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# Infectious Thromboembolism related to prolonged reduced motility in ill acute patients

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## Abstract

***Update in new clinic complications related to infectious diseases reveals emergent, at present, the thromboembolic risk. Clinicians must examine carefully thromboembolism related to prolonged reduced motility during acute invalidating infectious diseases because it represents major risk during clinical conditions involving enzimatic procoagulant activity, fibronectine production, procoagulant autoimmunitary damage. Recent experimental data show that pathogenesis of thromboembolic lesions involves clinical risk factors, vascular anatomic features and bacterial genetic properties.***

Prolonged immobility for acute infectious diseases (ID) produces same thromboembolic risk as in internal medical patients. So, infections represent a veritable cause of thromboembolic disease. Disease caused by infectious thromboembolism (TE), related to prolonged reduced motility, is an highly specialist clinic problem in ID. At present, this risk is similar to the internal medicine (IM), mostly in acute ID [1]. If ID pathogenesis does not imply the TE risk only a general risk exists, but if ID is characterized by TE, a greater risk occurs, both general and specific of the disease itself.
According to experimental data, pathogenesis shares: endothelial damage, stagnation or disturbance of haematic flow, blood hypercoagulability. In the five major studies about thromboprophilaxis in IM (MEDENOX, PREVENT, ARTEMIS, EXCLAIM, ENDSORGE) produced in 1999-2010 years, TE related risk was 39.5%-63% [2-6]. In other two outpatients studies, the same datum was included between 1.1% and 3.1% [7]. In these experimental studies, patients with ID were numerous and well represented.
According to the scientific literature, endothelial damage caused by clinical devices (central vascular catheter or pace-maker) resulted an emerging risk factor. Strains of *Staphylococcus aureus* and *Bacteroides fragilis* which produce, respectively, intrinsic enzimatic procoagulant activity, endocellular internalization through fibronectine and production of eparinase can have a pathogenetic role in TE. *S. aureus* is involved also in a mechanism of endotelial flogosis, production of fibronectine binding proteins and interaction with thrombocytic integrins. *Candida albicans* can produce septic flebitis. High levels of soluble fibrine (SF) or D-Dimer are considered markers of TE risk, with secure diagnosis in more than 50% (statistical significance) [8]. Antiphospholipidic antibodies (APA) indicate statistically significant (65%) thromboembolic risk in endocarditis [9]. Therefore, critical patients for severe ID with various thromboembolic risk factors generate TE in high percentage. Prophylaxis with eparin is helpful and can reduce this risk until 73% [10].
Personalization of the protocol for age, reduced mobilization, gastric or cerebral bleeding is mandatory. Patients can continue a long term prophylaxis, also at home [5, 10].
Microbiological thrombotic characterization of described bacteria and APA monitoring, as D-Dimer and SF represent an alert condition in the ID with greater thrombotic risk.

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