Testing for thrombophilia in cryptogenic stroke in the young: is it useful?

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Abstract

Point of view: No

The epidemiological evidence linking protrombotic states to arterial stroke is much weaker than that with venous stroke. Despite that evidence, screening for acquired and genetic thrombophilia is performed in young stroke patients in many stroke centers. This means searching for protein C and S and antithrombin deficiencies, factor V Leiden and prothrombin G20210A mutations and homocysteine plasma levels and for lupus anticoagulant and autoantibodies ( anticardiolipin and anti-beta2 glycoprotein) linked to the antiphospholipid syndrome. This screening must also take in consideration the effect of acute stroke phase inflammatory reaction, the influence of anticoagulants, the need for repeated testing and in some instances the need for additional genetic or familiar studies. All these evaluations take time and lead to incremental costs in the standard work up package of “young stroke”. With the exception of hyper-homocysteine plasma and antiphospholipid syndrome all the other prothrombotic condition are very rare. Therefore systematic screening for thrombophilia in young stroke victims has a low yield. Basing screening on clinical hints such as recurrent stroke, strong family history, combination of venous and arterial thromboembolic events or on clinical features suggesting antiphospholipid syndrome, such as recurrent thrombotic events or unfavorable pregnancy outcomes, in particular miscarriage, increases the efficiency of the screening. The therapeutic consequence of editing a prothrombotic state is in general lifetime anticoagulation. However the evidence supporting long term anticoagulation after stroke in patients with a prothrombotic condition is weak and it is unknown if long term anticoagulation improves the outcome and Quality of Life of young stroke patients. Therefore systematic testing for thrombophilia is unlikely to improve the outcome and to be cost-effective in young stroke patients.