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Ways to Improve the Results of Treatment of Idiopathic Thrombocytopenic Purpura in Adults

Shomuradov Qodir Ergashevich Shomuradov[1], Temirov Nuriddin Najmitdinovich[2], Madasheva Anajan Gazkhanovna[3]
[1]*Ordinator of the department of Hematology Samarkand regional multi-network medical center*
[2]*Ordinator of the department of Hematology Samarkand regional multi-network medical center*
[3]*PhD of the department of Hematology Samarkand State Medical University*

Abstract: ***After long experience of various therapeutic attempts. Treatment of idiopathic thrombocytopenic purpura is prescribed in accordance with the clinical and hematological form, determined through special tests and consists, for curable forms, of corticotherapy, in some cases splenectomy and in special cases immunosuppressive therapy. The first condition for treatment with an accurate indication is the correct diagnosis of idiopathic thrombocytopenic purpura, with the elimination of secondary forms, which, along with the treatment of purpuric syndrome, require treatment of an etiological nature.***
Keywords: thrombocytopenic purpura, adults, innpvative treatment methods, idiopathic thrombocytopenic, cause and effects.

I. INTRODUCTION

Corticoid therapy for idiopathic thrombocytopenic purpura has been experimented for a long time. In large groups of patients who were not treated with prednisone, the evolution was good in 85% of cases, and in those treated with prednisone, the evolution was similar to the first, with no statistical difference between the groups (Choi and McClure, McClure). If purpura persists, as often happens in adults, corticotherapy is prescribed in variable doses. High doses - 1.5-2 mg/kg body weight are indicated only in cases resistant to low doses, when platelets are less than 20,000. We must not forget that long-term treatment with high doses can reduce medullary thrombocytopoiesis (Shulmann). For forms that do not resolve spontaneously within 3-4 weeks, but the platelet count varies between 25-50,000 or more, doses of 0.5-1 mg/kg body weight should be used, which should be suspended when the platelet count approaches 100,000 In general, 60% of patients respond to doses of 1-1.5 mg/kg body weight or less, and only 10% require higher doses (Baldini). According to McClure, in 75% of patients thrombocytopenia persists after 3 months of evolution, requiring continued corticotherapy in high doses. Half of them do not go into remission, so it is necessary to resort to splenectomy or other treatment.

II. LITERARY REVIEW AND METHODOLOGY

The effect of cortisone appears to be rather symptomatic, favoring capillary stability and thus shortening the evolution of purpura, even if the platelet count does not increase at the same rate. However, it is recognized that platelet lysis decreases and the number of platelets increases due to the action of cortisone, its property of inhibiting thrombocytophagy in the hepatosplenic macrophage system. Harrington's studies showed, however, that the effect decreases over time, with thrombocytophagia reduced, as well as antibody titers due to the immunosuppressive, lymphocytotoxic effect of cortisone. ACTH, recommended in some resistant cases, does not seem to have a better effect in any of the phases of severe evolution. Consequently, cortisone treatment has a limited indication in young adults, when spontaneous remission occurs in most cases. In an adult, especially after 50 years of age, the evolution of acute forms is more severe, hemorrhages are more severe, and the risk of chronicity, according to some statistics, is 25-35%. In these cases, corticotherapy in high doses is necessary. However, it should be borne in mind that many of the forms in adults, with chronic evolution, can occur due to sensitization to medications and less in the form of primary forms of idiopathic thrombocytopenic purpura. In chronic forms, in adults and especially in women, purpura prolongs but becomes less severe. If the purpura is mild and only cutaneous, patients can lead an almost normal life with a minimum platelet count varying between 30,000 and 70,000. Corticotherapy is prescribed with the aim of cure in cases with severe relapses, with increasing doses and continuing treatment for 2-3 months. If cure is not achieved, splenectomy should be recommended. If the latter poses some risk or there are other pathological conditions that contraindicate it, the dose can be reduced within certain limits in order to maintain the optimal platelet count. Combining hormonal treatment in a woman with the use of testosterone in particular can lead to a cure or significant improvement.

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III. DISCUSSION AND RESULTS

For example: A pregnant woman with idiopathic thrombocytopenic purpura is prescribed corticotherapy, especially during the last third of pregnancy, when the purpura becomes worse. Chronic forms are better tolerated, while acute forms occurring during pregnancy can be severe and require high doses of cortisone. All authors agree that idiopathic thrombocytopenic purpura, like corticotherapy, does not aggravate pregnancy for the mother; however, 60% of children are born with purpura, and up to 10% of them die. It is likely that treatment with cortisone in the last 3 months, due to the reduction of antibodies, improves purpura in the newborn or even prevents its occurrence altogether (Baldini, Schenker et al.). Despite the fact that the pregnancy generally evolves normally, intrauterine bleeding still occurs during the last three months with premature rejection of the placenta and premature birth. In those cases that are not affected by corticotherapy, platelet transfusions should be performed until postpartum hemorrhage stops. In the treatment of idiopathic thrombocytopenic purpura, platelet transfusion is necessary only for serious hemorrhagic complications and especially for intracranial hemorrhages. It is not known to what extent their evolution and prevention of severe platelet depletion is influenced by very high doses of cortisone and platelet transfusions. It is known that in the acute form in which hemorrhages occur, the life of transfused platelets lasts only a few hours, so their effect is very fleeting. Platelet transfusions can have a good effect in chronic forms that are resistant to corticotherapy, and when patients need to be prepared for splenectomy. The number of transfused platelets must be calculated depending on the number of platelets present in the patient at the time of perfusion, in order to obtain an increase of up to 30-50,000 platelets. For this purpose, concentrated fresh platelets taken from 2-6 units of blood are recommended. In practice, this is only recommended for splenectomy 1-2 hours before surgery. Viability and hemostatic effect are maintained only for platelets taken at most 24 hours before use (Baldini et al.). The use of platelets preserved after freezing is still being studied and is likely to yield good results (Iercan et al.). Splenectomy is considered a specific therapy for idiopathic thrombocytopenic purpura, along with corticotherapy and immunosuppression; it is, as it were, an intermediate method between the first two. When indicated, the results of splenectomy are good in 80–90% of cases, but there is considerable variation in different groups of patients followed over the past 25 years (Dameshek et al., Gardner). Apparently a high percentage of good results - up to 85% - (McGlure) is obtained through splenectomies in children with acute forms, but it is likely that cures could be obtained with the help of long-term corticotherapy; in an adult with a predominance of chronic forms, good results in recent years do not exceed 65%, that is, as it was 30 years ago (Harrington). However, even in children with chronic forms, according to other statistics, cure rates did not exceed 60% (Chioi and McGlure). In adults over 50 years of age, remission after splenectomy does not exceed 50% of cases (Aster). Our experience in more than 20 cases of idiopathic thrombocytopenic purpura has shown that properly indicated splenectomy gives complete or partial remissions in 85% of cases (Butoianu et al.). Undoubtedly good results are obtained in patients who respond to cortisone, but are dependent on this medication, so that there is an indication for splenectomy.

According to our experience and from the literature, it follows that splenectomy is indicated for forms with relapses or for chronic forms that are resistant to treatment with cortisone in high doses - 1.5-2 mg/kg gel weight, for 3-6 months. Unavoidable intervention can be carried out under the protection of high doses of cortisone at the moment when the platelet count is 20-25,000; in cases with visceral bleeding, the dose of cortisone is increased and platelet perfusion is performed 1-2 hours before the intervention; perfusion is repeated after the intervention if a rapid and immediate increase is not achieved. With good preoperative preparation, mortality does not exceed 1% of operated patients. As we have already indicated, it is possible to predict a good effect of splenectomy in cases with maximum sequestration in the spleen, although there are indications for the opposite results: maximum sequestration in the liver and still good effects after splenectomy (Najean et al.). Removal of the spleen also has the effect of biological immunosuppression, since the main source of autoagglutinin production is eliminated. However, the relationship between the effect of splenectomy and antibody titer has not been confirmed. In any case, the intensity of sequestration in the liver or spleen is not a more important criterion for the indication of splenectomy (Aster and Keep, Baldini, Najean and Ardailan). One criterion for predicting the outcome of splenectomies is the immediate response after splenectomies. If within 1-3 days platelets reach high numbers, more than 500,000, they will remain within normal limits, with clinical cures in 84% of cases (Orringer, Najean and Ardailan). If the increase reaches only 100-200,000, cures are obtained in only 25% of cases, and in 85% of cases, platelets drop a few weeks later to the previous numbers and the purple appearance resumes. The pathogenetic hypothesis that splenectomy removes the maximum focus of sequestration (Shulmann et al., Aster and Keena, Najean and Ardailan) and antibody production (McMillan et al., Karpatkin et al.) is not supported by the inconsistent results of splenectomy.

Antibodies disappear in some cases studied, but often they persist and platelets remain low, and in some cases rise to normal values (Berceanu et al.). Despite this, experience has shown that patients who do not fully remit after splenectomy require lower doses of cortisone and cytotoxic drugs. In some of the cases we studied, corticotherapy continuously applied in case of failure after splenectomy led to a number of definitive cures.

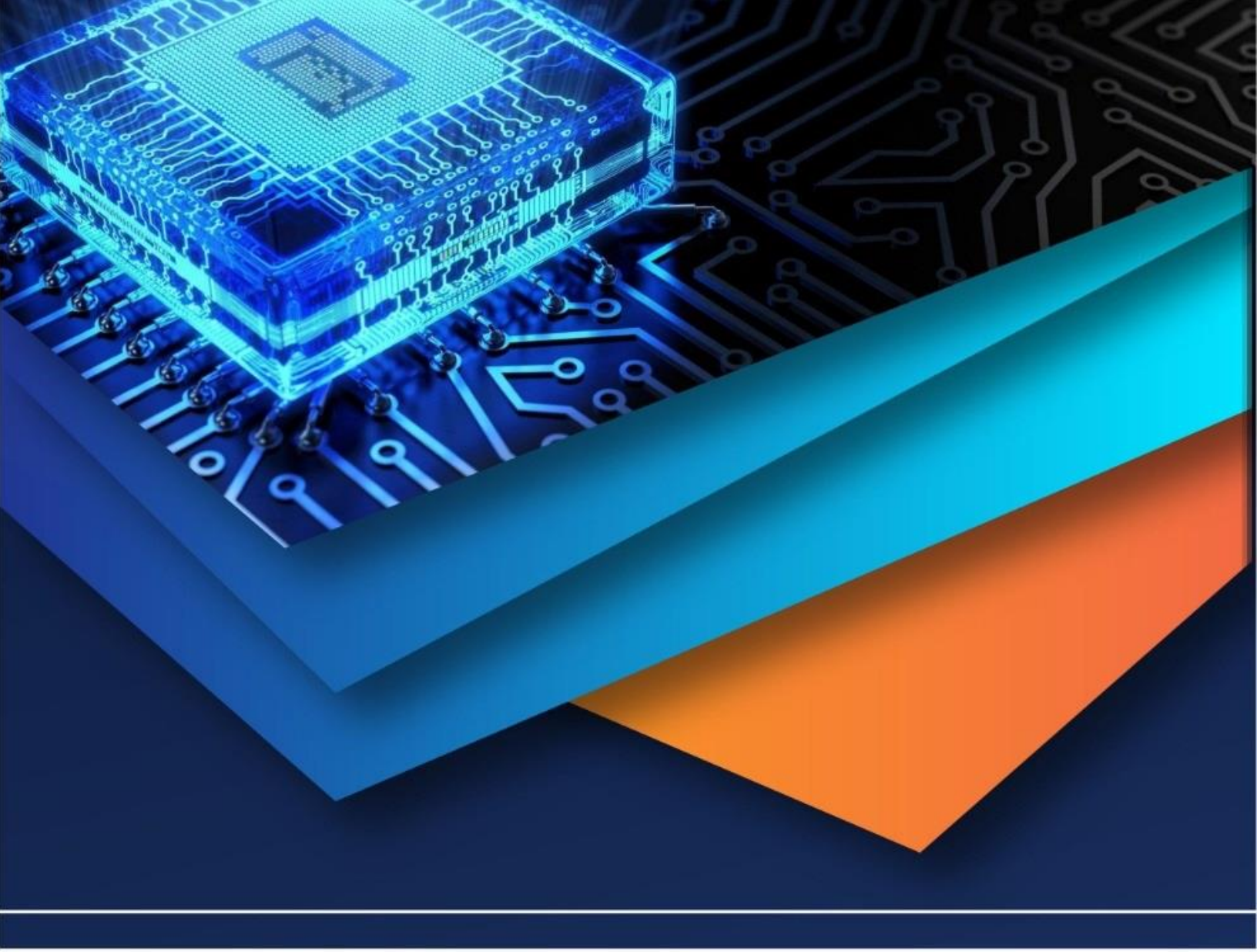
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However, it is unknown if these cures would not have occurred without splenectomy. Indications for therapy with immunosuppressive medications should be made only in cases of extreme necessity, only in cases where splenectomy has failed and when the evolution of a severe chronic form persists. As stated earlier, corticotherapy for idiopathic thrombocytopenic purpura has an immunosuppressive effect only secondary and indefinitely, without controlling the autoimmune process, which creates the need to use other medications with a firmly proven immunosuppressive effect. Until now, mainly imuran (azothioprine) has been used, which gave results in 65% of cases of chronic forms in adults, but also in children (Goebel et al.). According to others, good results do not exceed 20-30% of cases resistant to corticotherapy or splenectomy, where the indication of immunosuppression is paramount. Other medications with antimetabolic effects were also used, such as 6-methylpurine and thioguanine, which have a pharmacodynamic effect close to that of imuran. The doses used are known, respectively 2.5-3 mg/kg body weight for 6 MP and imuran and somewhat less, 2 mg/kg body weight for thioguanine. Attempted treatment with endoxan, usually 50–100 mg orally, produces approximately the same or better results (Finch et al.), but with more significant secondary toxic effects. As with any immunosuppressive therapy, treatment should be continued for several months and stopped when platelets approach normal levels. In some cortico-dependent cases, the use of an immunosuppressive drug leads to a reduction in the dose of cortisone in patients treated for a long time with this drug and showing clear signs of therapeutic hypercortisolism. There is no extensive experience with the use of actinomycin C, which in a small number of cases appears to have produced spectacular results (Baldini). However, very important secondary toxic effects limit the indications of this drug (Martin et al.). Recently, Vinca rosea derivatives, vinblastine and especially vincristine have entered the treatment of resistant forms of idiopathic thrombocytopenic purpura (Marmont et al., Harrington). They are used in slightly lower doses than for malignant diseases, namely 1/20 mg/kg body weight of vinblastine and 0.5 mg/m2 of vincristine every 2 or even 3-4 weeks. In some cases with long-term resistance to other methods, complete or even partial remissions have been obtained, with a reduction in platelets and complete disappearance of clinical signs of purpura. If after 2-3 injections no signs of remission appear, these medications should be discontinued. The pharmacodynamic action determining platelet growth is a topic of debate. It is likely that both medications have an immunosuppressive effect, but vincristine appears to have an additional stimulating effect on central thrombocytopoiesis. Bearing in mind the good results we obtained in cases of dysglobulinemic purpura, we should recognize the immunosuppressive effect affecting inflammatory lesions of the vascular wall.

Besides these three main treatments, which, when properly sequenced and properly applied, produce effective results in most forms of idiopathic thrombocytopenic purpura, in some cases resistant to all these methods, other treatments have been attempted and then rejected. For example, treatment with heparin or dicoumarol (Cohen), accepted on the hypothesis that it reduces the effect of antigen-antibody reactions, especially due to the anticomplementary action of hemarin, was rejected, especially since cases of thrombocytopenia after heparin therapy are known (Natelson et al., Fratantoni et al. .); anticoagulation therapy may have some effects in cases where the process of disseminated intravascular thrombocytopenic coagulation occurs.

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