v. 4, n. 31, 2024

International Journal of Health Science

HOW TO ANTICOAGULATE: CLINIC AND MANAGEMENT OF PULMONARY THROMBOEMBOLISM – LITERATURE REVIEW

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Conflict of interests: nothing to disclose.

Abstract: Introduction: Pulmonary thromboembolism is a clinical syndrome characterized by obstruction of the pulmonary artery or one of its branches by thrombi, air or fat droplets, originating from the systemic venous circulation (Konstantinides SV, et al. 2014). **Goal**: To review anticoagulation and management of pulmonary thromboembolism. **Result**: We must reach the state of full anticoagulation within the first 24 hours of therapy. This is only possible with the use of parenteral medications or new oral anticoagulants (direct thrombin or factor Xa inhibitors), whose onset of action is immediate (Tapson VF, 2008). **Conclusion:** Anticoagulation reduces the mortality rate by around 80-90%, basically due to its ability to prevent PTE recurrence (secondary prevention) (Kline JA, et al. 2008).
Keywords: Pulmonary embolism; Anticoagulants.

INTRODUCTION

Pulmonary thromboembolism is a clinical syndrome characterized by obstruction of the pulmonary artery or one of its branches by thrombi, air or fat droplets, originating from the systemic venous circulation (Konstantinides SV, et al. 2014).

The pathophysiology involves Virchow's triad, which comprises blood stasis, endothelial injury and hypercoagulable state. After a thrombus forms in another part of the body (mainly lower limbs), it travels through the circulation, impacting the lung (Goldhaber SZ, 2002).

The clinical presentation is very variable, ranging from asymptomatic conditions (the majority) to severe conditions with hemodynamic instability, or even sudden death (European Society of Cardiology, 2000).

In medical practice, the majority of cases are due to Venous Thromboembolism (VTE), an entity that is based on Deep Vein Thrombosis (DVT), and as an acute complication Pulmonary Thromboembolism (PTE). Other less common causes of pulmonary embolism are: gas bubbles (careless handling of equipment, sudden decompression in divers), foreign bodies (e.g. talc, in illicit drug users) and fat droplets (polytraumatized patients) (European Society of Cardiology, 2000).

In this topography, the DVT with the highest risk of significant embolism is iliofemoral (50% occur with PTE), while the risk of PTE with clinical manifestations, in calf DVT, is considered extremely low (because such emboli are very small) (Konstantinides SV, et al. 2014).

However, calf DVT is the main cause of iliofemoral DVT (due to the upward propagation of the thrombus), in addition to being the main cause of paradoxical embolism (in patients with patent foramen ovale). Thrombosis in pelvic veins also confers a high risk of PTE (around 50%) (Konstantinides SV, et al. 2014).

Therapeutic modalities in VTE include the use of anticoagulants, thrombolytics, vena cava filter and invasive procedures (surgical or endovascular) (Kline JA, et al. 2008).

Anticoagulation reduces the mortality rate by around 80-90%, basically due to its ability to prevent PTE recurrence (secondary prevention) (Kline JA, et al. 2008).

No anticoagulant exerts a direct action on the thrombus. The dissolution is carried out by the endogenous fibrinolytic system: what the anticoagulant does is inhibit the continuity of the thrombotic process, through fibrinolysis (Goldhaber SZ, 2002).

We must reach the state of full anticoagulation within the first 24 hours of therapy. This is only possible with the use of parenteral medications or new oral anticoagulants (direct thrombin or factor Xa inhibitors), whose onset of action is immediate (Tapson VF, 2008). In patients

receiving coumarins (vitamin K antagonist oral anticoagulants), it takes about 5-7 days for the anticoagulant effect to be achieved. For this reason, when we choose to use these medications, it is necessary to maintain a parenteral drug until the full anticoagulant effect of the coumarin is proven to be obtained (for example: two consecutive INR measurements within the therapeutic range, which is between 2-3) (Goldhaber SZ, 2002).

Thrombolytics can be administered up to 14 days after the onset of the PTE episode. We must bear in mind that thrombolytics can only be administered once the diagnosis of PTE has been confirmed. The only absolute indication for the use of thrombolytics in PTE is the presence of hemodynamic instability (Goldhaber SZ, 2002).

MATERIAL AND METHODS

The search was carried out in the PubMed database and was limited to articles between 2000 and 2023 that met the criteria of being literature reviews and case reports.

Next, the keywords in the article titles were analyzed and those whose themes best fit our objective were selected.

5 articles were selected for full reading.

DISCUSSION

No anticoagulant exerts a direct action on the thrombus. The dissolution is carried out by the endogenous fibrinolytic system: what the anticoagulant does is inhibit the continuity of the thrombotic process, through fibrinolysis (Goldhaber SZ, 2002).

CONCLUSION

Anticoagulation reduces the mortality rate by around 80-90%, basically due to its ability to prevent recurrence of PTE (secondary prevention) (Goldhaber SZ, 2002).

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