

TEST YOURSELF

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A cyclist with a focal neurological episode

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A 48-year-old right-handed man presented to a district general hospital accompanied by his wife. Since childhood he had had migraine with visual aura. Over the last few years he had continued to experience his usual auras infrequently but these were now seldom followed by headache and readily managed with simple analgesia. He was in excellent health, although perhaps exercising less than he had previously. He was not taking any regular medication, did not smoke or drink alcohol excessively, and he had no family history of note.

On the day of presentation, he had been cycling to work when he developed his typical visual aura, but he continued cycling. After a few minutes he reached the security entrance. At that stage he noticed that his right arm was not quite right and he had difficulty swiping his security pass. He felt vaguely disorientated and was unable to greet the security officer. After parking his bicycle he climbed up the stairs to his office. On the way, he tried to greet a colleague but could not reply with anything beyond “yes” and “no” and so he feigned exhaustion. He sat

down in his office chair about 15–20 minutes after the onset of his symptoms and for the first time noticed that he had marked weakness of his right arm. When his boss entered he again had difficulty speaking, although he could understand perfectly well. He went to the medical centre where the staff called an ambulance. An hour later his symptoms had all but resolved and by the time he reached the hospital emergency department he was back to normal. The first year medical resident documented a normal neurological and general examination. He was transferred to the CT scanner where he again noted right arm weakness. The weakness lasted about 5 minutes and was followed by headache—typical of his usual migraine—which lasted 3–4 hours and was associated with nausea. Over the course of the next 12–18 hours his symptoms fluctuated without full resolution. During that evening he again developed a headache with nausea, lasting several hours. He did not have any positive sensory symptoms at any stage.

The next morning his general examination was normal with a blood pressure of 135/80 mmHg, pulse of 55/min regular. He had a

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mild expressive dysphasia with poor naming, and use of neologisms and paraphrasias, but he could provide a clear account of what had happened to him over the previous 24 hours. His reading was poor and he could not interpret a paragraph of the morning newspaper, and nor could he repeat a simple phrase. When asked to write a sentence he wrote: "Thenty tow the toobbdl dogay I fashed the foobball witl my ya sun." He had mild sensory inattention on the right, agraphaesthesia on the right, poor calculation but normal constructional abilities. His visual fields were normal, as was the rest of the neurological examination. Two hours later his language (written and spoken) difficulty had almost fully resolved. Overnight he again had a severe migrainous headache, but by the next morning he had fully recovered both from the headache and any language difficulty.

Question 1

Where is the lesion?

COMMENT

The presence of dysphasia in this right-handed man clearly suggests a lesion in the left dominant hemisphere. A detailed discourse on the evaluation of dysphasia is beyond the scope of this series, and anyhow the patient recovered before we could complete a detailed neuropsychological or even bedside assessment. Nonetheless, it is worth considering the features of his language disturbance the morning after his admission; fluent dysphasia with paraphrasias and difficulty with confrontational naming. Fluent dysphasias include Wernicke's, anomic (or amnesic), conduction and transcortical sensory dysphasia. His auditory comprehension was excellent which suggested that this was not likely a Wernicke's or a transcortical sensory dysphasia. While he did have anomia his repetition was impaired and his clinical presentation about 24 hours after the onset of his symptoms was in keeping with a conductive dysphasia. Typical features of a conduction dysphasia are: fluent spontaneous speech, frequent paraphrasias with awareness of errors

and attempts to correct errors (conduit d'approche), anomia, and good auditory and reading comprehension. His reading comprehension was not good but impaired comprehension has been described in a series of patients with conduction dysphasia.¹ Lesions typically involve the posterior and inferior temporal region (particularly the supramarginal gyrus and insula), although involvement of the temporo-parietal junction has also been described. His difficulty with calculation, agraphaesthesia and sensory inattention would all fit with a dominant parietal lobe lesion. However, at the onset of his symptoms he had non-fluent speech with features perhaps of a Broca's dysphasia, at least on the history. At the same time he had weakness of his right arm, suggesting that his left frontal lobe was involved initially. Was this the consequence of a more severe lesion that had resolved by the next morning, or the result of more than one lesion, or perhaps not a structural lesion at all? It is difficult to be sure based on the available information and without brain imaging, but the temporal pattern with fairly quick resolution (albeit with fluctuation) might argue against a single large lesion and in favour of a functional disturbance of the cortex, or perhaps more than one lesion.

Question 2

What do you think caused his symptoms?

COMMENT

He suffered a relatively sudden onset of a focal neurological deficit which clearly sounds vascular in nature. It is more difficult to decide what type of vascular lesion would explain his signs and symptoms. There was definite progression over time (possibly about 15–20 minutes in the initial attack) from visual aura to mild right arm weakness and expressive dysphasia to more severe arm weakness. Progression over minutes does occur in stroke or transient ischaemic attack (TIA) but is unusual. The rapid resolution of his initial symptoms over an hour or two followed by a recurrence of similar symptoms might imply a second embolic event from a

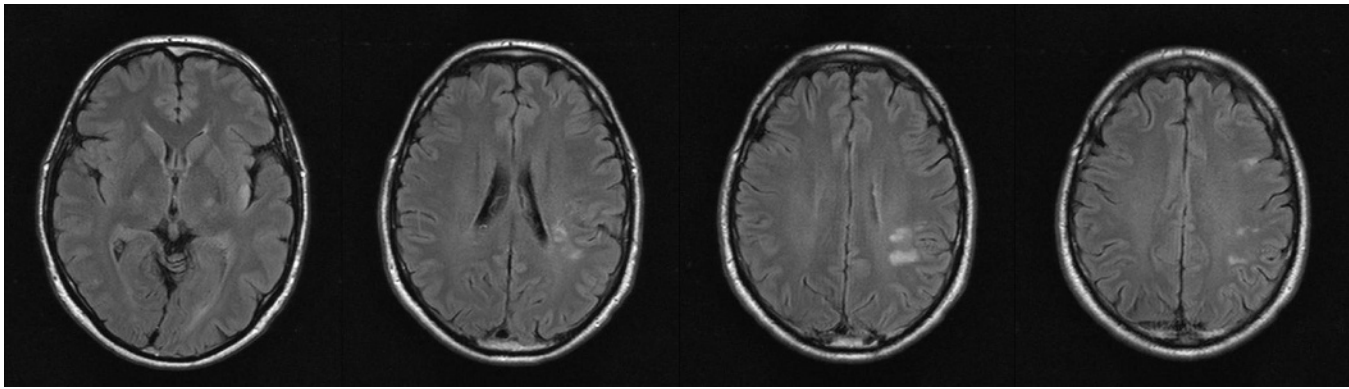


Figure
MRI scan with T2 FLAIR images showing high signal changes in the left frontal, parietal, posterior temporal and insular cortex.

proximal source (to explain the stereotyped nature of his symptoms) damaging the frontal cortex. However this would not explain his conduction dysphasia and parietal lobe signs, unless of course multiple emboli were involved.

On the other hand, he had a history of migraine with aura and clearly described experiencing his typical aura, albeit followed by motor symptoms and dysphasia which he had not had before. Moreover, following the later recurrence of his arm weakness he developed a migrainous headache with resolution of the weakness as the headache progressed. This might, therefore, all be explained by a migrainous aura and the later more persisting parietal lobe signs and conduction dysphasia by migrainous infarction.

The differential at this point included migrainous aura/infarction and ischaemic stroke due to multiple emboli.

The CT brain scan did not show any significant abnormality, but the MR brain scan 48 hours after the onset of his symptoms showed several areas of high signal on FLAIR, proton density and T2 weighted images. These involved the frontal, parietal, posterior temporal and insular cortex on the left (see fig). DWI images were not obtained. MR angiography was normal. He was started on aspirin and a statin.

COMMENT

The MRI features are in keeping with ischaemic lesions. While these are certainly compatible with small infarcts as a consequence of multiple emboli, migrainous infarction may also result in multiple ischaemic lesions.² Further investigation should be aimed at excluding the more serious alternative which would require specific treatment, in other words a proximal embolic source.

He was anxious to get home and was discharged without any residual signs or symptoms four days after admission, to come back for echocardiography with contrast. This showed mild concentric left ventricular hypertrophy, no significant valvular heart disease but an aneurysmal interatrial septum. Saline contrast revealed spontaneous right to left shunting without the need for a Valsalva manoeuvre, all consistent with a patent foramen ovale (PFO) with an interatrial septal aneurysm (ASA). Further investigations were all normal including: urea and electrolytes, liver function, thyroid function, protein electrophoresis, full blood count, erythrocyte sedimentation rate, autoimmune and vasculitis screen, syphilis serology, serum angiotensin converting enzyme inhibitor, cardiolipin antibodies, thrombotic screen, chest x ray, electrocardiogram and carotid ultrasound.

Question 3

How would you interpret his MRI findings?
And what would you do next?

Question 4

How would you manage this patient?

COMMENT

Taking the worst case scenario that the diagnosis was embolism from or through the heart, the management of PFO with or without an associated atrial septal aneurysm is highly controversial. PFO is found in about 30% of the general population and is associated with an ASA in about one fifth of cases—so it may be coincidental, even in a young patient with an ischaemic stroke and no other cause (as here).³ There appears to be an association between the risk of ischaemic stroke in younger (<55 years of age) people and PFO (by about three times), ASA (by about six times) and both together (by 16 times).⁴ One study of *recurrent* stroke (600 consecutive patients <55 years of age with recent cryptogenic ischaemic stroke) found a fourfold increase in risk in patients with both PFO and ASA after a mean of three years.⁵ However, the PICSS (Patent foramen ovale In Cryptogenic Stroke Study) of 301 patients with recent ischaemic stroke who had been randomised in the Warfarin–Aspirin Recurrent Stroke Study did not find any increased risk of recurrent stroke or death in patients with PFO, whether or not they had an associated ASA. Warfarin did not reduce recurrent stroke significantly compared to aspirin in patients with PFO.⁶

The role of percutaneous (or even open) closure of PFO with or without an ASA has not yet been answered by randomised controlled trials (RCTs). Given the uncertainty of the relevance of these anomalies in a case like this, the small but definite risk associated with the procedure (1.5% major complication, 7.9% minor complications), and the lack of any RCTs, we would argue that patients who present with stroke and are found to have a PFO or ASA or both should be encouraged to consider participation in an ongoing trial (<http://www.clinicaltrials.gov/show/NCT00166257>).^{7, 8}

The added difficulty in this case is that neither his clinical presentation nor imaging convincingly distinguished between migrainous infarction and ischaemic stroke due to embolism. Bousser has argued that in the past patients have not been investigated in

sufficient detail to adequately exclude an alternative cause before the diagnosis of migrainous infarction was made.² She also pointed out that cerebral ischaemia may precipitate migraine. This could have been the case in this patient although the purists may argue that simply finding the PFO and ASA does not mean that this was indeed the cause of stroke in an otherwise fit young man, particularly as he had no clinical evidence of any deep venous thrombosis (DVT), no reason to develop a DVT, and no clotting abnormality.⁸ To complicate matters further there is debate about the role of PFO closure in migraine with aura. However, the only randomised controlled trial (MIST; results available at <http://www.clinicaltrialresults.org>) did not show convincing evidence to support this approach.

We have discussed the management options with the patient and he is mulling them over. He has remained on aspirin in the meantime.

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