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A REVIEW: THROMBOCYTOSIS

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Abstract: Thrombocytosis is a clinical condition that is frequently seen, with a high percentage of instances being found by chance. The diagnosis of thrombocytosis might be difficult due to the wide range of possible diagnoses. Thrombocytosis may be fake, the result of a reaction, or the result of a clonal problem. This distinction is significant because it has effects on assessment, prognosis, and treatment. The myeloproliferativeneoplasms, particularly essential thrombocythemia and polycythemia vera, are linked with clonal thrombocytosis, which has a distinct prognosis profile and a significantly elevated risk of thrombosis. The motivation behind these illnesses' treatment plans is this risk. New therapeutic targets are awaiting investigation in clinical studies using targeted treatments for thrombocytosis.[1]

Keywords: thrombocythemia, polycythemia, Thrombosis

I. INTRODUCTION

Blood platelets, a component generated in the bone marrow, are essential to the process of blood clotting. Adults and children should have platelet counts between 150,000 and 450,000 microL (150 and 450 x 10/L),while the normal range may change between clinical laboratories. [2]

A condition known as thrombocytosis occurs when the platelet count is higher than 450,000/l.[3] It is additionally known as thrombocythemia.

Primary and secondary (or reactive) thrombocytosis are the two categories under which thrombocytosis canbe categorised. [4]

Thrombocytosis Caused by Mutations in the Thrombopoietin Receptor:

The thrombopoietin receptor (MPL) was identified as a multiline age myeloproliferative leukemia- causing retroviral oncogene (v-mpl). [5]

In cell lines that were tumorigenic when injected into mice, a mutation intended to dimerize the extracellular domain of mouse Mpl protein (Mpl-S386C) by allowing thecreation of a disulfide bond between two receptor molecules resulted in constitutive activation of signalling.[6]

These findings suggested that people with MPN may have MPL mutations. When a small sample of patients were first investigated, no abnormalities were discovered. In a family with autosomal dominant thrombocytosis, a serine at position 505 of the MPL protein was changed to an asparagine (MPL-S505N). This was the first MPL mutation . [7]

Risk Factors for Thrombosis:

Age is a major risk factor for thrombosis in the general population, and numerous epidemiologic studies have revealed an increase in risk for PV and ET thrombosis with ageing. Prior thrombotic events have been repeatedly shown to increase the risk of thrombosis in patients with PV and ET.[8] With a thrombosis rate inthe European Collaboration on Low-Dose Aspirin in Polycythemia Vera (ECLAP) trial of 10.9 events/100 persons/year compared to 2.5 events/100 persons/year in those without either risk factor, age >65 and a priorthrombotic event are significantly associated with an increased risk of thrombosis in PV.[9]

Increasing platelet count has not consistently been linked to an increased risk of thrombotic events, unlike the danger associated with leukocytosis. Although 80% of thrombotic events happen in those with a platelet count >600 109/L, numerous studies have been unable to demonstrate a direct association between rising platelet count and thrombotic risk.[10] It has been demonstrated that cytoreductive therapy that resultsin lower platelet counts reduces microcirculatory vasomotor symptoms and thrombosis, although it is unclear whether this is due only to lower platelet counts or to additional side effects of cytoreductive therapy, such as a lower leukocyte count.[11]

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Treatment of Thrombocytosis:

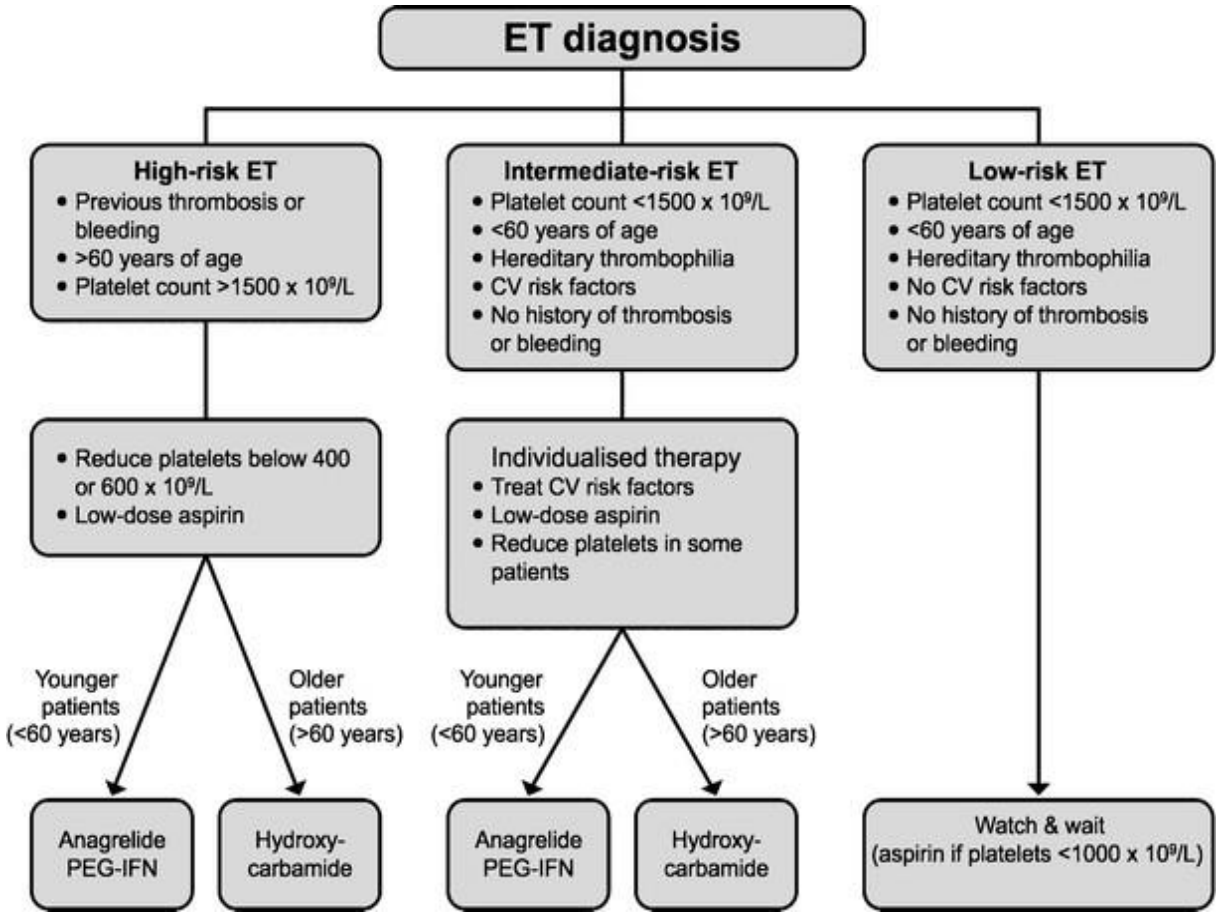
ET is a persistent disease with a protracted clinical history that is now incurable. Therefore, managementfocuses on preventing major side effects, particularly thrombohaemorrhagic events, by lowering platelet counts or changing platelet function.[12]

Although the precise definition varies by investigator and country, management is categorised as high, middle, and low risk. Other less conventional risk variables have also been proposed that may affect treatment choices, in addition to typical risk factors including age, past thrombosis or significant bleeding, and increased platelet count. [13]

They consist of molecular indicators (such as the existence of a PRV-1 or JAK2 mutation) and cardiovascular risk factors. It is still unclear if the JAKV2617F mutation should be taken into account when determining risk. As previously indicated, reactivethrombocytosis is thought to be self-limited with little additional associated thrombotic risk. [14]

Even for significant thrombocytosis, no antiplatelet medication is advised due to this absence of thrombotic risk and a potential danger of paradoxical bleeding. When reactive thrombocytosis is suspected in a patient, the development of thrombosis may be cause for further investigation of a concurrent clonal thrombocytosis, especially when splanchnic or cerebral thrombosis is present.[15]

A suggested treatment protocol is shown in Fig. 1 and takes into account the clinical experience and data that are currently available for various ET treatments. High-risk ET patients are thought to have the following risk factors: A very high platelet count (>1,500 109/l), an older age than 60 years, or a history ofsignificant bleeding or thrombosis. Patients with ET who don't meet the high-risk criteria but have genetic thrombophilia or cardiovascular risk factors are categorised as intermediate-risk ET patients. All of these risk variables are absent in low-risk ET patients.



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II. CONCLUSION

Thrombocytosis can occur in a variety of clinical settings and may result from a wide range of underlying aetiologies. Clonal thrombocytosis is frequently brought on by one of the Ph- MPNs, and in this situation, the risk of thrombosis should be the main element dictating the course of treatment. The processes underlying this elevated thrombotic risk are still poorly understood, and it is also unclear what role risk variables like leukocytosis and JAK2 status play. A risk-based treatment decision model that incorporates these and other risk indicators should make it possible to choose patients for cytoreductive therapy more effectively. Clonal thrombocytosis diagnosis and treatment should advance as novel molecular anomalies inthese diseases are continually found and described.

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