THROMBOCYTOSIS IN A PATIENT WITH SCHIZOPHRENIA: ESSENTIAL THROMBOCYTHEMIA OR CLOZAPINE-INDUCED THROMBOCYTOSIS?

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Introduction

Essential thrombocythemia (ET) is characterized by thrombocytosis and bone marrow megakaryocytic hyperplasia associated with a high risk of bleeding, vasomotor symptoms, and thrombosis. ET has traditionally been a diagnosis of exclusion, requiring the absence of reactive conditions and other myeloproliferative and myelodysplastic syndromes.[1,2] On the other hand, clozapine is known to cause blood dyscrasias, typically neutropenia and agranulocytosis. Likewise, some cases of clozapine-induced thrombocytosis have also been reported.[3,4]

Methods

We perform a case study research design.

Results

We show a study case of a 37-year-old man diagnosed by paranoid schizophrenia who presented an acute ST-elevation myocardial infarction who required percutaneous coronary intervention with stent implantation on the anterior descendant coronary. Subsequent performance of a blood test revealed a platelet account above 530 × 10^9/L (normal range: 150-450 × 10^9/L) which confirmed the finding of a thrombocytosis with no alterations in non-specific inflammatory parameters, including a raised white cell count, erythrocyte sedimentation rate and C-reactive protein.

To make a confirmation diagnosis of a myeloid neoplasm, we performed the JAK2 mutation test which resulted positive for JAK2 V617F variant status. It's known that the combination of an isolated thrombocytosis with this pathogenetic mutation, in the absence of iron deficiency (which may mask polycythemia vera) or features of primary myelofibrosis is considered sufficient to make a diagnosis of ET.

500 mg per day of hydroxyurea (this one is the only cytoreductive agent proven to reduce thrombotic events in an ET patients) and 100 mg per day of acetylsalicylic acid were prescribed; shortly after the platelet accounts significantly decreased to normal. The patient remains asymptomatic until now, without cardiovascular complications.
Interestingly, this clinical picture concurred with the onset shortly before of clozapine treatment reaching a dose of 100 mg twice daily.

Conclusion

It is well known that clozapine causes hematological side effects such as agranulocytosis, neutropenia, and leukocytosis. But the results about the effects of clozapine on the number of platelets were not consistent. A raised platelet count, with clozapine as the sole implicated agent, had been reported previously.

As we know, ET has traditionally been a diagnosis of exclusion, requiring the absence of reactive conditions. We hypothesize about the possible relation of the treatment with clozapine as a possible reactive condition to the presence of thrombocytosis in this patient.

Finally, although thrombocytopenia or agranulocytosis are regarded as more clinically important side effects, thrombocytosis should be monitored in a patient under clozapine treatment because it may be related with increased the risk of thrombosis and pulmonary embolism.

References

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