Neurology Asia 2020; 25(2) : 189 – 192

CASE REPORTS

Acute ischemic stroke in systemic lupus erythematosus: Challenges for thrombolytic therapy

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Abstract

Multiple mechanisms can cause ischemic stroke in s*ystemic lupus erythematosus* (SLE) patients. When a known case of SLE present with an acute ischemic stroke, the decision for offering thrombolytic therapy can be challenging since some of the mechanisms which may have caused the stroke can be a contraindication or a relative contraindication for thrombolytic therapy. Here we present a case where successful intravenous thrombolytic therapy was offered in an SLE patient where a careful history, clinical examination and imaging features were helpful in taking a judicious decision. This is only the third case in the English literature of a known SLE patient being offered thrombolytic therapy.

Keywords: SLE, stroke, thrombolysis

INTRODUCTION

Systemic lupus erythematosus (SLE) can cause ischemic strokes through multiple pathophysiological mechanisms. When SLE patients present with acute ischemic stroke within the golden hours for thrombolysis therapy, one need to rule out several etiologies of stroke in the setting of SLE, such as infective endocarditis, vasculitis, arterial dissection and others, which are contraindications or relative contraindications for thrombolysis. The history, clinical examination and imaging features will be helpful in taking a correct decision. We report here a young woman who was a known case of SLE presenting with acute ischemic stroke who was treated with thrombolysis therapy.

CASE REPORT

A 49-year-old women was brought to the emergency department of our hospital with a history of progressive right sided body weakness and inability to speak. Her symptoms started one hour prior to arrival to our emergency department. She was a known case of SLE since the last 18 years currently in remission and was on maintenance prednisolone. She did not have any atherosclerotic risk factors like diabetes or hypertension. Her blood pressure at the time of presentation was 110/70 mm Hg, pulse rate was 88/minute and regular, and she was afebrile at 37.2C. Cardiac examination was normal, S1, S2 was heard with no added sounds and no cardiac murmur. She was conscious, but globally aphasic. She had right sided hemiplegia with 2/5 power in upper limb and 1/5 in lower limb. Her NIHSS score was 15.

The CT angiogram showed total occlusion of the distal M1 segment of the left middle cerebral artery (MCA) (Figure 1 A, B). There was faint hypodensity involving the cortical and subcortical areas of the left temporooccipital lobes, left insular sub cortical and left external capsule with early signs of sulcal effacement on the plain CT brain (Figure 1 C). The CT perfusion (Figure 1 D), Cerebral blood volume (CBV) map (Figure 1 E) and Time to peak (TTP) maps (Figure 1F) showed perfusion deficit in left temporoparietal lobes denoting an infarct core with a significantly large penumbra Involving the left frontal, parietal, temporal and occipital lobes

She was treated with IV thrombolysis with recombinant tissue plasminogen activator (rtPA) 45mg (4.5mg as bolus and 40.5mg as continuous infusion over one hour). Repeat imaging, MRI brain showed complete recanalization on MRA (Figure 2 A) with most of the penumbra salvaged as seen on the FLAIR, T2 and T1 Sequences (Figure 2 B, C, D). At discharge patient had improved to NIHSS score of 6. Power on the right

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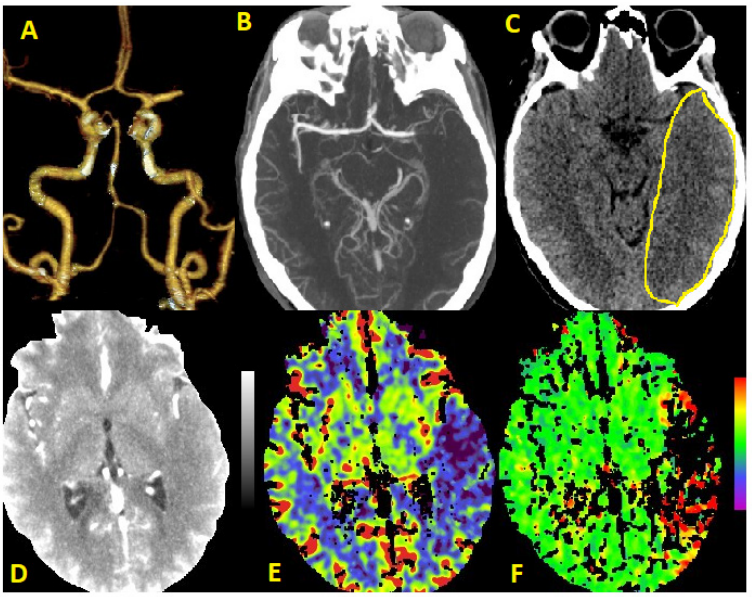

Figure 1. Imaging performed at baseline. (A) CT angiogram of cerebral arteries with reconstruction and (B) CT angiogram showing total occlusion of the distal M1 segment of the left MCA. (C) CT scan of brain showing faint hypodensity involving the cortical and subcortical areas of the left temporooccipital lobes, left insular, sub cortical and left external capsule with early signs of sulcal effacement. (D) CT perfusion (E) Cerebral blood volume (CBV) map, (F) Time to peak (TTP) maps showing perfusion deficit in left temporoparietal lobes, denoting an infarct core with a significantly large penumbra involving the left frontoparietotempro and occipital lobes.

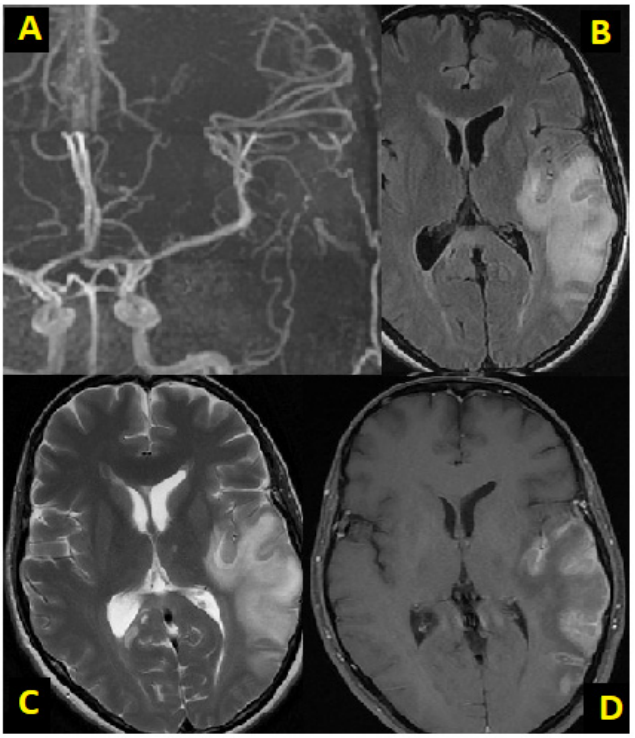

Figure 2. Follow up imaging. (A) MR angiogram showing complete recanalization of the left MCA. (B) FLAIR, (C) T2W and (D) T1W sequences showing residual infarct with most of the penumbra salvaged.

side was grade 4+/5 and she was able to ambulate independently, comprehend and obey commands but still not able to verbalize completely.

There were no markers of disease activity; erythrocyte sedimentation rate (ESR) and c reactive protein (CRP) were normal. Anticardiolipin IgG and beta 2 glycoprotein levels were within the normal range.

DISCUSSION

Stroke and cerebrovascular diseases contribute to 10–15% of all deaths in SLE patients.[1] SLE can affect both arterial and venous vascular beds and vessels of all calibre through both multiple inflammatory and noninflammatory mechanisms as follow: (1) Premature atherosclerosis: SLE can induce premature atherosclerosis through multiple mechanisms.[2] The autoantibodies produced in SLE patients[3] and also autoantibodies produced against lipid components like HDL, Apo-A1 and lipoprotein lipase (LPL)[4]; can bind on to molecules expressed on the surface of endothelial cells, this can impair endothelial vasodilatation and repair; and also release proinflammatory cytokines resulting in increased oxidative stress on the endothelial wall. Some drugs used in SLE like steroids are not only atherogenic but can also promote and aggravate diabetes and hypertension. (2) Secondary antiphospholipid syndrome (APS): can occur in 25–40% of SLE patients and can be the cause for both ischemic and venous strokes.[5] (3) Vasculitis: In SLE, immune complex deposition can occur on the vascular walls and subsequent action of antibodies against them can cause endothelial damage resulting in secondary vasculitis.[6] Apart from this drug-induced or infective vasculitis can also occur in SLE patients. Vasculitis can be seen in up to 10 % of SLE patient and can contribute to strokes.[6] (4) Arterial dissection: Dissection are more common and often can be the initial manifestation in SLE.[7] (5) Cerebral small vessel disease with lacunar infarcts, white matter hyperintensities, cerebral microbleeds, and enlarged perivascular spaces, is more common in SLE and contributes to the increased stroke burden.[8] (6) Endocarditis: In patients with SLE, the prevalence of valvular involvement can ranges from 18% to 61%[9]; either infective endocarditis or Libman-Sacks endocarditis can contribute to strokes.

When a patient with SLE presents with an acute ischemic stroke within the window period for thrombolysis, the decision for offering thrombolysis can be difficult and challenging. Some of the pathophysiological mechanism which may have cause the stroke can be contraindications for offering thrombolytic therapy and others can have high risk for haemorrhagic conversion of the infarct. Thrombolysis for acute ischemic stroke in infective endocarditis can have serious hemorrhagic complication and is not recommended.[10] If the underlying cause is a SLE induced vasculitis, again the risk for hemorrhagic conversion is high[11] Similarly in strokes due to dissections especially with intracranial extension, the role of thrombolysis is controversial and can only be based on a case to case basis.[12]

Apart from few case reports, there are no clear guidelines for treatment of patients with acute ischemic stroke with SLE. Chen and Xu[13] reported a case of IV thrombolysis in a SLE patient with occlusion of the right proximal MCA. Loharia *et al*.[14] reported a case of MCA occlusion with favourable outcome following IV thrombolysis where a diagnosis of SLE with secondary antiphospholipid syndrome was made subsequently. Majdak *et al*.[15] reported another case of thrombolysis of acute ischemic stroke in a SLE patient.

The decision of IV thrombolysis was clear in our case due to the absence of absolute contraindication. This is only the third case in the English literature and the first from Oman where successful IV thrombolysis was performed in a known case of SLE presenting with acute ischemic stroke

In conclusion, multiple pathophysiological mechanisms can result in ischemic stroke in SLE patients, some with contraindications for thrombolysis. A careful history, clinical examination and reviewing the imaging features will help to take a judicious decision before offering thrombolysis.

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