About the Editor

Abdulgabar Salama

Abdulgabar Salama is Head of the Institute of Transfusion Medicine, Charité University Medicine Berlin, Germany. His medical specialty is internal medicine and immune hematology. He treats patients with immune thrombocytopenia and/or autoimmune hemolytic anemia since 1980. He has published over 200 original original papers on this field. He also introduced anti-D for treatment of immune thrombocytopenia in 1983.

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Foreword

Addressing current treatment options for autoimmune thrombocytopenia

Abdulgabar Salama

There is no doubt that autoimmune thrombocytopenia (ITP) is an acquired autoimmune disease that affects approximately 5% of the world's population. This disorder may occur at any age, with and without the coexistence of predisposing factors, that is, evident or inapparent infections, immune defects and other immune abnormalities. ITP represents the best characterized human autoimmune disease. From a clinical perspective, it is defined by a low platelet count and increased bleeding risk, with therapeutic effects objectively measured within a short amount of time.

However, based on recent studies, it is now clear that ITP, similar to other autoimmune diseases, is also multifactorial and heterogeneous. The etiology of ITP, particularly in adults, is largely unknown; the clinical course is unpredictable and variable. In addition, there remains no test that can confirm the diagnosis of ITP. Finally, numerous drugs are now available for the treatment of ITP, but at times there remains doubts whether any treatment is necessary and, if so, which therapy best suits the patient.

In an effort to update understanding and knowledge on ITP, and to determine the accurate assessment of diagnostic and therapeutic strategies, the authors involved in this book have summarized and commented on the most important published data in this field, including guidelines.

The significance of an impaired platelet production and T-cell-mediated platelet destruction in ITP are incorporated. It is also clearly demonstrated

that conventional treatment for ITP predominantly aims at suppressing the production of autoantibodies and/or inhibition of macrophage-mediated platelet destruction. This treatment often remains ineffective and many patients develop severe adverse effects. During the last 20 years, several observations have resulted in the development and use of thrombopoietin receptor agonists. Two of these drugs, romiplostim and eltrombopag, have been shown to increase the production of platelets in a dose-dependent manner. This results in an increase in platelet counts in the circulation of the majority of ITP patients. Both drugs are being increasingly used, and the reported adverse effects seem yet to be rather tolerable in most cases. However, these drugs are not curative and continuously stimulate the production of platelets. Therefore, the concerns regarding long-term side effects remain considerable.

In conclusion, in addition to some recommendations provided by the authors, many important questions remain unanswered. These include the failure to detect autoantibodies in 50% of ITP patients that might be related to a limited sensitivity of applied tests, unidentified antigens or low-avidity autoantibodies. It is unknown whether any biomarkers may help in determining the diagnosis and therapy of ITP. The need for specific therapy remains quite obvious. This can only be achieved by clarifying and halting the true mechanisms involved in the development of ITP. Can we justify the use of immune steroids and/or other suppressive drugs for the treatment of ITP anymore? It must be emphasized that guidelines only represent the viewpoints of a certain number of people, and some important aspects cannot be sufficiently covered by any set of guidelines, and ultimately, independent clinical trials are invariably essential prior to drawing a final conclusion about the use of any drug.

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A Salama has participated in Advisory Boards for Amgen and GlaxoSmithKline. He is involved as a principle investigator in clinical studies dealing with eltrombopag (GlaxoSmithKline), Nplate® (Amgen) and IVIgG (Biotest, Octapharma, and LFB Biotechnology). He has received accommodation expenses from Biotest, Octapharma, CSL Behring and Intersero for scientific meetings. The author has no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

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