

Tromboembolismo venoso: aspetti clinici e diagnostici

HYPERCOAGULABILITY AND THROMBOTIC RISK IN MODERATE-TO-SEVERE OBSTRUCTIVE SLEEP APNEA: A PROSPECTIVE OBSERVATIONAL STUDY

L. Spiezia[1], S. Carraro[2], E. Bicego[1], A. Napolitano[1], C. Samà[1], A. Benetti[1], B. Serena[1], R. Marchese Ragona[2], P. Simioni[1]

[1]Dipartimento di Medicina, Azienda Osp. Università di Padova, Italy; [2]Dipartimento di Neuroscienze, Azienda Osp. Università di Padova, Italy

Background and Aims. Obstructive sleep apnea (OSA) is a common and clinically significant disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia and sleep fragmentation. Increasing evidence suggests that OSA may promote a prothrombotic state through several interconnected mechanisms, including oxidative stress, endothelial dysfunction, platelet activation, and alterations in coagulation and fibrinolysis pathways. These changes may contribute to an elevated risk of cardiovascular and thrombotic complications in affected individuals.

Methods. We conducted an observational study involving adult patients (≥18 years) diagnosed with untreated moderate-to-severe OSA, defined by an apnea–hypopnea index (AHI) greater than 15 events per hour. Patients were excluded if they had experienced recent cardiovascular events, had known coagulation disorders, active prothrombotic conditions, or were receiving anticoagulant therapy. For each participant, demographic and clinical characteristics, including body mass index (BMI), were collected. Both routine coagulation tests and more specific hemostatic markers were assessed through blood sampling. Participants were prospectively followed for a period of six months to document the occurrence of thrombotic events. The study group consisted of 41 patients with OSA, who were compared with 41 healthy controls matched for age and sex.

Results. Standard coagulation parameters, including prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), as well as fibrinogen, antithrombin, plasminogen, and plasminogen activator inhibitor-1 (PAI-1), showed no significant differences between OSA patients and controls. In contrast, specific procoagulant factors were significantly elevated in the OSA group, including factor VIII ($P = .011$), factor XI ($P < .001$), and the factor VIIa–antithrombin complex ($P = .009$). No significant differences in these markers were observed between patients with moderate versus severe OSA, and no correlation was found between AHI values and the levels of these coagulation factors. During the six-month follow-up period, 17% of patients experienced thrombotic events. These events were associated with higher baseline levels of the identified procoagulant factors.

Conclusions. Moderate-to-severe OSA is associated with a selective increase in specific procoagulant factors, despite largely normal findings in routine coagulation tests. This pattern suggests the presence of a hypercoagulable state that may contribute to an increased risk of thrombotic events. Notably, these hemostatic alterations appear to be independent of OSA severity, indicating that prothrombotic mechanisms are already active even in moderate forms of the disease.