EUSEM 2018 GLASGOW, 8-12 SEPTEMBER
THE EUROPEAN EMERGENCY MEDICINE CONGRESS
EUSEM EUROPEAN SOCIETY FOR EMERGENCY MEDICINE

A case of vermiculate tongue

Phoolchund A[1], Singh D[1]
[1]St Mary's Hospital, Isle of Wight NHS Trust, Newport, UK

Isle of Wight NHS
NHS Trust

Background

Unexplained thrombocytopenia is a common clinical problem, and the possibility of drug-induced thrombocytopenia (DITP) must be considered, especially in hospitalized patients.

DITP is a form of secondary ITP where the thrombocytopenia is caused by drug-dependent antibody-mediated platelet destruction; where the drug could be a prescribed medicine or its metabolite, herbal supplement, food, beverage or other substance.

We present a case report where a rather commonly used medicine induced DITP with a nadir platelet count of <20,000/microliter and cutaneous ecchymosis needing hospitalisation.

Case Report

A 65 year old female presented to emergency department with a one-week history of insidious onset multiple skin bruises without any other symptoms.

On examination she had multiple cutaneous ecchymosis, petechiae, purpura all over body but no obvious evidence of mucosal bleeding.

Rest of systemic examination was unremarkable especially absence of fever, hepato-splenomegaly, lymphadenopathy, neurological findings or features of connective-tissue disorder. Investigations revealed a platelet count of 3,000/microliter (normal-range 140,000-400,000/microliter).

On direct questioning there was no history of recent viral infection or flu-like symptoms, no consumption of quinine-containing beverages like tonic water, no high-risk behaviour and no excessive alcohol uptake. There was no family history of platelet disorders.

After discussion with the haematologists, thrombocytopenia was confirmed on peripheral blood smear and repeating her platelet counts to rule out pseudo-thrombocytopenia, inherited platelet disorder or thrombotic thrombocytopenic purpura (TTP) or any features suggestive of myelodysplasia.

Her last platelet count was confirmed to be normal 2 weeks before this presentation.

During medicine reconciliation on admission, it was noted that she was started on frusemide one week ago by her general-physician for pedal oedema as presumptive feature of clinical fluid overload.

After excluding common differentials and a medication history suggestive of recent exposure to frusemide, cutaneous findings with absence of other clinical features; a diagnosis of probable Frusemide induced immune thrombocytopenia was made.

Rest multitude serological investigations were negative for infective hepatitis, retroviral infection, coagulation disorder, thyroid-dysfunction or nutritional deficiencies (B12/folate).

Her frusemide was discontinued. Empirically treatment was started as initially DITP cannot be distinguished from Idiopathic-Immune-Thrombocytopenia (ITP). Oral steroids (using 1mg/kg of prednisolone) and intra-venous-immune-globulin (IVIG) was administered.

After reassuring investigation reports, the steroids were later tapered and patient's platelet count recovered within 2 days of drug discontinuation and returned to a normal range at day 6.

She made a full recovery with tapering oral-corticosteroids; and complete symptomatic resolution with no recurrence of her symptoms at one–month follow-up. Her medical healthcare records were updated to record this association to avoid future inadvertent administration of frusemide and she was referred to cardiologist for formal assessment of fluid overload.

Conclusion

Our case report reiterates that medical community should be reminded about this important reversible association where a commonly used diuretic frusemide can induce DITP. Although frusemide induced DITP is reported in literature, this case report re-emphasises the prescriber to deeply bear in mind that the bleeding risk and mortality rates are reported to be greater with DITP than with primary ITP. Early recognition, drug discontinuation, specialist involvement can result in favourable clinical outcome.