Blurring of vision with malaise

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Abstract

We present the case of a 56-year-old female brought to the Emergency Department via routine ambulance transport with complaints of blurred vision and malaise. She was screened by ambulance crew using the facial arm speech time (FAST) tool and a basic top-to-toe assessment as per current routine. The examining practitioner performed a thorough assessment of the patient, revisiting the initial examination findings, and establishing new clinical features of visual field deficit and pan-systolic murmur. The likely diagnosis of septic emboli or stroke with infective endocarditis was identified through the power of rigorous history taking and examination. These were then supported by investigation with blood tests and imaging. This prompted discussion with a tertiary centre and subsequent transfer for further investigation and management. The patient's journey shows that there may indeed be a role for a more comprehensive (but not exhaustive) initial screening from ambulance services in order to help appropriately stream specific patients to hospital in a timelier manner (to meet the thrombolysis window). This case supports the addition of V (visual fields) to the FAST screening tool.

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Introduction

A 56-year-old female, discharged two weeks prior, following treatment for community-acquired pneumonia (CAP) and a *staphylococcus aureus bacteraemia* referred this time via the Emergency Department through routine ambulance transport, for generalised malaise, weakness and blurry vision. On clinical examination during the current visit she was found to have right homonymous hemianopia, a low-grade fever and a new pansystolic murmur radiating to left axilla Broad spectrum antibiotics were started and a CT head scan confirmed left occipital lobe infarct. Her case was discussed with a tertiary centre and an MRI head scan confirmed the CT head scan findings. A transthoracic echocardiogram (TTE) confirmed a mitral valve vegetation. Dental review identified an abscess requiring extraction of the lower right incisor tooth (LR1). She responded to the antibiotics. Repeat TTE was normal. She is currently back at work, with advice not to drive, until further review by the Driver and Vehicle Licensing Agency (DVLA).

Case presentation

A 56-year-old female was discharged two weeks prior when she was admitted to the hospital with fever and chest tightness. In that admission, a chest X-ray showed consolidation in upper lobes and blood culture was positive for *staphylococcus aureus*. A transthoracic echocardiogram (TTE) was normal. She was treated for *staphylococcus aureus bacteraemia* with flucloxacillin and gentamicin based on sensitivity patterns. She improved and two subsequent blood cultures and an echocardiogram (ECHO) were negative. She was discharged with a course of antibiotics to complete in the community.

During this recent visit she was brought in to the hospital via ambulance and following was referred via the Emergency Department for generalised malaise, weakness, low grade fever and blurry vision to the acute on call medical team for further assessment.

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Figure 1 CT head scan. Features of focal infarct in left occipital lobe, no abnormal enhancement

Her past history included hypertrophic obstructive cardiomyopathy (HOCM) for which she takes bisoprolol, clopidogrel, and atorvastatin and is under active surveillance, and haemochromatosis C282Y homozygous, for which she has regular venesection.

On clinical examination she had a low-grade fever, right homonymous hemianopia and a new pansystolic murmur radiating to the left axilla. This new murmur was distinguishable from a known high-pitched, crescendo-decrescendo, mid-systolic murmur heard best at the left lower sternal border.

A CT head scan confirmed left occipital lobe infarct (Figure 1). An ECHO confirmed new vegetation on the mitral valve, with (as before) reduced LV cavity size due to asymmetric septal hypertrophy; a dynamic left ventricular outflow tract (LVOT) gradient 64 mmHg at rest, increasing to 89 mmHg post valsalva (LVOT obstruction in HOCM is defined by >30mmHg change); and ejection fraction 76% (Figure 2). An MRI confirmed the CT head scan findings of left occipital lobe infarct (Figure 3). Maxillofacial review revealed a dental abscess of the lower right incisor (LR1) (Figure 4).

She was started on broad spectrum antibiotics (meropenem and vancomycin) and aspirin. Her case was discussed with a tertiary centre and the local stroke consultant, and an MRI head scan and TTE was requested. It was decided not to thrombolyse her at the time as six hours had passed by the time the CT head scan results were reported. She was treated locally as infective endocarditis (IE) on the basis of fever, new murmur, raised inflammatory markers, imaging suggestive of both septic emboli and anterior mitral leaflet vegetation. Day 10 blood cultures grew staphylococcus aureus from two bottles with cultural sensitivity to flucloxacillin, gentamicin and rifampacin.

Figure 2 Transthoracic echocardiography. Vegetation on the anterior mitral leaflet

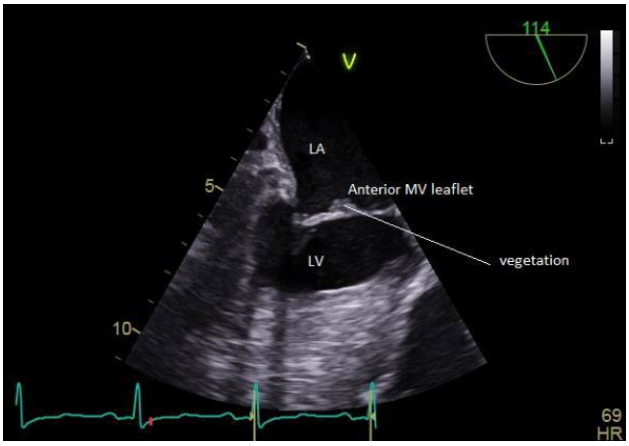

Figure 3 CT MRI head scan, showing recent/subacute left parieto-occipital posterior circulation infarct as evidenced by hyperattenuation in T2 weighted axial section – restricted diffusion and localised wedge shaped parenchymal swelling in the parafalcine left occipital lobe

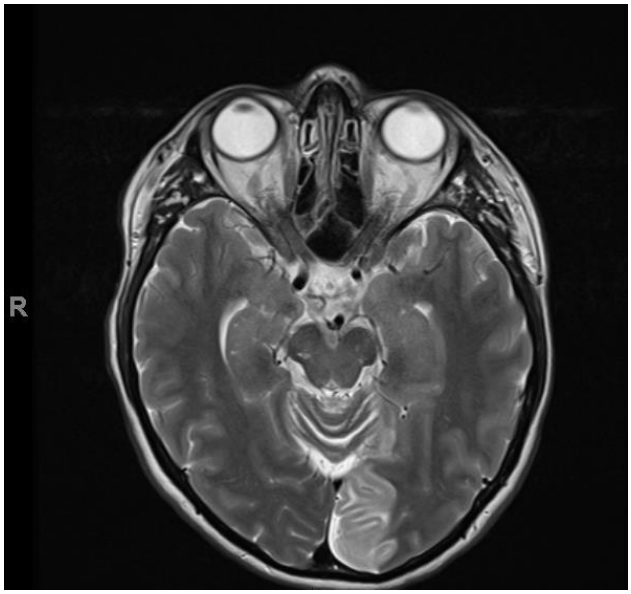

Figure 4 Orthopantomogram. Abscess in right central lower incisor. Abscess seen. It was tender on clinical examination and later drained

After initial treatment, the patient was transferred to a tertiary centre where she was assessed for mitral valve replacement. This was not clinically indicated due to significant improvement as a result of timely antibiotic therapy. She was transferred back to our hospital for LR1 extraction and completion of a four-week course of flucloxacillin and gentamycin.

Repeat TTE did not show any vegetations on the mitral valve. Repeat blood cultures were negative, and the patient became asymptomatic apart from the visual defect. A urine test did not show any blood either at the time of presentation or at discharge and she did not demonstrate any peripheral signs of IE i.e. splinter haemorrhages, finger clubbing, Janeway lesions on the palm nor Osler nodes. Inflammatory markers in the blood were raised at the time of presentation but normalised after antibiotic treatment.

In spite of successful treatment of the infection, there was persistence of right homonymous hemianopia. The patient was, however, able to go back to work following rehabilitation involving scanning exercises which improved visual cueing. The patient was advised not to drive until further assessment by the Driver and Vehicle Licensing Agency (DVLA).

Discussion

William Osler first presented the manifestations of IE in the English literature in 1851, although Lazare Rivière in 1674 described IE in a deceased patient as 'cluster of hazelnuts in the opening of the aorta'.[1]

Infective endocarditis, previously thought to be rare, seems to be occurring more frequently with increasing morbidity and mortality, with greater incidence in males and the elderly.[2] Staphylococcus aureus has become the primary pathogen in IE.[3,4] The incidence was 3.4 cases per 100,000 person-years in Herefordshire and surroundings.[5]

The incidence of staphylococcal aureus-related dental abscess has been reported to be between 0.7% to 15%.[6-9]

Duke criteria are used to establish the diagnosis of IE.[10] It relies heavily on echocardiogram findings. There has been research on indications of requesting echocardiogram based on signs and symptoms both in patients with a history of prior intravenous drug use[11,12] and without.[13,14]

Septic embolus has been commonly reported to be associated with bacterial endocarditis, septic thrombophlebitis, periodontal, and central venous catheter infections.[15]

In a study of 198 critically ill patients with IE, 55% (n=108) experienced at least one neurological complication, of which 40% had ischaemic stroke at presentation.[16] Infrequent findings included cerebral haemorrhage, meningitis, brain abscess, and mycotic aneurysm.

This case highlights the need to consider a diagnosis of IE in any patient presenting with focal neurological deficit and a new or changing heart murmur. Patients with a pre-existing cardiac murmur which has changed in quality, position, or timing should prompt consideration of a new condition. Prior history of bacteraemia with staphylococcus may be the only clue, and a detailed history is of paramount importance. Additionally, clinicians should consider the differentials for embolic disease in any patient with ischaemic stroke.

Independent of IE, any patient with visual field deficit, if diagnosed promptly at the community level, would prompt blue-light transfer to the hospital to be assessed and managed within the thrombolysis window. Rowe et al (2009) conducted a multicentre observational study of 323 patients across 14 acute trust hospitals, finding that only 8% of stroke patients had normal vision, while 92% of them suffered some form of visual impairment, of which 49% had visual field impairment.[17]

In 2003 Harbison et al. recommended use of FAST (Facial – Arm – Speech – Time) assessment in the community setting for quick transfer of stroke patients to the hospitals.[18] In 2016 this was adopted in National Institute for Health and Care Excellence (NICE) guidance as outlined by the Royal College of Physicians Stroke Working Party and remains in place to date.[19]

We recommend the addition of 'V' – visual field changes, as outlined by Aroor et al. in 2013.[20] In this case the patient was assessed as FAST negative and hence not blue lighted to hospital. The thrombolysis window elapsed and a potential treatment was not delivered. The patient now has residual visual field defect and reduced quality of life which may have been prevented if she was assessed using a more comprehensive and specific assessment tool: FAST-V.

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