



Leech induced nasal bleeding with thrombocytopenia - A case report

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Abstract

A component of the innate immune system are platelets that interact with viruses, bacteria, and parasites, and in some situations, these interactions result in the loss of platelets. As a result there can be reduced platelet count as well as micro thrombi formation in any parasitic infestation. The most common parasite that has platelet interaction is with that of malarial parasite. This case report is that of a patient who presented with complaints nasal bleeding which turned out to be leech induced nasal bleed as a result of thrombocytopenia.

Keywords: innate immune system, parasitic infestation, leech induced nasal bleed

Introduction

Leeches are segmented parasites they are muscular, soft and segmented body that can lengthen and contract. They have suckers at both the ends with a spacious body cavity. Leeches are found in freshwater while other are habitants of terrestrial and marine environments. Leech bites are generally alarming rather than dangerous with local anaphylactic reactions to the leech bite.

Case report

A 52 yr. old female presented to Sree Balaji medical college and hospital with complaints of bluish red patch over left arm, bleeding from bilateral nostrils for the past 15 days and bleeding from gums for the past 2 day. She had visited to a nearby hospital for nasal bleeding for

which cauterization was done and was referred to SBMCH. There is no history of fever, arthralgia, retro-orbital pain. There is no h/o of bleeding manifestations, no h/o any constitutional symptoms, No h/o similar illness in the past, No h/o similar illness in family

The patient gives a positive travel history 1 month back. She is a k/c/o of type 2 DM - now on regular treatment with insulin and k/c/o systemic hypertension- diagnosed 1 month and on regular anti-hypertensive medications. On examination she is conscious, oriented, afebrile. Pulse is normal is rate, rhythm and character, BP-120/90mmhg, respiratory rate-18/min. General physical examination-normal. On examination of oral cavity peritodontitis+, petechial lesions (+) In the oral cavity and on hard plate and soft palate, poor oral hygiene. A brown lesion (+) in the right side of the nasal cavity, probably a blood clot. No other bleeding manifestations, no petechiae and ecchymosis. On systemic examination CVS-S1, S2 present, RS-Bilateral air entry +, P/A- soft, non-tender, CNS- no focal neuro logical deficit

From the above-mentioned examination finding a differential diagnosis of bleeding disorder secondary to Thrombocytopenia was made and to identify the Cause for further treatment. The patient came with the above-mentioned complaints, all necessary baseline investigations done, (mentioned in table 1), Renal function test and Liver function test (Table 2&3). I/v/o reduced platelet count Peripheral smear done showed relative neutrophilia with Thrombocytopenia. Viral serology - non reactive. ECG done normal sinus rhythm, chest X-ray done-normal study. USG abdomen done - normal study.

Table 1

DATE	19/3/2021	20/3/2021	21/3/2021
Hb	14.0	14.1	13.6
TC	6400	5700	13900
DC	P43L40M17	P49L35M16	P88L8M4
PLATELET	30000	6000	33000
ESR	15		
T. B		0.9	
D.B		0.4	
SGOT		31	
SGPT		38	
ALP		121	

Table 2

RBS	251
B. UREA	22
S.CR	1.1
S. Na	137
S. K	3.6

Based on the investigations above I/v/o low platelet count multiple platelet transfusion was done but the cause was unknown. Inj. Methyl prednisolone 1gm IV OD, T. Nicardia 20mg OD, Inj. Plain insulin was given to the patient. During the course of stay in the hospital on D3 of admission the patient developed epistaxis for which ENT opinion was obtained. Incidentally a leech was found in her nostrils when nasal packing was done for epistaxis. Repeat investigations was done values mentioned below. Bone marrow biopsy was done which

showed normal trilineage hematopoiesis. She is clinically and symptomatically better, labs values was on a increasing trend (TABLE 4). Hence the patient was discharged.

DATE	25/3/2021	28/3/2021
Hb	13.9	13.0
TC	9200	9000
DC	P84L11M5	P60L39M1
PLT	1.14 L	2.1 L

Discussion

The above case is leech induced nasal bleeding with thrombocytopenia. Causes of parasitic thrombocytopenia are malaria, schistosomiasis, babesiosis, leech induced. Leech belong to the kingdom – Animalia, Phylum - Annelids, Class - Clitellata, Subclass-Hirudinea. Certain compounds present in saliva of leech interferes with platelet and coagulation pathways. The compounds are mentioned below

Hyaluronidase

- An antibiotic that lowers hyaluronan viscosity (raising tissue permeability) and raises interstitial viscosity.
- The enzyme a gyrase prevents host platelet aggregation.
- Enzymes for wound and burn debridement include proteases.
- Enzymes called lipolytics break down lipids by hydrolyzing triglycerides.
- Fibrin is broken down by destabilase (thrombolytic effects)
- Bdelines: Trypsin, plasmin, and acrosin-inhibiting anti-inflammatory drugs

- Eglines - Anti-inflammatory; block the actions of cathepsin G, elastase, substilisin, alpha-chymotrypsin, and chymase.
- Calin - prevents blood clotting (blocks the binding of van Willebrand factor to collagen). Inhibits platelet aggregation caused by collagen.
- Tryptase inhibitor: Prevents host mast cells' proteolytic enzymes from acting.
- Factor Xa inhibitor – Creates equimolar complexes that suppress the function of coagulation factor Xa.
- Vasodilator: Acetylcholine
- Carboxypeptidase Increases the flow of blood at the bite while using A inhibitors.
- a thrombin inhibitor called hirudin
- A factor Xa inhibitor called antistasin
- Platelet membrane glycoprotein IIb-IIIa antagonist decorsin.



Figure 1



Figure 2



Figure 3

Conclusion

It is obvious that platelets are an essential part of the innate immune system, acting as the body's first line of defense against all pathogens. They appear in full range to pathogens like bacteria, viruses, parasites hence causing Thrombocytopenia. From the clinical case mentioned above where leech-induced Thrombocytopenia are due to certain compounds that are extracted from the leech that interact with the platelet pathways which causes defective platelet coagulation and activation and causes platelet aggregation leading to potential Thrombocytopenia and bleeding.

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