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 Platelet count.

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 chronic refractory ITP. Perhaps novel agents like Thrombopoietin Receptor Agonists should be considered for these patients with chronic ITP in the setting of a Clinical Trial.

1428 EFFECTS OF VARIOUS THERAPEUTIC REGIMENS ON PLATELET FUNCTIONS IN PATIENTS WITH MYELOPROLIFERATIVE DISORDERS

M. Akay, E. Akin, Z. Gulbas
Eskisehir Osmangazi University Hospital, ESKISEHIR, Turkey

Blinding 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 hyperfunction, 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 luminescence 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 hyporeactive if at least one result (aggregation or ATP release with one agonist) was below the reference range. Mixed hyper- and hyporeactive 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 luminescence 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 5/20 patients had normal results. By optical method; before therapy 18/45 patients had platelet hyperfunction, 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 can be found in patients with MPD. 2. Patients with CML have platelet hyporegulability while patients with PCRV and ET have platelet hyperfunction. 3. Our observations highlight the need to use WBPA to select patients for antiplatelet therapy in MPD. 4. Luminescence method appears to be more sensitive than optical method to evaluate platelet functions.

1429 ADIPONECTIN ADDED INTO THE PLASMA OF HEALTHY PROBANDS DOES NOT AFFECT PLATELET AGGREGABILITY

J. Proskova, D. Stejskal
Sternberk Hospital, STERNBERK, Czech Republic

Background. Adiponectin exhibits important anti-diabetic and antiatherogenic effects. Although hypo-adiponectinemia 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 thrombus formation and platelet aggregation. Aims. Evaluate 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 (R&D Systems; 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 distribution in tested group (15.7-15.8 ng/l). Neither of tested probands had significant 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.

1430 PRESENTATION OF NEW METHOD FOR ASA RESISTANCE DETECTION

J. Proskova, D. Stejskal
Sternberk Hospital, STERNBERK, Czech Republic

Background. 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 inducer 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 resistance. 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.

Comparison of CPG aggregation values (AUC) after the ASA incubation

Methods. 20 healthy individuals and 20 patients with metabolic syndrome were evaluated. No individuals were ASA treated. In all of probands was performed platelet aggregation (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 patients 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 in vitro method for ASA resistance detection which eliminate patient non compliance errors.