for reducing thrombotic risk and fatigue in sLE patients. Further studies are needed to address this issue.

**P4** ANTIPHOSPHOLIPID SYNDROME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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**Background** Antiphospholipid Syndrome (APS) is a known cause of thrombotic disorders, including Acute Myocardial Infarction (AMI). Although its incidence in AMI patients is not known, it may be an important factor in precipitating infarction, especially in younger adults.

**Methods** This is a case-control study consisting in 73 patients with Acute Myocardial Infarction, hospitalized at Cardiovascular Reanimation Clinic from 10 December 2015-10 October 2019. All patients included in the study were from 23 to 50 years old. In the case-group were included 24 patients with Antiphospholipid Syndrome and Acute Myocardial Infarction, and 49 patients were included in the control group, which consisted only in patients with AMI, with no presence of APS. In every patient were gathered data such as complete blood count + ESR, Antinuclear Antibodies (ANA), Anti-cardiolipin antibodies (ACA), C3 and C4 complement fraction, Anti-ds-DNA, CRP and Ejection Fraction (EF) in echocardiography. Female patients were asked about their abortion history. As recommended in the guidelines, in positive results for APS, ACA levels were repeated after 12 weeks in order to establish the diagnosis. All data were gathered and statistically analyzed using Excel 2010 and IBM SPSS.

**Results** After comparing all gathered data, it was found that the patients with APS and AMI had a more significant tendency to have C3 hypocomplementemia (p=0.006), thrombocytopenia (p=0.002), a lower ejection fraction on transthoracic echocardiography (p=0.04) and a more elevated number of abortions before acute myocardial infarction (p=0.03) in comparison to the controls.

**Conclusions** From our study it was found that APS is not rare in young adults with AMI. It should be always suspected in young patients with no cardiovascular risk factors and there may be a characteristic clinical and laboratory picture in patients with AMI, which may suggest the APS diagnosis.

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**P5** RECOGNIZING THE DISEASE, TREATING THE PATIENT: A MIXED-METHOD EVALUATION OF CARE FOR THE ANTIPHOSPHOLIPID SYNDROME (APS) IN THE NETHERLANDS

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**Background** Catastrophic antiphospholipid syndrome (CAPS) - thrombotic microvasculopathy, characterized by involvement of systems and organs with formation of their insufficiency. Therapy of glucocorticoids and immunosuppressants with plasmapheresis and intravenous immunoglobulin demonstrates lack of effectivity. Currently, there are reports of rituximab effectiveness in APS and CAPS, but they are not numerous, especially CAPS.

**Methods** Assessment of the rituximab effectiveness in patients with resistant CAPS. We present the treatment results of two patients with CAPS, who were treated in our clinic.

**Results** Patient V, 21, was admitted in the early postpartum period. Diagnosis: Systemic lupus erythematosus, positivity for ANA: ds-DNA, Sm. Secondary antiphospholipid syndrome (positivity for β2-glycoprotein-1, cardiolipin, lupus anticoagulant), catastrophic APS with multiorgan failure: epilepsy, sopor, psychosis; acute renal injury, signs of acute respiratory distress syndrome and hypocoagulation. SLEDAI score was 56 points, GAPSS ≥ 17 points. Pulse therapy with methylprednisolone, cyclophosphamide, plasmapheresis and intravenous immunoglobulin wasn’t effective enough. Rituximab course 375 mg/m² 1 time per week for 4 weeks was administered. Manifestations
of CAPS in multiorgan failure form regressed 14 days after the first administration, SLEDAI score decreased to 32 points, GAPSS to 4 points. After 4 weeks complete B-cell depletion was achieved. A month after the course of rituximab she achieved remission, which lasts 4 years already. ANA and anti-phospholipid antibodies weren’t detected.

Patient E., 20, with primary APS, cardiolipin positivity, β2-glycoprotein-1 and lupus anticoagulant, thrombocytopenia, livedo reticularis; CAPS-like thrombotic microangiopathy type with damage to cerebral vessels, lungs vessels, recurrent pulmonary embolism for six months, deep leg vein thrombosis. GAPSS activity before treatment was 17 points. Pulse therapy wasn’t performed. Therapy rituximab 375 mg/m² 1 time per week, 4 weeks was administered. Multiple organ failure also regressed 10–14 days after 1 administration of rituximab. GAPSS score decreased to 10 points. Incomplete B-cell depletion was achieved.

Conclusion Thus, rituximab demonstrated high effectiveness in CAPS in both cases. Rituximab allowed to reach multiple organ failure regression and a persistent effect was achieved.

P8 REFRACtORY THROMbocyTPeRiA IN A PATiENT wITh SYSTEmIC LUPUS ERYTHEMATOUS AND SECONDARY ANTIphoSLiPid SYNDROME

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Background Catastrophic antiphospholipid syndrome (CAPS) is the most severe complication of antiphospholipid syndrome (APS). Vitamin K antagonists (VKAs) are the reference treatment for preventing relapsing thrombotic complications in patients with APS, but direct oral anticoagulants (DOACs), such as rivaroxaban, are nonetheless sometimes used in patients with antiphospholipid antibody profiles or APS. Recent studies showed that DOACs were associated with more arterial thromboses among patients with APS. The potential role of DOACs as a trigger factor of CAPS is not known.

Methods We describe two patients who developed a CAPS in the week following the institution of rivaroxaban.

Results We report the onset of definite CAPS in the week following introduction of rivaroxaban treatment in two patients, one with APS and the other with antiphospholipid laboratory findings only. Both were triple positive for antiphospholipid antibodies. The affected organs were the heart, kidneys, skin, and liver for Patient 1, and the heart, kidneys, skin, adrenal gland, and central nervous system for Patient 2. The causative role of rivaroxaban is highly probable given that (1) CAPS occurred rapidly after this treatment was started, (2) an alternative trigger factor was found in Patient 1 only (a colonoscopy), and (3) Patient 1 had been clinically stable for 18 years with VKA as anticoagulant treatment, while Patient 2 did not have APS and had had no symptoms for 4 months (rivaroxaban had been introduced because at a scheduled visit, she reported neurological symptoms that occurred four months before and were retrospectively compatible with a brain transient ischemic accident). One similar case was reported in 2017, also following the introduction of rivaroxaban, in a patient with triple positive venous thrombotic APS who was stable for years on warfarin and who developed definite CAPS (involvement of the myocardium and adrenal glands as well as a pulmonary embolism) in the week after rivaroxaban 20 mg daily replaced warfarin to meet the patient’s desire for a less burdensome treatment. Finally, in the randomized study published by Ordi-Ros et al, one of the patients treated with rivaroxaban developed a CAPS.

Conclusions These two cases, as well as two previous reported cases, underline the importance of avoiding DOACs in patients with APS, especially those triple positive for antiphospholipid antibodies. VKAs must remain the reference anticoagulation treatment in this setting.

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