

## Research Article

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## Abnormal Uterine Bleeding due to Coagulopathies (AUB-C)

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**Received:** January 25, 2024; **Accepted:** February 16, 2024; **Published:** February 22, 2024**Introduction**

AUB-C encompasses the spectrum of systemic disorders of hemostasis that may be associated with coagulopathy. 13% of women with HMB have biochemically detectable systemic disorders of hemostasis, most often von Willebrand disease [1]. Inherited abnormalities of hemostasis include Von Willebrand disease, platelet function defects and other rare defects of coagulation. Thrombocytopenia is the most common cause of acquired abnormality [2]. Case series presented to us in 2023 are being discussed.

**Case Series**

|                                    | Case 1   | Case 2  | Case 3  | Case 4  |
|------------------------------------|--|---|---|---|
| Clinical Presentation              | Acute episode of Heavy menstrual bleeding  | For Medical termination of pregnancy  | Dysmenorrhea h/o endometriosis+   | Abdominal pain  |
| Age in Years                       | 23   | 38  | 22  | 27  |
| Marital and Past Obstetric History | Unmarried  | G2P1L1<br>1FTND   | Nullipara<br>Married*3 years  | P2L2<br>2 FTND  |
| Past History at Diagnosis          | *VWD TYPE 3- 7yrs (epistaxis)<br>*H/o multiple blood transfusion for HMB.                                | *GLANZMANN THROMBASTHENIA (gum bleeding) 32 years<br>*h/o multiple blood transfusion for HMB and 2* PPH | *BERNARD SOULIER SYNDROME 16yrs (petechia)<br>*h/o endometriosis multiple episodes of hemoperitoneum- conservatively managed<br>*h/o multiple blood transfusion for HMB and on trivial trauma | *h/o Epistaxis and hematemesis at 5 years<br>*h/o multiple blood transfusion for HMB<br>*h/o Hemoperitoneum 1 year back conservatively managed<br>IMMUNE THROMBOCYTOPENIA diagnosed now |
| Family History                     | 2 brothers VWD type 3  | -   | Sister<br>? VWD   | Sister<br>? VWD   |
| Diagnostic Criteria                | *Von Willebrand factor antigen assay,<br>*Ristocetin factor activity<br>*Factor 8 coagulant activity (3) | *Light transmission aggregometry<br>*Flow cytometry (4)   | *Flow cytometry<br>*Ristocetin induced platelet aggregation test (5)  | *Low platelet count<br>*Peripheral smear- large platelets and tiny platelet fragments<br>*Bone marrow-increased megakaryocytes (6)  |
| Management                         | *Von Willebrand factor replacement<br>*Desmopressin<br>*Anti fibrinolytic (3)                            | *Platelets recombinant activated clotting factor VII<br>*Hormone therapy (4)                            | *Platelet transfusion<br>*Antifibrinolytic therapy<br>*Desmopressin (5)   | *Corticosteroids<br>*Intravenous immunoglobulin<br>*Anti-RHD immune globulin (6)  |
| Management given                   | *3unit PRBC transfused<br>*Supportive measures<br>*Started on progesterone tablets                       | *Evacuation done<br>*2unit PRBC transfused<br>*1 unit SDP transfused                                    | *Hemoperitoneum 1unit PRBC, 3 units PRP, 2 units SDP transfused<br>*Started on OCP  | *Ruptured ovarian hemorrhagic cyst<br>*4unit PRBC, 7 PRP, 2unit FFP transfused<br>*Methylprednisolone 3 doses given   |

## Discussion

VWD is caused by decreased or defective production of VWF protein, involved in platelet function and serves as a stabilizing agent for coagulation [3]. During pregnancy, in antenatal period- levels of VWF:RCO and FVIII:C assessed, during labour- VWF/ FVIII concentrates and tranexamic acid used to control PPH and newborn is screened for the disease[4].

Glanzmann thrombasthenia is caused by a deficiency of the platelet integrin alpha IIB beta3 essential for platelet aggregation and hemostasis [5]. During pregnancy, in antenatal period- test for anti- $\alpha$ IIB $\beta$ 3 and anti-HLA class I antibodies, during labour- anti-fibrinolytic & R FVIIA used to control PPH and newborn is screened for the disease [6].

Bernard-Soulier syndrome is a defect of the GPIB-IX-V complex, a platelet receptor complex that binds with the Von Willebrand factor (VWF). During pregnancy, in antenatal period- test for HLA type, anti-platelet antibodies, during labour- HLA-matched platelets and tranexamic acid kept ready. RFVII can be used, monitored for 8 weeks postpartum for PPH and newborn is screened for the disease and development of FNAIT [7].

Immune thrombocytopenia is an autoimmune pathology caused by antiplatelet antibodies causing increased destruction and inadequate production of platelets [8]. During pregnancy, in antenatal period-CBC monitoring, if PLT low- prednisone/ IVIg, during labour- platelets and tranexamic acid kept ready and newborn has risk of fetal thrombocytopenia and intracranial hemorrhage [9].

Management of Heavy Menstrual bleeding-in a study conducted by Marnach et al, in women treated with IV estrogen for HMB, 72% had controlled bleeding; in women taking oral contraceptive pills (OCPs) 88% had controlled bleeding compared with 76% using medroxyprogesterone acetate. When medical therapies fail for coagulopathies, endometrial ablation or hysterectomy may be warranted after childbearing is completed [10].

## Conclusion

AUB C should be considered in women with heavy, prolonged menses from an early reproductive age with history of frequent bruising, epistaxis, gum/dental bleeding, postpartum haemorrhage and severe surgical bleeding and a family history of these issues. Specific diagnostic modalities to be used to arrive at a diagnosis. Blood and blood products should be arranged in obstetric management. Family members should be screened for the disease.

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