Response Criteria: A Complete Response(CR) was defined as a rise in platelet count >100×10⁹/L,a Partial Response(PR) as a rise in Platelet count >50×10⁹/L and a minor response(MR) as a rise in Platelet count <50×10⁹/L. No Response(NR)was defined as no increase in Plateletcount. *Results.* Of the 4 patients 1 had a Minor Response and 3 had No Response. However Rituximab was well tolerated in all 4 cases with no major side-effects. *Conclusions.* Our results suggest that Rituximab hardly made any impact on the platelet count of these 4 patients with chronic refractory ITP. Previous studies of Rituximab in ITP has shown an overall response rate of around 50%.However such initial results must be considered in the light of positive report bias,small numbers, lack of long term follow up and lack of randomised controlled trials.In addition, data on short and long term side-effects of Rituximab are lacking. Thus, Rituximab is an unproven treatment for chronicrefractory ITP. Perhaps novel agents like Thrombopoeitin Receptor Agonists should be considered for these patients with chronic ITP,in the setting of a Clinical Trial.

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EFFECTS OF VARIOUS THERAPEUTIC REGIMENS ON PLATELET FUNCTIONS IN PATIENTS WITH MYELOPROLIFERATIVE DISORDERS

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Bleeding and thrombosis are common causes of morbidity and mortality in patients with myeloproliferative disorders (MPD). Qualitative platelet abnormalities are frequently found in these patients and range from platelet hypofunction, acquired storage pool disease and/or platelet membrane defects, to abnormalities suggesting increased platelet reactivity, increased plasma β thromboglobulin levels or shortened platelet survival. In the present study, we aimed to investigate platelet function abnormalities using both optical platelet aggregometry and whole blood platelet aggregometry and evaluate the effects of various therapeutic regimes on these abnormalities, in patients with MPD. 45 patients with newly diagnosed chronic myeloproliferative disorders (26 CML, 11 PCRV, 8 ET) were enrolled. Median age was 54; there were 23 females and 22 males. At the study entry, whole blood count, PT, aPTT, fibrinogen, platelet aggregation studies by luminesance method in whole blood and by optical method in PRP, ristocetin cofactor activity were performed. The agonists used were; ADP, Arachidonic acid (AA), Ristocetin and Collagen. Platelets were considered to be hyperactive if at least one result (aggregation or ATP release with one agonist) was above the reference range, and hyporactive if at least one result (aggregation or ATP release with one agonist) was below the reference range. Mixed hypo- and hyperactive platelets were considered present when at least one result (aggregation or ATP release) was below and above the reference range, respectively. Repeat platelet function studies were performed in 20 patients, following specific therapy regimes. By luminesance method; before therapy 15/45 patients had platelet hyperfunction, 17/45 patients had coexistence of hyper- and hypofunction and 12/45 patients had platelet hypofunction. 1/45 patient had a normal result. After therapy 13/20 patients had platelet hypofunction, 2/20 patients had platelet hyperfunction, 2/20 patients had coexistence of hyper- and hypofunction while 3/20 patients had normal results. By optical method; before therapy 18/45 patients had platelet hypofunction, 9/45 patients had platelet hyper- and hypofunction, 7/45 patients had platelet hyperfunction whilst 11 had normal results. After therapy 15/20 patients had coexistence of hyper- and hypofunction, 4/20 patients had platelet hyperfunction, 1/20 patients had platelet hypofunction while none of the patients had normal results. We conclude that; 1. Different platelet function defects are observed in most of patients with MPD 2. Patients with CML have platelet hypoaggregability while patients with PCRV and ET have platelet hypoaggregability. 3. Our observations highlight the need to use WBPA to select patients for antiplatelet therapy in MPD. 4. Luminesance method appears to be more sensitive than optical method to evaluate platelet functions.

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ADIPONECTIN ADDED INTO THE PLASMA OF HEALTHY PROBANDS DOES NOT AFFECT PLATELET AGGREGABILITY

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Background. Adiponectin exhibits important antidiabetic and antiatherogenic effects. Although hypoadiponectinemia is associated with obesity-related metabolic and vascular diseases, the role of adiponectin in thrombosis remains elusive. Recent paper informed that adiponectin deficiency in adiponectin knockout male mice leads to enhanced trombus formation and platelet aggregation. *Aims*. Evaluace of added adiponectin effect into the plasma in platelet aggregability. *Methods*. 6 healthy nonobese healthy probands were tested. In all of them platelet aggregability and adiponectin values were measured. Human adiponectin (Biovendor; Czech Republic) was added to PRP in different concentrations (100; 75; 50 and 25 ng/l). Than PRP was 5 min incubated and was evaluated induced platelet aggregation using CPG (Analytical Control Systems) at 3 μmol/l as the final concentration of CPG added to PRP with an Apact II platelet aggregometer (Labitec GmbH). Induced aggregation extent was defined by the slope of aggregation curve. *Results.* Adiponectin values had normal distibution in tested group (13,7-15,8 ng/l). Neither of tested probands had significat difference of the slope CPG values, even if 100 ng/l adiponectin concentration was added. *Conclusions*. The present study did not verify hypothesis about the in vitro human hyperadiponectinemia as an antithrombotic factor. Adiponectin concentration about 10 ng/l have similar antithrombotic *in vitro* action as values upper 100 ng/l.

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PRESENTATION OF NEW METHOD FOR ASA RESISTANCE DETECTION

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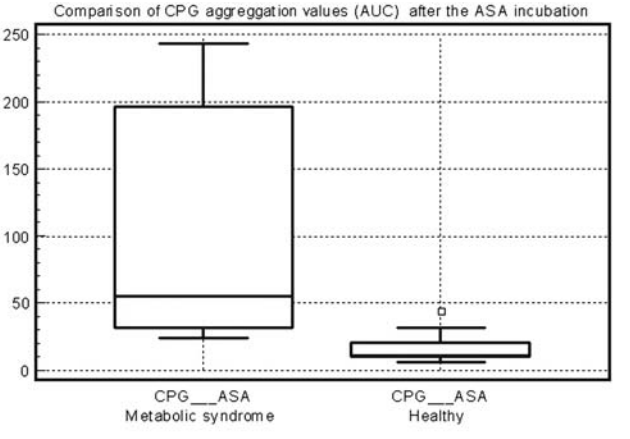
Backgrounds. aspirin resistance seems to be an important prognostic factor in patients with coronary artery disease, but there is limited data on its correlation to clinical outcomes. Various methods for both in vivo and in vitro platelet function exist. In late 1990s, a novel in vitro inductor of platelet aggregation - cationic propyl gallate (CPG) was introduced into clinical practice, announced as an *unprecedented*, highly sensitive and specific method for assessment of aspirin resistence. In classic aggregometry problem with patients compliance remain unresolved. Recently there were present information about chance for ASA resistance testing by virtue of in vitro aggregation test with ASA addition. *Aims.* evaluate platelet ASA resistance with platelet CPG aggregation after ASA addition.



Methods. 20 healthy individuals and 20 patients with metabolic syndrome were evaluated. No individuals were ASA treated. In all of probands was performed platelet aggegometry (Multiplate) after CPG induction. In part of whole blood was supplement solution of ASA (Aspisol, Bayer) and was perform aggregometry, over again. *Results.* healthy probands have higher difference between AUC before and after the ASA pretreatment. (p<0,01, Kruskal Wallis) than probands with metabolic syndrome. CPG have higher difference before collagen (p< 0,05). AUC of aggregometry line in all of healthy probands had significant reaction after Aspisol addition. On the contrary, AUC of patiens with metabolic syndrome reacted different. *Conclusion*: authors presented frequent ASA resistance existence in individuals with metabolic syndrome against healthy. At the same time presented new ijn vitro method for ASA resistance detection which eliminace patient non compliance errors.