

CASE REPORT

Simultaneous transient global amnesia and right MCA stroke after Valsalva manoeuvre

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SUMMARY

A 61-year-old man suffered an episode of transient confusion and anterograde amnesia after a Valsalva-related manoeuvre. The MRI diffusion weighted imaging (DWI) sequences showed a left hippocampal and two right parietal lesions that were deemed as acute. The MR angiography disclosed a high-grade stenosis in the right middle cerebral artery as was described by a transcranial colour-coded ultrasound as well. Ultrasound investigation of the jugular veins showed a right jugular venous reflux after a Valsalva manoeuvre. The patient was diagnosed with transient global amnesia based on clinical grounds and the right parietal lesions were considered as silent strokes. The Valsalva manoeuvre could have played as a common trigger for both diseases.

BACKGROUND

This case highlights the importance of taking into account the clinical information to be able to understand the results of ancillary investigations.

CASE PRESENTATION

A 61-year-old man with a medical history of high blood pressure presented to hospital with sudden

onset acute confusion. His wife described that shortly after having ‘opened his bowels’ he repeatedly started to ask where they were and what they were doing there, but he was not able to remember the answers his wife gave to him and repeatedly asked the same questions. She immediately called the paramedics and as a stroke was suspected he was urgently referred to the hospital.

The initial neurological examination did not show any focal neurological deficit but an overt amnesic syndrome was present at that moment. He was constantly asking the same questions (‘where am I?’, ‘what are we doing here?’) and was not able to recall any information he was given after a short time interval, but there were not any cognitive deficits otherwise. The amnesic episode subsided completely after 4 hours, and the patient and his wife described that it was the first time that he had suffered an episode like that. In view of the clinical symptoms, the patient was diagnosed with transient global amnesia (TGA).

INVESTIGATIONS

The laboratory investigations excluded toxic, infectious or metabolic causes and an electroencephalogram was normal. The initial cranial CT was unremarkable but the brain MRI revealed small cortical lesions hyperintense on the DWI sequences. One of them was located in the left hippocampus and two in the right parietal region. The time-of-flight MR angiography (MRA) disclosed a high-grade stenosis in the right middle cerebral artery (MCA) (figure 1).

The duplex ultrasound investigation showed non-significant carotid atherosclerosis. The transcranial colour-coded sonography (TCCS) revealed an increased flow velocity that reached a peak systolic velocity (PSV) of 313 cm/s at a depth of 53 mm in the right M1-MCA. A mild poststenotic flow was detected in the right M2-MCA branches. In comparison with the left side, the right P2-/P3-PCA segments had an increased PSV without turbulence (75 cm/s vs 54 cm/s). Both A1-anterior cerebral artery and the left M1-MCA segment showed normal flow parameters (figure 2).

The investigation of the internal jugular veins (IJVs) showed a right-dominant IJV defined by a larger area and higher flow velocity. During a Valsalva manoeuvre (VM), valve competence was demonstrated on the left side. On the right side, a marked reflux that lasted up to 1.28 s confirmed an incompetent IJV valve (figure 3). The investigation

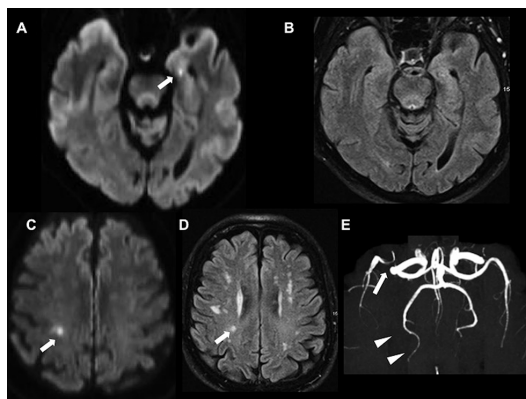


Figure 1 Brain MRI showed a left hippocampal diffusion-weighted hyperintense lesion (arrow, A) not evident on FLAIR sequence (B). The right parietal lesions were clearly visible on both diffusion-weighted and fluid attenuated inversion recovery (FLAIR) sequences (arrows, C and D). The MR angiography disclosed a high-grade stenosis in the right middle cerebral artery (arrow, E). The prominent right posterior cerebral artery signal (arrowheads, E) denotes leptomeningeal collateral flow activation.



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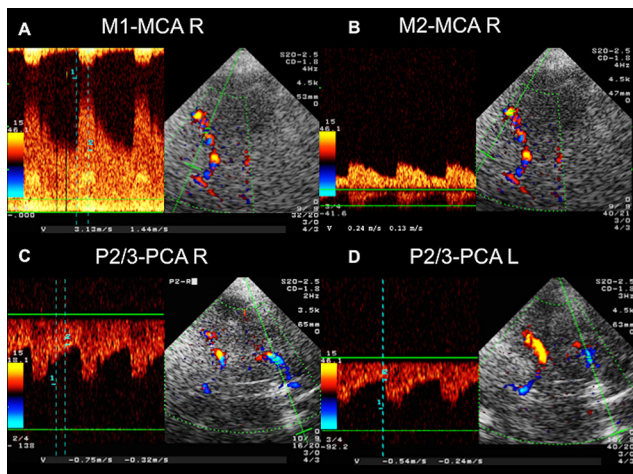


Figure 2 The transcranial colour-coded sonography disclosed high-flow velocities at a depth of 53 mm in the right MCA (A) with a poststenotic flow in M2 segments (B), corresponding to a high-grade stenosis. The right PCA (C) showed increased velocities compared with the contralateral PCA (D) which suggested collateral flow carried by leptomeningeal collateral. M1-MCA-R: M1 segment of the right MCA. M2-MCA-R: M2 segment of the MCA. P2/3-PCA-R: P2-P3 segments of the right PCA. P2/3-PCA-L: P2-P3 segments of the left PCA. MCA, middle cerebral artery; PCA, posterior cerebral artery.

of the intracranial veins showed normal flow signals in all examined vessels including the basal vein of Rosenthal (BVR), the deep middle cerebral vein, the vein of Galen (VG), and the straight sinus (StS). A mild flow velocity reduction was observed during a forced VM in the examined BVR.

Cardiological work-up including transoesophageal echocardiography and 24-hour telemetry showed neither cardiac nor aortal sources of emboli.

Considering the left-sided hippocampal lesion and the clinical presentation and evolution, a TGA was diagnosed whereas the right-sided ischaemic lesions and high-grade M1-MCA

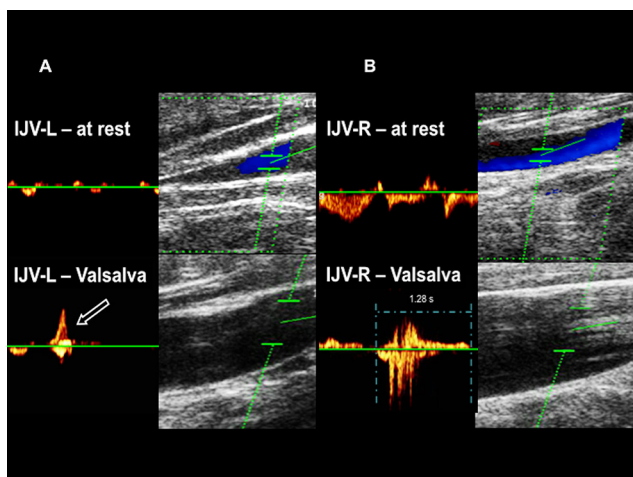


Figure 3 (A) Left IJV with undulating flow and low flow velocities at rest and physiological flow arrest during a Valsalva strain. Note the sharp and short retrograde flow resembling physiological valve closure (arrow). (B) Right IJV with undulating but mostly anterograde flow direction with higher flow velocities compared with the left IJV at rest. An abnormal flow reflux is seen during a Valsalva strain of 1.28 s. IJV, internal jugular vein.

stenosis were considered incidental findings. Aspirin and statins were added to his antihypertensive medication. After 3 years of follow-up, the patient remained clinically stable without any changes on the TCCS and MRA. The left-sided hippocampal lesion was not detectable in the FLAIR sequences at that time.

DISCUSSION

Herein we describe a 61-year-old patient who suffered a first episode of TGA after a Valsalva-related manoeuvre. The DWI-MRI revealed a hyperintense lesion in the Sommer sector of the left hippocampus and two small parietal acute ischaemic lesions likely to be embolic in the context of a high-grade MCA stenosis.

Hippocampal DWI-positive lesions have been previously described in TGA. These lesions are reversible and can be found in up to 84% of the cases between 24 and 74 hours after symptoms onset.^{1 2} Compared with lesions observed in patients with transient ischaemic attack (TIA), TGA DWI-positive lesions appear later, tend to be smaller and do not lead to permanent FLAIR hyperintensities.³

The pathogenesis of TGA is controversial and several mechanisms have been proposed, including epilepsy, arterial ischaemia and venous congestion. Although an amnesic syndrome can be caused by an ischaemic stroke, it usually appears as part of other neurological symptoms.^{4 5} Furthermore, patients with TGA usually have lower rates of vascular risk factors and future vascular events than patients with TIA, arguing against an ischaemic aetiology for TGA.⁶

Venous congestion has also been considered a potential pathological mechanism given the close temporal relationship observed in many cases between VM and episodes of TGA. VM leads to a transient increase of the intrathoracic pressure that can limit the venous reflux coming from the head and limbs.⁷ The resulting venous congestion might block the draining veins of the medial temporal lobe and especially the inferior ventricular vein (IVV), where the anterior hippocampus drains.⁸ The IVVs merge on both sides into the BVR. Both BVR drain into the singular VG and/or StS and then preferentially into the left transverse sinus (TS) and left IJV. Subsequently, venous reflux into the dominant IJV might affect both hippocampal areas.

A cessation or decrease of anterograde venous flow during repetitive VM can be assessed with ultrasonography and indicates competent jugular valves, whereas retrograde flow indicates internal jugular valve insufficiency (IJVVI). A cut-off value of 0.88 s has been defined to securely differentiate between transient physiological reflux (<0.88 s) caused by valve closure and real IJVVI (>0.88 s).⁹ IJVVI is present in approximately 30% of healthy subjects.¹⁰

A series of ultrasound studies found a significantly higher, mostly unilateral IJVVI prevalence in patients with TGA compared with controls.¹¹⁻¹⁶ The largest study, analysing 142 patients and 40 controls found an IJVVI in 80% of patients with TGA and in 25% of controls.¹² In selected patients with an antecedent VM, IJVVI was even seen in up to 100%.¹⁰

However, the side of affected IJV valve does not always seem to match the observed StS drainage pattern, as demonstrated in a combined ultrasound-MRI study. However, the number of analysed patients was too small to draw definite conclusions.¹⁶ Using MRA at rest, a higher proportion of retrograde venous flow—distal of the IJV valves—was reported in patients with TGA.¹⁷ However, this observation has not been confirmed by others.¹⁸ Other authors suggest that patients with TGA can have an increased prevalence of stenosis of the jugular veins and hypoplasia of the TS.¹⁹ Furthermore, ultrasound studies

did not show a venous reflux during VM at least in the BVR and StS.^{11 13}

Apart from IJVVI findings in TGA, significantly higher rates have also been observed in primary exertional headache,²⁰ in idiopathic intracranial hypertension,²¹ in patients with transient monocular blindness, especially if recurrent and of undetermined origin,²² and also in leukoaraiosis.²³

The case we are discussing herein is complex as it combines TGA with concomitant silent brain infarctions. Silent DWI-positive lesions can be observed in up to 5% of the cases in some groups of patients.^{24 25} In our case, the DWI-positive parietal lesions were interpreted as incidental and presumably caused by an artery-to-artery embolism coming from the high-grade right MCA stenosis.

However, apart from 'lesions just found by chance', it is possible that those parietal lesions could share a common origin with the episode of TGA and the left hippocampal lesion. In that sense, phase IV of VM is characterised by a sharp increase of the arterial blood pressure caused by both increased sympathetic tone and systemic vascular resistance. During this latest phase of the VM, an increase of the cerebral blood flow velocity is observed in the MCA when it is monitored with transcranial Doppler.²⁶ Accordingly, we could hypothesise that the increase of the arterial blood pressure and consequent stress over the surface of an intracranial stenosis could favour plaque rupture and subsequently thrombosis and embolism.²⁷

This clinical case points out the importance of clinical history and examination to make an accurate interpretation of the results we get from ancillary investigations. Furthermore, although merely hypothetical, our case also shows how two unrelated diseases can happen at the same time given a common element that is able to trigger different pathogenic mechanism.

Learning points

- ▶ The clinical information that we get from our patients is the cornerstone to understand the results of the ancillary investigations.
- ▶ A Valsalva manoeuvre can be the trigger for transient global amnesia (TGA) and acute stroke.
- ▶ Jugular vein insufficiency is more frequent in patients with TGA.
- ▶ In contrast to ischaemic lesions, TGA DWI-positive lesions do not lead to permanent lesions on FLAIR sequences.

Contributors JMV and AFM: acquisition, analysis and interpretation of data and critical review of the manuscript. CG: acquisition, analysis and interpretation of data. MGC: interpretation of data, writing and critical review of the manuscript.

Competing interests None declared.

Patient consent Obtained.

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REFERENCES

- 1 Sedlacek O, Hirsch JG, Grips E, *et al.* Detection of delayed focal MR changes in the lateral Hippocampus in transient global amnesia. *Neurology* 2004;62:2165–70.
- 2 Lee HY, Kim JH, Weon YC, *et al.* Diffusion-weighted imaging in transient global amnesia exposes the CA1 region of the Hippocampus. *Neuroradiology* 2007;49:481–7.
- 3 Toledo M, Pujadas F, Grivé E, *et al.* Lack of evidence for arterial ischemia in transient global amnesia. *Stroke* 2008;39:476–9.
- 4 Greer DM, Schaefer PW, Schwamm LH. Unilateral temporal lobe stroke causing ischemic transient global amnesia: role for diffusion-weighted imaging in the initial evaluation. *J Neuroimaging* 2001;11:317–9.
- 5 Ott BR, Saver JL. Unilateral amnesic stroke: six new cases and a review of the literature. *Stroke* 1993;24:1033–42.
- 6 Mangla A, Navi BB, Layton K, *et al.* Transient global amnesia and the risk of ischemic stroke. *Stroke* 2014;45:389–93.
- 7 Lewis SL. Aetiology of transient global amnesia. *Lancet* 1998;352:397–9.
- 8 Park YH, Jang JW, Yang Y, *et al.* Reflections of two parallel pathways between the Hippocampus and neocortex in transient global amnesia: a cross-sectional study using DWI and SPECT. *PLoS One* 2013;8:e67447.
- 9 Nedelmann M, Eicke BM, Dieterich M. Functional and morphological criteria of internal jugular valve insufficiency as assessed by ultrasound. *J Neuroimaging* 2005;15:70–5.
- 10 Nedelmann M, Eicke BM, Dieterich M. Increased incidence of jugular valve insufficiency in patients with transient global amnesia. *J Neurol* 2005;252:1482–6.
- 11 Baracchini C, Tonello S, Farina F, *et al.* Jugular veins in transient global amnesia: innocent bystanders. *Stroke* 2012;43:2289–92.
- 12 Cejas C, Cisneros LF, Lagos R, *et al.* Internal jugular vein valve incompetence is highly prevalent in transient global amnesia. *Stroke* 2010;41:67–71.
- 13 Lochner P, Nedelmann M, Kaps M, *et al.* Jugular valve incompetence in transient global amnesia. A problem revisited. *J Neuroimaging* 2014;24:479–83.
- 14 Maalikjy Akkawi N, Agosti C, Anzola GP, *et al.* Transient global amnesia: a clinical and sonographic study. *Eur Neurol* 2003;49:67–71.
- 15 Sander D, Winbeck K, Etgen T, *et al.* Disturbance of venous flow patterns in patients with transient global amnesia. *Lancet* 2000;356:1982–4.
- 16 Schreiber SJ, Doepp F, Klingebiel R, *et al.* Internal jugular vein valve incompetence and intracranial venous anatomy in transient global amnesia. *J Neurol Neurosurg Psychiatry* 2005;76:509–13.
- 17 Chung CP, Hsu HY, Chao AC, *et al.* Detection of intracranial venous reflux in patients of transient global amnesia. *Neurology* 2006;66:1873–7.
- 18 Kang Y, Kim E, Kim JH, *et al.* Time of flight MR angiography assessment casts doubt on the association between transient global amnesia and intracranial jugular venous reflux. *Eur Radiol* 2015;25:703–9.
- 19 Han K, Chao AC, Chang FC, *et al.* Obstruction of Venous Drainage Linked to Transient Global amnesia. *PLoS One* 2015;10:e0132893.
- 20 Doepp F, Valdueza JM, Schreiber SJ. Incompetence of internal jugular valve in patients with primary exertional headache: a risk factor? *Cephalalgia* 2008;28:182–5.
- 21 Nedelmann M, Kaps M, Mueller-Forell W. Venous obstruction and jugular valve insufficiency in idiopathic intracranial hypertension. *J Neurol* 2009;256:964–9.
- 22 Hsu HY, Chao AC, Chen YY, *et al.* Reflux of jugular and retrobulbar venous flow in transient monocular blindness. *Ann Neurol* 2008;63:247–53.
- 23 Chung CP, Wang PN, Wu YH, Yh W, *et al.* More severe white matter changes in the elderly with jugular venous reflux. *Ann Neurol* 2011;69:553–9.
- 24 Auriel E, Martinez-Ramirez S, Gurol ME, *et al.* Incidental diffusion-weighted imaging lesions in patients with cognitive decline. *Stroke* 2013;44:AWMP116.
- 25 Saini M, Ikram K, Hilal S, *et al.* Silent stroke: not listened to rather than silent. *Stroke* 2012;43:3102–4.
- 26 Tiecks FP, Lam AM, Matta BF, *et al.* Effects of the valsalva maneuver on cerebral circulation in healthy adults. A transcranial Doppler Study. *Stroke* 1995;26:1386–92.
- 27 O'Rourke M. Mechanical principles in arterial disease. *Hypertension* 1995;26:2–9.

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