PATHOPHYSIOLOGY OF DVT

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BACKGROUND

- DVT, one of the most common and serious forms of VTE, has an incidence of about 200,000 cases per year.

METHODS

- We researched the literature and pathophysiology of DVT.
- Virchow's triad (Figure 1) consists of venous stasis, vessel wall damage, and a hypercoagulable state.

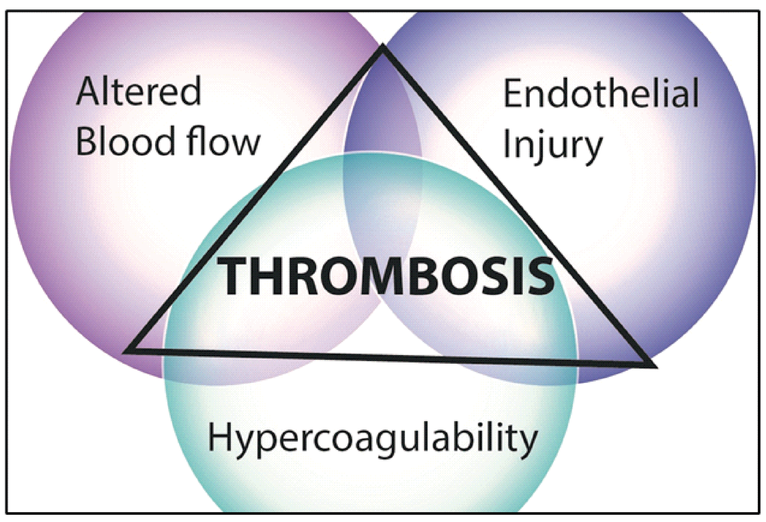

Figure 1: Virchow's Triad

RESULTS

- Seat-edge pressure to the popliteal area can aggravate venous stasis and contribute to vessel wall damage.
- Factors specific to air travel may increase coagulation activation, particularly in travelers with preexisting risk factors for VTE.
- Deep venous thrombosis usually begins in venous valve cusps.
- The thrombus consists of thrombin, fibrin, red blood cells, and platelets.
- Without treatment, thrombi may propagate proximally or travel to the lungs.
- Coagulation activation may result from an interaction between cabin conditions (such as hypobaric hypoxia) and individual risk factors for VTE.
- Injury to the endothelium results in formation of a thrombus, resulting in hypoxia, followed by a release of enzymes, and finally symptoms of DVT.

RESULTS, CONTD.

- Inappropriate thrombus formation is a disruption of homeostasis and may result from an alteration in any of the factors listed below.
- Endothelial injury causes subendothelial collagen exposure and platelet adherence.
- Factors contributing to the injury (hypertension, vasculitis, scarred valves, bacterial endotoxins, and chemicals from cigarette smoke) should not be overlooked.
- Stasis can cause endothelial injury, predisposing a patient to thrombosis.
- Hypercoagulability, caused by advancing age, surgery, fractures, burns, myocardial infarction, and cancer can put them at risk for thrombosis.
- Increased hypercoagulability causes an increase in platelet aggregation, which results in reduction of prostacyclin, a potent vasodilator, and inhibitor of platelet aggregation that is released by the endothelium (Figure 2).

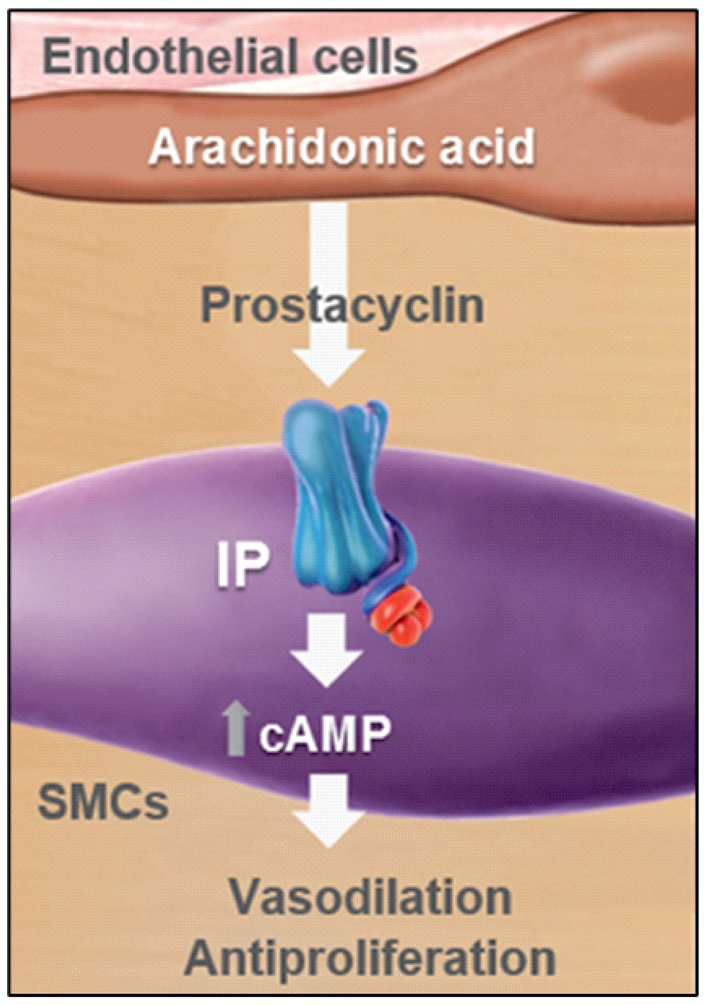

Figure 2: Prostacyclin pathway

RESULTS, CONTD.

- DVT can cause serious complications if thrombi travel to the lungs resulting in PE.
- Post thrombotic syndrome is a serious complication of DVT.
- DVT and PE have worse outcomes than DVT alone.

CONCLUSIONS

- In conclusion, DVT is a global problem affecting all ages, races, and genders. The primary problem with DVT is sluggish circulation, followed by hypoxia, endothelial injury, and the release of enzymes.
- Prevention of DVT is much easier than treating DVT. Virchow's Triad plays a key role in the genesis of DVT.

IMPORTANCE OF OUR WORK

DVT can cause serious complications if thrombi travel to the lungs resulting in PE. DVT and PE have worse outcomes than DVT alone. Prevention of DVT is much easier than treating DVT. Virchow's Triad plays a key role in the genesis of DVT.

The authors disclose no conflict of interest.

REFERENCES

1. Navarrete, Simon; Solar, Carla; Tapia, Roberto, et al: Pathophysiology of deep vein thrombosis, Clin Exp Med, Apr 26, 2022
2. Reyes, N, and Abe, Karon: Deep Vein Thrombosis & Pulmonary Embolism: CDC Yellow Book 2024
3. Voicu, S., Ketfi, C., Stépanian, A., et al: Pathophysiological processes underlying the high prevalence of deep vein thrombosis in critically ill covid-19 patients, Frontiers, November 5, 2022
4. Zagaria, M.: Venous thrombosis: Pathogenesis and Potential for Embolism. U.S. Pharmacist, 2022

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