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Primary antiphospholipid syndrome in a male with myocardial infarction with non-obstructive coronary arteries and a history of stroke

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A 55-year-old man with obesity, hypertension, hyperlipidemia and a history of right-sided ischemic stroke at the age of 45 was referred to a cardiology outpatient clinic after hospitalization for non-ST-elevation myocardial infarction with non-obstructive coronary arteries (Figure 1A; Supplementary material, *Video S1–S6*). Non-ST-elevation myocardial infarction with non-obstructive coronary arteries was diagnosed based on typical symptoms and dynamics of high-sensitive cardiac troponin T levels: 4.62 ng/l on admission and 114.80 ng/l after 6 hours, respectively (normal: 0–14 ng/l). No ischemic changes were observed on electrocardiogram. Other causes of increased serum high-sensitive cardiac troponin T were

excluded. Transthoracic echocardiography revealed normal ventricular function, a left ventricular ejection fraction of 55%, without significant valve defects.

Three months later, at a follow-up outpatient clinic visit, he was completely free of cardiovascular symptoms. The electrocardiogram showed pathologic Q waves in lateral leads, and the transthoracic echocardiography revealed no abnormalities. Cardiac magnetic resonance imaging showed no pathology. No stenosis was found in carotid duplex ultrasound.

Routine blood test results were normal, except for mildly reduced red blood cell parameters and elevated levels of low-density lipoprotein cholesterol (2.90 mmol/l) and triglycerides (2.02 mmol/l). Inherited thrombophilia was excluded.

Considering the suspected antiphospholipid syndrome (APS), the plasma levels of antiphospholipid antibodies (aPL) were measured. The lupus anticoagulant in the dRVVT and APTT assays was absent. The anticardiolipin antibodies (aCL) IgM were elevated (32.5 MPL (normal, 0-17.0 MPL)), while aCL IgG as well as anti-β2-glycoprotein I antibodies (aβ2GPI) IgM and IgG were normal. A weakly positive titer of antinuclear antibodies (ANA1 1:320) was also found. A single-positive APS was diagnosed.

According to current recommendations [1–3], aPL levels were reassessed after ≥12 weeks. Elevated aCL IgM were found to persist both after 8 months (20.0 MPL) and 20 months (39.1 MPL) (Figure 1B). Despite the still weakly positive titers of antinuclear antibodies (ANA3 1:320), elevated levels of anti-dsDNA, anti-nucleosome and anti-histone antibodies were not detected. There was also no clinical evidence of systemic connective tissue disease or another secondary cause of APS at 2-year follow-up.

Magnetic resonance imaging of the brain revealed an extensive area of malacic lesions in the right temporal lobe. Moreover, in the cerebral hemispheres, there were single small areas of raised signal in the sequence with a long time of repetition, consistent with nonspecific demyelinating lesions, primarily ischemic (Figure 1C).

In chronic pharmacotherapy, aspirin 75 mg/d, metoprolol succinate 25 mg/d, valsartan 80 mg b.i.d., and rosuvastatin 10 mg/d were used.

APS is characterized by venous, arterial or microvascular thrombosis and/or an adverse pregnancy outcome in the presence of persistent laboratory evidence of aPL [1–3]. As an acquired thrombophilia, APS can be diagnosed at any age, but is 5 times less common in men [1–3]. AMI is a very rare (2.8%) manifestation of APS [4, 5]. Despite indications to use warfarin in APS with arterial thromboembolism, our patient was treated with aspirin given a relatively low aCL levels with close ambulatory surveillance and follow-up outpatient visits

every 6 months. It is also important to search for other cardiovascular risk factors and their appropriate treatment, which can significantly improve the prognosis of APS patients.

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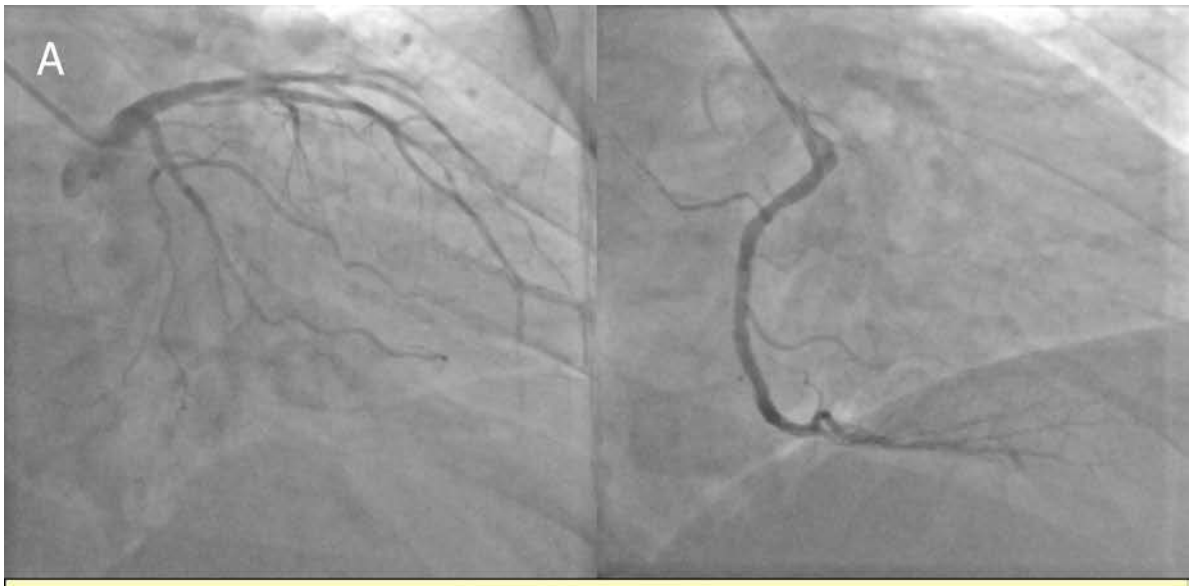
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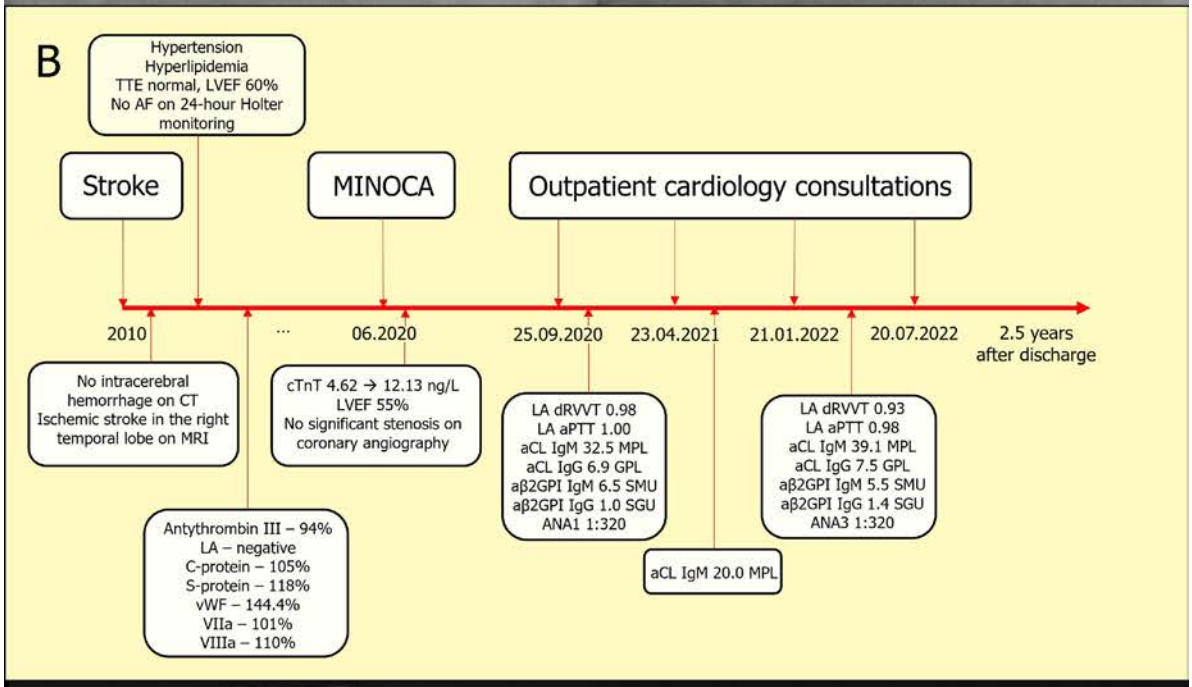
B
Hypertension
Hyperlipidemia
TTE normal, LVEF 60%
No AF on 24-hour Holter monitoring
Stroke
MINOCA
Outpatient cardiology consultations
2010
...
06.2020
25.09.2020
23.04.2021
21.01.2022
20.07.2022
2.5 years after discharge
No intracerebral hemorrhage on CT
Ischemic stroke in the right temporal lobe on MRI
cTnT 4.62 → 12.13 ng/L
LVEF 55%
No significant stenosis on coronary angiography
LA dRVVT 0.98
LA aPTT 1.00
aCL IgM 32.5 MPL
aCL IgG 6.9 GPL
aβ2GPI IgM 6.5 SMU
aβ2GPI IgG 1.0 SGU
ANA1 1:320
LA dRVVT 0.93
LA aPTT 0.98
aCL IgM 39.1 MPL
aCL IgG 7.5 GPL
aβ2GPI IgM 5.5 SMU
aβ2GPI IgG 1.4 SGU
ANA3 1:320
Antythrombin III – 94%
LA – negative
C-protein – 105%
S-protein – 118%
vWF – 144.4%
VIIa – 101%
VIIIa – 110%
aCL IgM 20.0 MPL

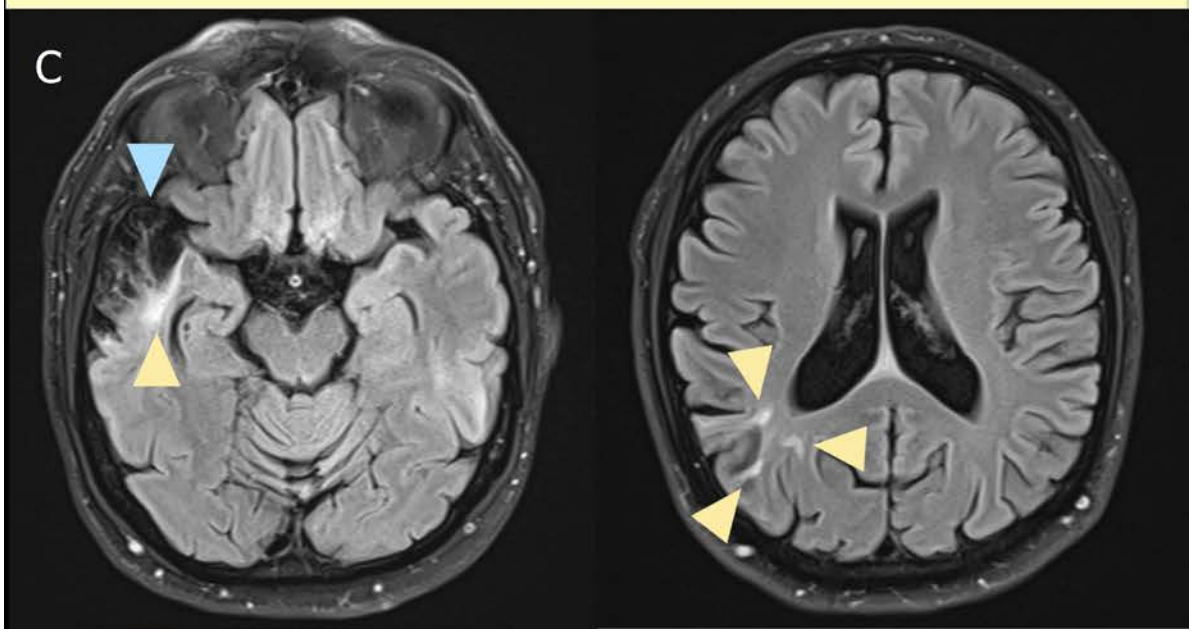
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Figure 1. Coronary angiography showing non-obstructed epicardial coronary arteries (**A**). The timeline of subsequent diagnostic and therapeutic stages (**B**). Magnetic resonance imaging brain FLAIR sequence showing an extensive area of malacic lesions in the right temporal lobe (blue arrow). Furthermore, there were single small areas of raised signal in the right cerebral hemisphere, consistent with nonspecific demyelinating lesions, primarily ischemic (yellow arrows) (**C**)