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Case Report

Presumed Passive Immune Thrombocytopaenia in twin infants, secondary to Maternal Autoimmune Disease. A Case Report.

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Abstract

Background: Passive immune thrombocytopaenia (PIT)in infants results following transfer of maternal anti-platelet antibodies trans-placentally or via breastmilk particularly in mothers with autoimmune disorders.
Objectives. This report aims to highlight the possibility of occurrence of PIT beyond the neonatal period and the treatment strategies employed.
Case Presentation: A set of 4-month-old female twins presented with recurrent melaena, nasal and gingival bleeds of 2 months duration, fever of 2 weeks and tachypnoea and generalised weakness of 2 days duration. Both infants were severely pale and laboratory evaluation revealed profound thrombocytopaenia (14 $x10^9$ /l and 8 x 10^9/L respectively) and severe anaemia. Their mother had severe anaemia in the third trimester of pregnancy requiring repeated blood transfusions and following admission of the twins, was further evaluated and a diagnosis of Systemic Lupus Erythematosus made. The twins received fresh whole-blood transfusions, initial treatment with intravenous methylprednisolone with no improvement and subsequently received Intravenous Immunoglobulin which led to a rapid rise in platelet counts and resolution of bleeding.
Discussion: The effect of maternal anti-platelet antibodies in the baby may persist beyond the neonatal period even up till 4 months of age, as in this report. Despite paucity of literature on this as a cause of immune thrombocytopaenia in infants, a high index of suspicion should be maintained.
Conclusion: Persistent, severe or unexplained thrombocytopaenia in an infant should prompt careful review of maternal autoimmune status if previously unknown and antiplatelet antibody investigations. Intravenous Immunoglobulin is very effective in treatment of steroid-refractory passive immune thrombocytopaenia.

Keywords: Case report, Thrombocytopaenia, Infants, Passive Immune thrombocytopaenia, intravenous immunoglobulin.

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INTRODUCTION

Thrombocytopaenia is defined as a platelet count below the age-appropriate reference value (150 × 10^9/L in older children and adults, or 120 x 10^9 /L in extremely small preterms). [1] It may result from decreased platelet synthesis , increased platelet destruction or consumption, sequestration or a combination of these mechanisms and may be immune or nonimmune in origin.[1,2] The differential diagnoses in infants include, immune thrombocytopaenia, congenital and inherited disorders, infection-associated thrombocytopaenia, bone marrow failure or infiltration and consumptive coagulopathies.[2-4]

Immune thrombocytopaenia(ITP) previously called "Idiopathic thrombocytopaenic purpura", is the most common cause of acquired bleeding in childhood,[3] with an annual incidence of 1.9-6.4 per 100,000 children below 15years of age.[5] It is a disorder that occurs usually following Immunoglobulin G sensitisation of platelet membrane proteins, leading to their destruction or removal from circulation by tissue macrophages. It is characterised by low platelet count, haemorrhagic episodes and purpura. Immune thrombocytopaenia may be primary, when aetiology is unknown, or secondary when the immune process is triggered by other conditions like; viral infections, drug reactions, malignancies or autoimmune disorders e.g. Systemic lupus erythematosus and Evans syndrome.[6]

Passive immune thrombocytopaenia (PIT) is an immune thrombocytopaenia in which antiplatelet antibodies formed in a mother, are transferred to the affected infant. This can be seen for instance in neonatal alloimmune thrombocytopaenia transplacentally transferred maternal antiplatelet antibodies recognise only the paternally derived foetal/neonatal platelets as foreign,[7,8] or in mothers with pre-existing immune disorders e.g. ITP, SLE, in which maternal autoantibodies destroy both maternal and foetal platelets. Passive autoimmune thrombocytopaenia is usually a transient disorder[8] presenting commonly within the neonatal period where it manifests early (<72hours of life) or late(> 72 hours of life)[1] with the infant's platelet count normalising within 2 months as maternally derived antibodies decline. [7,9] Persistent passive immune thrombocytopaenia beyond 2 months of age is unusual and its overall incidence is unknown. It has however been described in breastfed infants in association with maternal IgA anti-platelet antibodies transferred via breastmilk,[9] though this mechanism could not be established in this present case.

This report describes the occurrence of persistent severe thrombocytopaenia beyond the neonatal period in twins whose mother was subsequently managed for a presumptive diagnosis of Systemic Lupus Erythematosus. The unusually late presentation and the subsequent response to IVIG, make this case clinically important.

Case Presentation

A set of 4-month-old female twins was brought to the Children Emergency Ward of the University of Port Harcourt Teaching Hospital with recurrent passage of melaena stools and occasional nasal and gingival bleeds of 2months duration, low-grade, intermittent fever of 2weeks, fast breathing, pallor and generalised weakness of 2 days duration. There was no history of prolonged bleeding from the umbilical stump in the neonatal period and no trauma or medication use preceding the onset of symptoms. The babies received oral antibiotics, antimalarials, multivitamins and acetaminophen at a private hospital but with no improvement in earlier symptoms and the onset of tachypnoea, pallor and weakness, they were referred to our institution.

The mother was a 37-year-old who had received antenatal care for the twin pregnancy from 3 months of gestation. The pregnancy was uneventful until the third trimester when she developed a febrile illness, palpitations and excessive weakness. She was admitted at the private facility and managed for severe anaemia and cardiomyopathy, receiving 4 units of blood over approximately 3-weeks on admission and then discharged home. The pregnancy was again complicated at term by Severe Ecclampsia and she had a spontaneous vaginal delivery of the twins who weighed 2.5 kg and 2.3 kg respectively. After delivery, the mother was continued on trimethadione and also received torsemide, dabigatran, trimetazidine and digoxin. Further details of her management at the private facility were unavailable.

The babies had neonatal jaundice which was first noticed on the 3rd day of life and received

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phototherapy for approximately 1 week. They were breastfed from birth until 3 months of age, when breastfeeding was stopped on medical advice because of the maternal medications listed previously and breastmilk substitutes were commenced. There was no known family history of bleeding disorders.

On examination at presentation, both infants were in respiratory distress, severely pale, anicteric, with low grade fever (37.9°C-38.3°), tachycardia and tachypnoea. Both twins had tender hepatomegaly and Twin 1 who was also observed to have a splenomegaly. Their weights at admission were 5 kg and 5.2kg respectively. The initial working diagnoses were severe anaemia secondary to upper gastrointestinal bleeding with anaemic heart failure and differential diagnoses were congenital leukaemia and Immune thrombocytopaenia.

Table 1: Clinical Timeline

Time	Clinical event
Antenatal period	Mother developed severe anaemia and systemic symptoms in the third trimester; received four units of blood during a 3-week admission.
Term	Pregnancy complicated by severe eclampsia. Spontaneous vaginal delivery of female twins weighing 2.5 kg and 2.3 kg.
Day 3 of life	Neonatal jaundice developed; treated with phototherapy for approximately one week. No umbilical-stump bleeding reported.
Birth to 3 months	Twins were breastfed.
2 months of age (2 months before presentation)	Recurrent melaena and occasional nasal/gingival bleeding began.
3 months	Breastfeeding stopped on medical advice because of maternal medications; breastmilk substitutes commenced.
2 weeks before presentation	Low-grade intermittent fever developed.
2 days before presentation	Tachypnoea, severe pallor and generalized weakness developed.
Admission at 4 months	Severe anaemia and profound thrombocytopenia documented. Whole-blood transfusion and supportive treatment commenced.
During admission	Maternal autoimmune evaluation was requested; parents carried out testing in the mother but not in the infants. Bone marrow biopsy was declined in the infants.
Following steroid therapy	No clinical improvement in bleeding or platelet count was observed in the infants.
Following IVIG	Platelet counts increased and bleeding stopped; infants were discharged on haematinics alone.
Follow-up	Platelet counts remained within the normal range on subsequent review. Mother underwent multidisciplinary review and was diagnosed with SLE and commenced on treatment.

Diagnostic Assessment

Initial laboratory investigations revealed severe anaemia and severe thrombocytopaenia in both infants (Table 2), and the peripheral blood films were reported as showing features of extravascular haemolysis with a leuco-erythroblastic picture. Clotting time was not prolonged in both. The infants also had leucocytosis which widened the differential diagnosis further. Blood cultures yielded Staphylococcus aureus in Twin 1 and Micrococcus species in Twin 2. Imaging studies were not done in the infants as care was out-of-pocket and parents had financial constraints.

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Table 2: Initial Investigation on the 1st day on admission

Parameter	Twin 1	Twin 2	Reference Range
Haematocrit (%)	12.4	21.3	35-48
WBC ($\times 10^9$/L)	71.34	16.08	4.0-10.8
ANC ($\times 10^9$/L)	26.64	3.7	
ALC ($\times 10^9$/L)	41.59	11.3	
Platelets ($\times 10^9$/L)	14	8	150-450
Neutrophils (%)	33.2	23	40-75
Lymphocytes (%)	58.5	70.6	20-45
Monocytes (%)	8.2	5.3	
Malaria parasite	Not detected	Not detected	
PT (seconds)	13	13	
Control PT (seconds)	13	13	
APTT (seconds)	35	37	
Control APTT (seconds)	36	36	
INR	1.0	1.0	
Blood group	A Rh D-ve	A Rh D-ve	
Blood Culture	Staphylococcus aureus	Micrococcus spp	

Therapeutic Intervention and Outcome

The twins were transfused with freshly donated whole blood and received Intravenous antibiotics, Vitamin K and Ranitidine. Platelet concentrates were not readily available and so could not be given. Direct antiglobulin tests performed after transfusion were negative in both infants.

Following review by the haematologists, the diagnosis of PIT was entertained and further investigations requested for both the mother and the babies (Table 3). Parents declined the requested autoimmune investigations in the infants and also refused a bone marrow biopsy. Parents carried out the Autoimmune Screen (ANA, DS-DNA, RH Factor) and bone marrow investigation on the mother alone.

Intravenous methylprednisolone was commenced during admission and subsequently escalated to high-dose therapy from the first week on admission. There was however no significant clinical or platelet response, with bleeding persisting and severe thrombocytopenia on serial testing. Repeated fresh whole blood transfusions were given as platelet concentrate remained unavailable throughout the admission.

Table 3: Post-transfusion investigation results, day 3 on admission

Investigation	Twin 1	Twin 2	Reference Range
Haematocrit (%)	25.6	34.4	35-48
WBC ($\times 10^9$/L)	30.08	14.73	4.0-10.8
ANC ($\times 10^9$/L)	13.37	4.84	
ALC ($\times 10^9$/L)	15.71	8.88	
Platelets ($\times 10^9$/L)	14.5	14	150-450
Neutrophils (%)	44	32.9	40-75
Lymphocytes (%)	52	56.8	20-45
Monocytes (%)	3	8.6	
Direct antiglobulin test	Negative	Negative	
Total bilirubin (µmol/L)	7	5	
Conjugated bilirubin (µmol/L)	Not reported	0	
AST (IU/L)	47	45	
ALT (IU/L)	37	22	
ALP (IU/L)	528	372	
γ-GT (IU/L)	46	76	
Total protein (g/L)	66	72	
Albumin (g/L)	25	35	

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Intravenous Immunoglobulin was administered by the third week on admission and a marked increase in platelet counts followed thereafter (Tables 5 and 6), accompanied by cessation of melaena and other bleeding manifestations. The infants were discharged on haematinics alone and their platelet counts and other haematologic parameters have consistently remained normal through the 1 year of follow up.

Maternal Autoimmune Evaluation

The mother had a multi-specialist review while the twins were on admission and based on clinical criteria, thrombocytopaenia, anaemia, lymphopaenia and a positive ANA, a diagnosis of systemic lupus erythematosus was made despite a negative Anti dsDNA and the absence of an Anti-Smith antibody result. Anti-Smith antibody assay and further immunological test were not carried out by the mother due to financial constraints.

She commenced initial treatment with IV Methylprednisolone with good clinical and laboratory response and then received further treatment for SLE and has been on follow up.

Table 4. Maternal laboratory investigations

Maternal assessment	Result available in supplied record	Reference range
Haematocrit (%)	26.4	35-48
WBC ($\times 10^9$/L)	5.7	4-10.8
ANC ($\times 10^9$/L)	3.1	
ALC ($\times 10^9$/L)	1.3	
Platelets ($\times 10^9$/L)	23	150-450
Lymphocytes ($\times 10^9$/L)	18	20-45
Direct Antiglobulin test	Negative	
Bone Marrow Aspiration	Features suggestive of Autoimmune disease	
ANA	9.50	(Positive)
Anti dsDNA	10.81 IU/mL	[<100 IU/ml is a negative result]
Anti smith antibody	Not done	
Rheumatoid factor	80.10IU/L	[0-14]
Final clinical assessment	Systemic lupus erythematosus diagnosed after multidisciplinary review	

Table 5: Serial Complete blood count results of Twin 1

Parameter	Day 4	Day 7	Day 8	Day 17	Day 25
Hb (g/dL)	11.6	12	6.7	4.8	14.1
HCT (%)	34.2	34	19.6	14.8	42.0
MCV (fL)	84.8	85	86	79.2	81.2
MCH (pg)	28.8	29	29.4	25.9	27.2
WBC ($\times 10^9$/L)	38.16	38.1	37.7	11.0	18.08
MCHC (g/dL)	33.9	34	34.1	32.7	33.5
Platelets ($\times 10^9$/L)	16	16	17	12	52
Neutrophils (%)	45.3	45	49.9	16.7	39.2
Lymphocytes (%)	45.6	46	46.9	76.4	44.6
Monocytes (%)	7.2	7	0.5	6.4	10.3

Table 6: Serial Complete blood count results for Twin 2

Parameter	Day 4	Day 7	Day 8	Day 17	Day 25
Hb (g/dL)	11.3	8.2	8	5.7	12.5
HCT (%)	34	25.3	25	16.4	36.1
MCV (fL)	79	79.1	79	85.6	80.9
MCH (pg)	26	25.5	26	29.5	28.0
WBC ($\times 10^9$/L)	14.7	25.99	25.9	44.38	23.71
MCHC (g/dL)	32	32.2	34.5	34.5	34.6
Platelets ($\times 10^9$/L)	14	9	9	15	42

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Neutrophils (%)	33	57.1	34	69.6	43.6
Lymphocytes (%)	57	—	57	28.8	54.3
Monocytes (%)	9	—	7	1.5	1.3
Eosinophils (%)	1	—	—	—	—

Table 7: Differential Diagnosis

Differential	**Comparison with this case**
Primary immune thrombocytopenia (ITP)	ITP is a diagnosis of exclusion and is a common acquired cause of thrombocytopenia in childhood. Classic childhood ITP more often presents after 6 months of age with otherwise relatively preserved blood counts and without a systemic illness. The marked anaemia in these infants was clinically attributed largely to gastrointestinal bleeding, while the maternal diagnosis of SLE provided an alternative source of antiplatelet antibodies. The absence of a platelet response to corticosteroids made primary ITP less compelling, although steroid non-response alone cannot exclude ITP.[4,10]
Secondary immune thrombocytopenia due to infection	Both infants had fever and positive blood cultures. Infection can cause thrombocytopenia through immune destruction, reduced production or consumption. However, the profound thrombocytopenia was present with the bleeding illness, the coagulation profile was not prolonged, and both infants subsequently achieved sustained platelet recovery after IVIG. Infection therefore remains an important contributing diagnosis but does not fully explain the overall pattern.
Neonatal alloimmune thrombocytopenia (NAIT)	NAIT results from maternal antibodies directed against fetal platelet antigens inherited from the father and typically causes severe thrombocytopenia in the neonatal period.[8] The absence of neonatal bleeding and the first documented thrombocytopenia at 4 months make NAIT unlikely. Definitive exclusion would require appropriate platelet antigen/antibody studies.
Evans syndrome	Evans syndrome combines immune thrombocytopenia with autoimmune haemolytic anaemia, usually supported by evidence of haemolysis and a positive direct antiglobulin test.[11] In this case, the direct antiglobulin tests were negative, bilirubin levels were not elevated, and the severe anaemia was clinically attributed largely to gastrointestinal blood loss. Thus Evans syndrome was considered but not supported by the available evidence.
Congenital leukaemia or marrow infiltration	The marked leukocytosis, leucoerythroblastic blood film and hepatosplenomegaly justified consideration of congenital leukaemia or another marrow-infiltrative disorder. Bone marrow examination was declined, so these diagnoses could not be excluded definitively by marrow morphology. Nevertheless, the subsequent sustained normalisation of platelet

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	counts and clinical recovery make an active marrow-infiltrative process less likely.
Congenital/inherited thrombocytopenia or marrow failure	Inherited platelet disorders and congenital marrow failure can present in infancy, but the absence of a family history, the absence of reported neonatal bleeding and subsequent recovery of platelet counts argue against a persistent inherited disorder.[3]
Disseminated intravascular coagulation (DIC)	DIC can cause thrombocytopenia and anaemia in a severely ill infant. However, the PT, APTT and INR were within the reported reference/control ranges, making overt DIC less likely at the time of assessment.
Passive maternal autoimmune thrombocytopenia	The maternal diagnosis of SLE, the occurrence of severe thrombocytopenia in both twins, and the absence of another confirmed haematological diagnosis support a maternal antibody-mediated mechanism. The unusually late presentation is atypical, and the absence of platelet-specific antibody testing means that this diagnosis remains probable rather than proven.

DISCUSSION

Thrombocytopaenia presenting in older infants is commonly autoimmune with antiplatelet autoantibodies formed by the child's immune system suddenly attacking the platelets. In the case of ITP, it may occur following an initial viral infection, immunisation (most commonly measles) in infants, or just be idiopathic.[1,4] The peak age of occurrence is 1-4years and presentation is usually that of a previously healthy child with sudden onset of petechiae or purpura and mucosal bleeds. An insidious onset of bleeds as seen in the twins above, usually indicates either a chronic ITP or a systemic illness.[1] A differential worthy of note is Evans Syndrome which occurs in less than 5% of patients with ITP and may present with pallor, fatigue, jaundice and other signs of anaemia in addition to easy bruising/bleeding, petechia, splenomegaly and recurrent infections.[8] It is an autoimmune disease comprising of both an Autoimmune Haemolytic Anaemia(AIHA) and autoimmune thrombocytopaenia with or without immune neutropaenia and due to IgG reaction with surface antigens on both the red blood cells and platelets, a confirmation of diagnosis can usually be made with a Positive Direct Coomb's test. This however was not the case for the twin infants in this report whose anaemia was deduced to be secondary to the bleeds.

Passive immune thrombocytopenia is classically a neonatal disorder, so the persistence of profound thrombocytopenia to 4 months in this case is atypical and is the principal feature that makes the report clinically noteworthy. The diagnosis in this report was based on a convergence of clinical and laboratory findings rather than a single confirmatory test as both maternal and infant platelet-specific antibody testing was not performed and the infants' autoimmune investigations and bone marrow examinations were not completed. Nevertheless, the profound thrombocytopaenia in both twins makes a shared exposure or maternal mechanism very plausible. Additionally, the maternal diagnosis of SLE, an autoimmune disorder associated with maternal antiplatelet antibodies and infant thrombocytopenia,[7,8]the absence of evidence of congenital platelet disorder (no symptomatic thrombocytopaenia at birth) and the quick response to IVIG with sustained normal platelet counts thereafter make the diagnosis of PIT very probable. The immediate response to IVIG also suggests that IgG antibodies were mostly responsible for the immune thrombocytopaenia.

It was unlikely that IgA antibodies in the breastmilk still played a part in the persistently severe thrombocytopaenia in the twins as breastfeeding had been discontinued 1 month prior to presentation, hence the differential diagnosis was not further explored.

Seventy-five (75%) of children with Immune thrombocytopaenia respond to steroids within 2-7

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days of use,[12]in this report however, there was no response in the infants to even high doses of corticosteroids after more than 1 week of treatment, while the mother's platelets responded rapidly to same steroids. This can be due to the fact that IVIG directly targets the mechanism of platelet destruction and not antibody production and since the maternal IgG are already in circulation and not being produced anew, there is a quick halt in platelet destruction. This is unlike steroids which suppress the infant's immune system in an attempt to halt antibody production.[13]

CONCLUSION

when faced with sudden onset of thrombocytopaenia in an infant, the need for the consideration of Passive immune thrombocytopaenia and investigation of the mother for an autoimmune thrombocytopaenia is necessary, to ensure wholesome management of mother and child. Also the importance of consideration of early use of IVOG in Immune thrombocytopaenia is paramount to ensure quick response, and forestall complications and financial implications that may arise from persistent and severe thrombocytopaenia in such infants.

Strengths and Limitations

Strengths include the presentation of twin siblings with a shared clinical phenotype, identification of previously unrecognised maternal SLE, serial platelet measurements and documented recovery after IVIG. Limitations include incomplete maternal autoimmune results in the available record, absence of maternal/infant platelet antibody testing, incomplete infant autoimmune evaluation, refusal of bone marrow examination, and concomitant bacterial infection. These limitations mean that the proposed mechanism is clinically supported but not laboratory-confirmed.

Declarations

Ethics approval and consent to participate: Informed consent for publication of this case report and the accompanying clinical information was obtained from the children's parents.

Consent for publication: Not applicable

Availability of data and materials:

Authors' contributions:

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