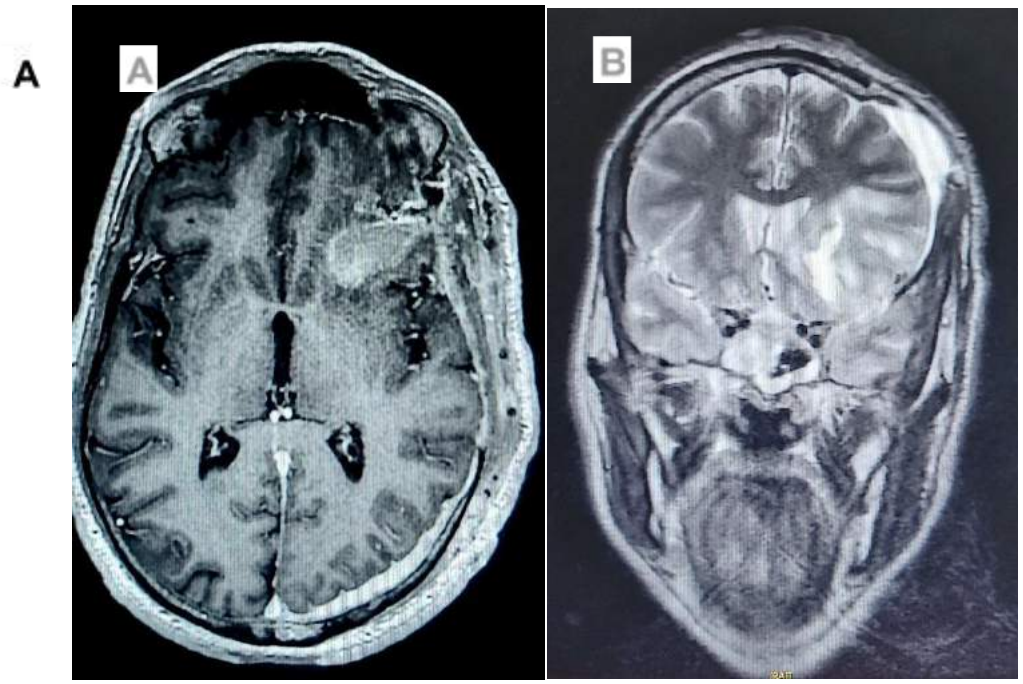

Figure 2: Head magnetic resonance imaging; A/ axial T1-Weighted, B/ coronal T2- Weighted with the left frontal ICH with the and the ASDH

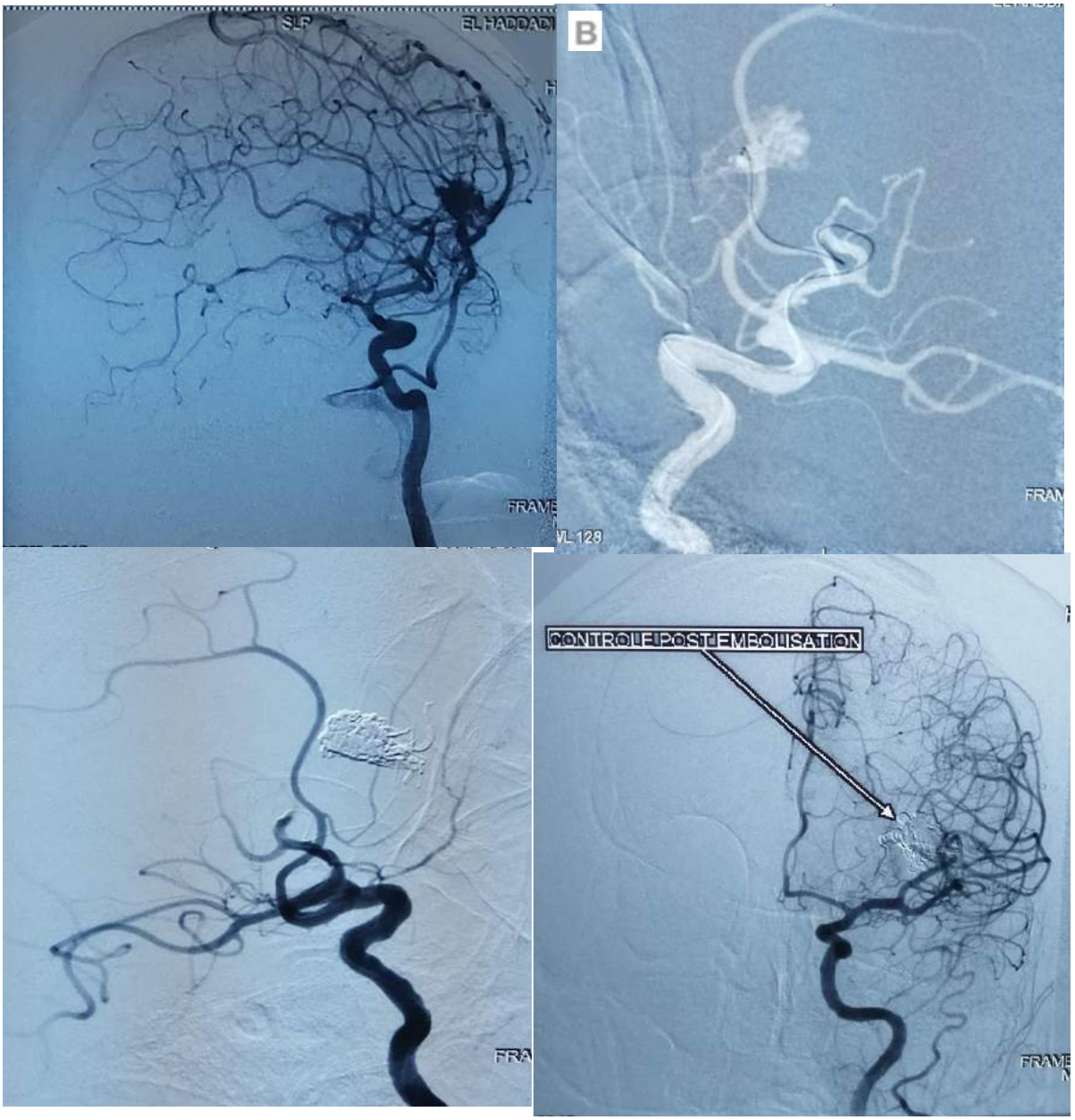

Figure 3: A/ Left internal carotid artery angiography, anteroposterior and lateral view revealing left fronto-insular arteriovenous malformation supplied from the insular branch of the left middle cerebral artery (circle). B/ Injection of embolization material (Onyx) C/ Exclusion of the nidus. D/ Angiographical control of the nidus after embolization