PB2232 | The Prothrombin Belgrade Mutation Causing Antithrombin Resistance Does Not Affect Fibrin Clot Formation

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Background: Prothrombin Belgrade mutation is a result of the c.1787G>T mutation in the prothrombin gene, which leads to the substitution of Arg596 by Gln. This mutation is located in the antithrombin binding site and leads to the impaired inactivation of thrombin by antithrombin and antithrombin resistance, resulting in a thrombotic phenotype. Previous studies have shown the complex mechanism of this mutation, manifested with higher endogenous thrombin potential, lower prothrombin activity with normal prothrombin levels in carriers' plasma.

Aims: Considering that Prothrombin Belgrade mutation mechanism is still not fully elucidated, our aim is to determine the effect of this mutation on fibrin clot formation and its lysis.

Methods: Recombinant wild type and mutated prothrombin was generated by transient transfection of HEK293T cell line. Using recombinant proteins and prothrombin deficient plasma, samples that correspond to plasma of a non-carrier, heterozygous and homozygous carrier was reconstituted. Reconstituted plasma samples with the addition of thrombin, tissue plasminogen activator (t-PA), phospholipids and calcium-chloride were used in OHP assay (Overall Hemostasis Potential) to determine the kinetic profiles of coagulation (Overall Coagulation Potential, OCP) and fibrinolysis (Overall Fibrinolytic Potential, OFP). Fibrin clots formed in reconstituted plasma samples with the addition of fluorescent fibrinogen, were analyzed by confocal microscopy in order to determine the fluorescence intensity and the density of fibrin network. In addition, fibrin clots prepared using OHP assay protocol, were observed using electron microscopy in order to determine the thickness of individual fibrin fibers.

Results: There was no significant difference between OHP, OCP and OFP parameters between tested reconstituted plasma samples ($p>0.05$). Fibrin network density and fibrin fiber thickness between non-carrier, heterozygous and homozygous carrier reconstituted plasma did not differ significantly ($p>0.05$).

Conclusions: Our results indicate that Prothrombin Belgrade mutation does not affect fibrin formation or its lysis.

PB2233 | Is May-Thurner Syndrome a Forgotten Cause of Deep Vein Thrombosis?

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Background: May-Thurner syndrome (MTS), also known as Cockett's syndrome or iliac vein compression syndrome, is a rare cause of iliofemoral deep venous thrombosis (DVT). An anatomic anomaly produces chronic compression of the left common iliac vein by the overlying right common iliac artery when it passes between the right common iliac artery and the spine. Prolonged compression on the vein potentiates thrombus formation by impairing the intima and by leading to the development of membranes within the lumen that may decrease and/or block venous flow.

Aims: Presentation of 4 cases of MTS.

Methods: Collection of clinical data in SClínico® application.

Results: Patient 1 is a 54-year-old male with signs of DVT. Computed tomography scan (CT-scan) confirmed MTS. Despite ongoing anticoagulation with rivaroxaban, he developed a post-thrombotic syndrome and is waiting for endovascular treatment. Patient 2, a 15-year-old female, initiated oral contraceptive 1 month before presenting with left DVT complicated with bilateral pulmonary thromboembolism. CT-scan confirmed MTS. She remains asymptomatic and hypocoagulated with warfarin (Time in Therapeutic Range >80%). Patient 3, a 19-year-old obese female, maintained leg pain after 6-month anticoagulation for DVT despite absence of DVT signs in duplex ultrasound. Magnetic resonance imaging (MRI) confirmed MTS. Rivaroxaban was restarted and she's currently asymptomatic. Patient 4, a 30-year-old female, presents acute pain and left leg swelling in her puerperium. Her workup revealed left-sided acute DVT secondary to MTS, confirmed by MRI.

Conclusions: MTS may be underdiagnosed, therefore all clinicians treating DVT should be aware of it. Anticoagulation, clinical follow-up and non-invasive imaging play critical roles in the diagnosis and prevention of relapse and complications associated with MTS. Despite none of our patients underwent stenting, guidelines nowadays recommend venous stenting to relieve symptoms and prevent relapse. Patient 4 emphasizes that DVT during puerperium should not only be attributed to hypercoagulability secondary to pregnancy.

PB2234 | Relevance of Thrombophilia Screening in Adult Renal Transplant Recipients

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Background: Thrombosis after kidney transplantation may result in catastrophic outcomes. Thrombophilia has been implicated in post-transplant thrombosis. However, data concerning the impact of thrombophilia on thrombotic risk in renal graft recipients are inconclusive.

Aims: Evaluate whether identifying patients with thrombophilia during pretransplant laboratoring screenig predicted post-transplant outcomes.

Methods: A single-center prospective study between 2011 and 2017 was performed. Pretransplant thrombophilia screening including antithrombin (AT), protein C (PC), protein S (PS) and activated