

should in general be confirmed by a second measurement obtained under ideal circumstances.

- Confirmation of the presence of a familial abnormality in first degree relatives of patients with functional test abnormalities is desirable.
- Comprehensive testing is recommended. Subjects with VTE often have more than one abnormality.
- The significance of anticardiolipin antibodies in subjects with VTE is currently controversial

The issue of screening for thrombophilic defects is controversial⁹. The laboratory evaluation is expensive, and the short-term treatment of venous thromboembolism is the same in all patients, regardless of cause; therefore, it is unclear which patients warrant screening. In addition, there is little evidence to show that testing would influence the intensity or duration of long-term anticoagulation, except in the antiphospholipid syndrome

Although certain tests can be performed at the time of the initial event, heparin interferes with clotting-based assays for APC resistance, the lupus anticoagulant, and factor VIII levels. Protein C, protein S, and antithrombin functional and antigenic tests should be performed only in strongly thrombophilic patients: those with a venous thromboembolism prior to age 50, with recurrent venous thromboembolism, or with an extensive family history of thrombus. In addition, testing for protein C, protein S, and antithrombin cannot be reliably performed during an acute event or while the patient is taking anticoagulants, because the levels fluctuate during active thrombosis and are suppressed by warfarin therapy. If indicated, testing for antithrombin, protein C, and protein S can be performed 3 weeks after anticoagulant therapy has been discontinued. In contrast, there are many compelling arguments to test *appropriate* patients for inherited thrombophilia. Testing advances the knowledge base of the pathophysiology of venous thromboembolism, although data on specific recommendations for length and intensity of anticoagulant therapy are lacking. More importantly, identifying patients at risk for thrombosis carries significant implications in *family counselling* and high-risk situations. The affected family members of individuals with an identified hypercoagulable defect are also at increased risk of thrombosis.

When venous thromboembolism occurs in a woman taking

oral contraceptives, testing may be warranted in order to provide adequate counseling about continued oral contraceptive use and the risks of thromboembolism in pregnancy. Similarly, testing for factor V Leiden may be warranted when venous thromboembolism occurs in breast cancer patients taking tamoxifen (Nolvadex), because of an increased risk of thrombosis in this group.

Venous thromboembolism is a common disease that causes significant morbidity and mortality⁴. In recent years, the ability to diagnose inherited genetic defects and common acquired conditions predisposing to thrombosis has greatly increased. Venous thromboembolism is now understood to be a complex interaction of genetic and environmental factors leading to thrombosis. Integrating the various factors to individually assess thrombotic risk still poses a challenging clinical problem that will likely become easier as more data accumulate. As the ability to accurately assess risk increases, the data can then be translated into tailored treatment regimens. Until then, only general guidelines regarding evaluation and management are available. In the future, it is likely that other prothrombotic conditions will be elucidated, adding to the pool of data

RECOMMENDED READING

1. Bauer KA, Goodnight SH, Ridker PM. Hypercoagulable states—translation of risk factors to clinical practice. *American Society of Hematology education session, Dec 1998*:255-73
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5. Bick RL, Kaplan H. *Syndromes of thrombosis and hypercoagulability: congenital and acquired causes of thrombosis. Med Clin North Am 1998*;82(3):409-58
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11. Seligsohn U, Lubetsky A. *Genetic susceptibility to venous thrombosis. N Engl J Med 344*:1222-1232, 2001.
12. Van Cott EM, Laposata M. *Laboratory evaluation of hypercoagulable states. Hematol Oncol Clin North Am 1998*;12(6):1141-66

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