Comparative Study of Different Regimens in Treatment of Immune Thrombocytopenic Purpura in Children

Thesis

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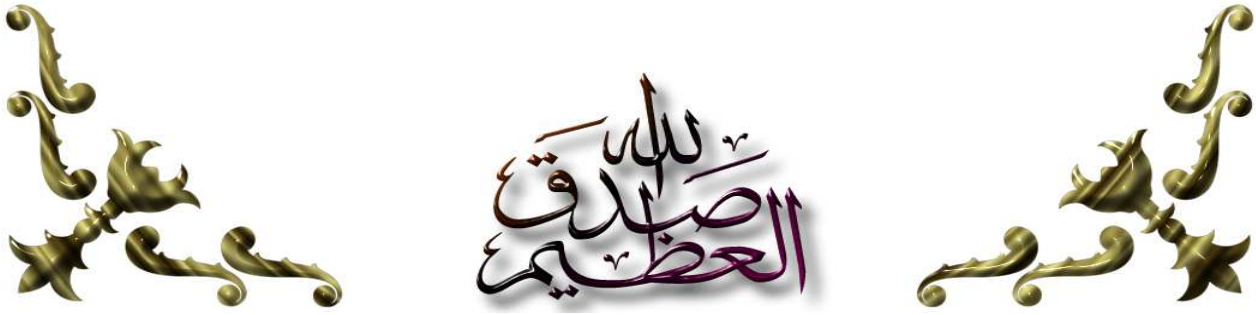
وَقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ
عَمَلَكُمْ وَرَسُولُهُ وَالْمُؤْمِنُونَ



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List of Abbreviations

- 111-I Indium-111
- AA Arachidonic acid
- ACTH Adrenocorticotropic hormone
- ADP Adenosine diphosphate
- ANA Antinuclear antibody
- ATP Adenosine triphosphate
- BCSH British Committee for Standards in Hematology
- B-TG B-thromboglubin
- C3b Complement 3b
- Ca Calcium
- CD Cluster of differentiation
- cITP - csITP Chronic ITP, chronic severe ITP
- CNS Central nervous system
- CR Complete response
- CSF Cerebrospinal fluid
- CVI Common variable immunodefficiency
- EDTA Ethylenediaminetetraacetic acid
- Epi Epinephrine
- GM-CSF Granulocyte-macrophage-colony stimulating factor
- GP Glycoprotien
- HDMP High dose methyl prednisolone
- HELLP Hemolysis,elevated liver enzymes, low platelets
- HIV Human immunodeficiency virus
- HUS Hemolytic uremic syndrome
- ICH Intracranial hemorrhage
- IG Immunoglobulin
- IL Interleukin
- INF Interferon
- ITAM Immunoreceptor tyrosin-based-activation motifs
- ITIM Immunoreceptor tyrosin-based-inhibitory motifs
- ITP Immune thrombocytopenic purpura

- IVIG Intravenous immunoglobulin
- LAT Lymphotoxin
- LDH Lactate dehydrgenase
- MHA Microangiopathic hemolytic anemia
- MMR Measles, mumps, rubella
- NR No response
- PAF Platelet activation factor
- PDGF Platelet-drived growth factor
- PF platelet factor
- PG Prostaglandine
- PL1A Platelet antigen 1A
- PLT Platelet
- PR Partial response
- PRP Platelet rich plasma
- PTP Post transfusion purpura
- RES Reticuloendothelial system
- SLE Systemic lupus erythrematosis
- TG-b Transforming growth factor b
- TH T-helper cell
- TMA Thrombotic microangiopathies
- TNF Tumor necrotic factor
- TPO Thrombopoietin
- TR T –regulatory cell
- TTP Thrombotic thrombocytopenic purpura
- TXA2 Thromboxane A2
- vW von Willebrand's factor

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Introduction

Introduction

Immune thrombocytopenic purpura (ITP) in children is usually a self limiting disorder presenting most commonly with a short history of purpura and bruising in children of either sex between the ages of 2 and 10 years of age. The incidence is at about 4 per 100 000 children per year. It may follow a viral infection or immunization and is caused by an inappropriate response of the immune system. Autoantibodies against platelet surface glycoproteins (particularly IIb/IIIa) can commonly be detected (60-70%), but are of no prognostic significance and this is not a useful diagnostic test (**Maggs et al, 2001).**

A wide range of therapeutic regimens are currently in use, including observation alone, as the majority of children recover within 4-6 months regardless of treatment. A growing understanding of the pathophysiology of acute ITP in children has not solved the controversy of treatment, but has clarified the mechanism of action of the most frequently used agents in chronic ITP **(Nugent et al, 2006).**Currently, there is no single optimal management for the child who is newly diagnosed with ITP. Most hematologists in the United States choose to treat a child with a platelet count $<10{,}000X10^3/\mu L$ or with mucous membrane bleeding. However, observation and education are appropriate for the child with mild thrombocytopenia and no clinical bleeding. Initial treatment options for childhood ITP include IVIG, anti-Rh immunoglobulin (“anti-D”), steroids (oral or IV), or combination therapy **(Beardsley et al, 2006)**.

About 20% of the children diagnosed with acute ITP will run a chronic course. Only in a minority of these, platelet-count-enhancing