Deep Venous Thrombosis: Review Questions

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QUESTIONS

Choose the single best answer for each question.

1 **The pathogenesis of deep venous thrombosis (DVT) involves 3 factors, known as Virchow's triad. All of the following are part of the triad EXCEPT**
(A) Damage to the vessel wall
(B) Hypercoagulopathy
(C) Past history of thrombosis
(D) Venous stasis

Questions 2 to 4 refer to the following case.

A 65-year-old woman presents to the emergency department with a swollen right calf. She has a history of lung cancer and is receiving chemotherapy. DVT is suspected, and a D-dimer test is ordered, the result of which is highly positive. Records of previous blood tests show that this patient's D-dimer levels were always higher than normal.

2. **What is the next step in the management of this patient?**
(A) Order a brain natriuretic peptide test to rule out heart failure caused by chemotherapy
(B) Order compression ultrasonography
(C) Order a venogram
(D) No further evaluation

3. **The patient is found to have DVT of the calf with extension to the right proximal vein. Anticoagulation therapy should be initiated. Which of the following is a relative contraindication to anticoagulation therapy?**
(A) Brain metastasis
(B) Gastrointestinal bleeding in the past 6 months
(C) Platelet count $< 60 \times 10^3/\mu L$
(D) Severe hypotension

4. **The patient is prescribed unfractionated heparin. All the following are adverse effects associated with unfractionated heparin EXCEPT**
(A) Elevated partial thromboplastin time
(B) Osteoporosis
(C) Thrombocytopenia
(D) Worsening of renal failure

(turn page for answers)

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ANSWERS AND EXPLANATIONS

1. **(C) Past history of thrombosis.** Past history of thrombosis is not part of the Virchow's triad. Damage to the vessel wall causes a dysfunctional endothelium followed by increased platelet activation and aggregation. This process also inhibits the initiation of local fibrinolysis. Venous stasis causes decreased flow, which in turn inhibits the clearance and dilution of activated coagulation factors. The normal clotting–fibrinolytic system involves a fine balance between activation and inhibition of platelets, procoagulant factors, anticoagulant factors, and fibrinolytic factors. The hypercoagulable conditions are a group of inherited and acquired disorders that predispose to venous thromboembolism.
2. **(B) Order compression ultrasonography**. Compression ultrasonography is noninvasive and has a sensitivity of 70% and a positive predictive value of 80% for isolated calf vein thrombosis. To evaluate a possible extension of the calf vein thrombosis into the proximal veins, a follow-up ultrasonography 1 week after initial ultrasonography is recommended. The sensitivity and specificity of compression ultrasonography for proximal DVT is more than 95%.[1] Venography can be used when ultrasonography is negative in a patient with highly suggestive clinical signs and symptoms (eg, pain in the leg, leg tenderness, swelling). The probability that the patient has decompensated heart failure is low (unilateral leg swelling and no shortness of breath), and therefore measurement of brain natriuretic peptide is not warranted.
3. **(A) Brain metastasis**. Absolute contraindications for anticoagulant therapy are active bleeding, platelet count less than $20 \times 10^3/\mu L$, history of neurosurgery or ocular surgery, and intracranial bleeding within the past 10 days. Brain metastasis, gastrointestinal bleeding within the past 14 days, and severe hypertension are the relative contraindications to anticoagulant treatment.
4. **(D) Worsening of renal failure.** Worsening of renal failure is not an adverse effect of heparin. Unfractionated heparin has been used in dialysis patients to prevent clots, and there is no need to adjust the heparin dose to the renal function. Heparin-induced thrombocytopenia (HIT) is an adverse effect of heparin treatment, and type II HIT is life-threatening and one of the most serious and common immune-mediated drug adverse reactions. Type II HIT typically occurs 4 to 5 days after initiation of heparin therapy. Type I HIT is benign and develops 1 to 3 days after heparin treatment is started but spontaneously resolves without stopping therapy.[2] The use of unfractionated heparin longer than 1 month can cause osteoporosis.[3] Doses of unfractionated heparin should be adjusted in accordance to the activated partial thromboplastin time.

REFERENCES

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