

YOUNG STROKE SECONDARY TO RHEUMATOID VASCULITIS



NF Abd Manaf¹, H Hashim¹, H Baharuddin¹ ¹Hospital Al-Sultan Abdullah, UiTM

INTRODUCTION

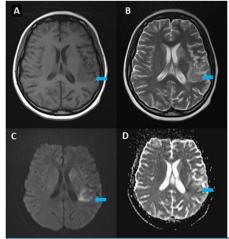
The pathogenesis of stroke in rheumatoid arthritis (RA) is due to chronic inflammatory process leading to either accelerated atherosclerosis or vasculitis, known as rheumatoid vasculitis (RV)⁽¹⁾ RV is an uncommon complication of RA, occurring in patients with an average disease duration of approximately 10 years⁽²⁾.

CASE REPORT

A 38-year-old woman with a 15-year history of seropositive RA, was referred to our clinic in April 2022. She selfpurchased prednisolone 5 to 10 mg daily since defaulted follow-up with another rheumatology centre in 2011. Methotrexate (MTX) was never started because she had been trying to conceive. She had a stroke two months prior to her presentation to our clinic. Neurological examination in the clinic revealed residual facial asymmetry and right-sided hemiparesis, with muscle power of 3/5. There was bilateral symmetrical polyarthropathy of the fingers and toes, and reduced range of motion of the wrists and elbows.

Rheumatoid factor and anti-cyclic citrullinated peptide were positive. Other immunological tests were conducted as part of young stroke work-up which revealed positive anti-nuclear antibodies and anti-Ro antibodies, and negative anti-dsDNA, anti-cardiolipin, p-ANCA and c-ANCA antibodies were negative. Echocardiogram and Holter were normal. MRI brain with MRA showed subacute left middle cerebral artery (MCA) infarct (Fig 1A-E). Total occlusion of the left ICA with presence of collaterals supplying the left MCA territory was revealed on the diagnostic cerebral angiogram, hence no endovascular intervention was made.

Double anti-platelet agents, prednisolone 40 mg (0.6 mg/kg), as well as sulphasalazine and hydroxychloroquine were started. Her RA became active when the prednisolone dose was reduced to 7.5 mg. She was counselled for MTX and she agreed. Six months later, anti-TNF inhibitor was started. Prednisolone was converted to hydrocortisone. To date, there has been no recurrence of either stroke or active RA.



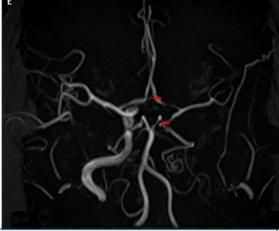


Fig 1: Transverse MRI brain images revealed a subacute infarct area at the left MCA territory (blue arrows) on T1 weighted image (A), hyperintense signal on T2 weighted (B) and DWI (C), and hypointense on ADC map (D). There was a long segment thrombosis in the left inferior cerebral artery (ICA) and enhancing wall suggestive of vasculitis. Sagittal MRA of the Circle of Willis (E) showed no flow in the left extracranial and intracranial ICA. The left MCA and anterior cerebral artery (ACA) (red arrows) are supplied by the anterior and posterior communicating arteries through the right ICA.

CONCLUSION

This unfortunate young woman with longstanding poorly-controlled RA developed stroke which we postulated was due to RV. In the era of advanced RA treatment, RA complications should not be allowed to happen. Patient education is paramount to enhance their commitment to RA treatment.

ACKNOWLEDGEMENT

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