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POREMEĆAJI KOAGULACIJE I FUNKCIJE TROMBOCITA U BOLESTIMA BUBREGA

DISORDERS OF COAGULATION AND PLATELET FUNCTION IN KIDNEY DISEASES

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SAŽETAK

Bolesnici s bubrežnom bolešću se suočavaju s povećanim rizikom i od krvarenja kao i od venske i arterijske tromboze. Bolesti bubrega su često praćene poremećajima hemostaze. Ovi poremećaji mogu biti posljedica abnormalnosti na nivou primarne hemostaze (najčešće disfuncije trombocita i von Willebrandovog faktora (vWF), ali i uskljed komplikacija krvarenja, koja uključuju bolesnike s bubrežnom disfunkcijom, kako one s akutnim oštećenjem bubrega, tako i one sa hroničnom bubrežnom bolesti i posebno bolesnike na nekoj od metoda zamjene bubrežne funkcije.

Poremećaj koagulacije može se javiti kako u bolesnika s akutnim oštećenjem bubrega, tako i u hroničnoj bolesti bubrega. U akutnom oštećenju bubrega različiti etiološki uzroci u uremijskom miljeu mogu uticati na nastanak koagulopatije neposrednim ili posrednim djelovanjem na kaskadu koagulacije, što može rezultirati hiperkoagulabilnim stanjem, ali i krvarenjem. Iako se često naziva "uremijskim krvarenjem", ono se može pojaviti čak i kod bolesnika koji ne pokazuju kliničke simptome ili znakove uremije. Hronična bubrežna slabost 4 i 5 stepena je povezana s promjenama u koagulaciji koja pogoduje hiperkoagulabilnom ili protrombotičkom stanju. Poremećena funkcija trombocita značajan je faktor abnormalnog krvarenja u bolesnika s niskom jačinom glomerulske filtracije. Povećani rizik od tromboze doprinosi povećano kardiovaskularnom morbiditetu i mortalitetu u ovih bolesnika.

Trombociti imaju ključnu ulogu u primarnoj hemostazi. Aktivacija trombocita uključuje interakcije između subendotelnog kolagena i receptora trombocitnog glikoproteina VI (GPVI), pojačane trombinskom aktivacijom receptora aktiviranih trombocitnom proteazom (PAR4). Uz to cirkulirajući von Willebrandov faktor (vWF) učvršćuje kolagen u subendotelnim tkivima preko receptora glikoproteina Ib (GPIb) na trombocitima. Aktivacija trombocita dovodi do oslobađanja sadržaja alfa i gustih granula, uključujući adenozin difosfat (ADP) i tromboksan A2, koji su neophodni za dodatno regrutisanje trombocita i stvaranje agregata.

Upravljanje koagulacijom u bolesnika sa bolestima bubrega zahtijeva delikatnu ravnotežu između antikoagulacije i trombotičkog rizika. I u akutnom i hroničnom oštećenju bubrežne funkcije ono uključuje procjenu rizika od krvarenja i tromboze i individualiziran pristup. Nažalost, ne postoje precizne smjernice za istraživanja i upravljanje, što komplikuje kompleksnost problema.

Ključne riječi: koagulacija, bolesti bubrega, trombociti

ABSTRACT

Patients with kidney disease face an increased risk of bleeding as well as venous and arterial thrombosis. Kidney diseases are often accompanied by haemostasis disorders. These disorders can be the result of abnormalities at the level of primary haemostasis (most often dysfunction of platelets and von Willebrand factor (vWF), but also due to bleeding complications, which include patients with renal dysfunction, both those with acute kidney damage and those with chronic kidney disease, and especially patients on one of the methods of replacement of renal function.

Coagulation disorders can occur both in patients with acute kidney damage and in chronic kidney disease. In acute kidney damage, various etiological causes in the uremic milieu can influence the occurrence of coagulopathy by direct or indirect action on the coagulation cascade, which can result in a hypercoagulable state, as well as bleeding. Although often referred to as "uraemic bleeding," it can occur even in patients who do not show clinical symptoms or signs of uraemia. Chronic renal failure of degrees 4 and 5 is associated with changes in coagulation favouring a hypercoagulable or prothrombotic state. Impaired platelet function is a significant factor in abnormal bleeding in patients with low glomerular filtration rate. The increased risk of thrombosis contributes to increased cardiovascular morbidity and mortality in these patients.

Platelets play a key role in primary haemostasis. Platelet activation involves interactions between subendothelial collagen and platelet glycoprotein VI (GPVI) receptors, enhanced by thrombin activation of platelet protease-activated receptors (PAR4). In addition, circulating von Willebrand factor (vWF) stabilizes collagen in subendothelial tissues via the glycoprotein Ib (GPIb) receptor on platelets. Platelet activation leads to the release of alpha and dense granule contents, including adenosine diphosphate (ADP) and thromboxane A2, which are essential for further platelet recruitment and aggregate formation.

Coagulation management in patients with kidney disease requires a delicate balance between anticoagulation and thrombotic risk. In both acute and chronic impairment of renal function, it includes an assessment of the risk of bleeding and thrombosis and an individualized approach. Unfortunately, there are no precise guidelines for research and management, which complicates the complexity of the problem.

Keywords: coagulation, kidney disease, platelets