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When Dengue Defies Expectations: Unmasking Immune Thrombocytopenic Purpura as a part of Expanded Dengue Syndrome

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Abstract

Dengue is a prevalent arthropod-borne viral disease in tropical and subtropical areas of the globe. Dengue clinical manifestations include asymptomatic infections; undifferentiated fever; dengue fever, which is characterized by fever, headache, retro orbital pain, myalgia, and arthralgia; and a severe form of the disease denominated dengue hemorrhagic fever/dengue shock syndrome, characterized by hemoconcentration, thrombocytopenia, and bleeding tendency. However, atypical manifestations, such as liver, central nervous system, respiratory, lymphoreticular and cardiac involvement, have been increasingly reported called expanded dengue syndrome. Thrombocytopenia usually gets better and platelet count normalizes by day 10 of fever. Chronic thrombocytopenia is not a feature of dengue fever. Immune thrombocytopenic purpura (ITP) is an autoimmune disorder characterized by low platelet count and skin-mucosal bleeding. It commonly affects adult female in an idiopathic and chronic manner. Thrombocytopenia associated with dengue viral infection seems to result in both from a reduction in the production of platelets from megakaryocytes and immune mediated destruction of platelets. Here we report a 30-year-old Bangladeshi lady who having persistent thrombocytopenia beyond two weeks of dengue illness who responded to steroid therapy on the line of immune thrombocytopenia after ruling out other common causes of thrombocytopenia. The report also highlights the value of monitoring platelet counts in post-recovery phase to ensure they have normalized.

Keywords: Immune thrombocytopenic purpura, expanded dengue syndrome, dengue fever

Introduction: Dengue, an arthropod-borne viral infection of humans, is endemic to tropical and subtropical regions of the world and represents an important public health problem. Dengue viruses are transmitted by the bite of the *Aedes aegypti* mosquito infected by the one of the four dengue virus serotypes: dengue-1, -2, -3, and -4. More recently, dengue disease has spread geographically to many previously unaffected areas and, as travelling around the world has become more accessible, physicians in temperate areas are more likely to see returning travelers with dengue infection.[1,2]

World Health Organization (WHO) classification of symptomatic dengue infection, continuously evolved, first in 1997 it divided into dengue fever (DF), dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). In 2009 it improved into dengue with or without warning signs and severedengue.3 However, in 2011, WHO Regional Office for South East Asia (SEARO) revised and further improving the classification, divided into DF, DHF without shock or with shock (DSS) characterized by increased vascular permeability, thrombocytopenia (platelets <100,000), bleeding tendency, and, in a small percentage of patients, circulatory shock.[4,5,6,7] and expanded dengue syndrome.[8]

Expanded dengue syndrome is a new entity added to the classification system to incorporate a wide spectrum of unusual manifestations of dengue infection affecting various organ systems that had been reported including gastrointestinal, hepatic, neurological, cardiac, pulmonary and renal systems.[8]

Unlike other viral infections, immune thrombocytopenia determined by dengue infection is a rare complication.

Thrombocytopenia and hemorrhagic manifestations are consistent features of dengue fever (DF). Usually, thrombocytopenia is resolved by day 10 of fever. Very few cases have been reported worldwide and there may be thrombocytopenia lasting more than ten days. All these reported cases behaved like immune thrombocytopenia (ITP) and improved completely with steroid therapy with prolonged maintenance of platelet counts. Usual thrombocytopenia of DF does not respond to steroids. Probably previous reports, including ours, indicate that dengue virus infection can produce a persistent thrombocytopenia like immune mediated thrombocytopenia.

Immune thrombocytopenic purpura (ITP) is a form of acquired thrombocytopenia triggered by antiplatelet antibodies that destroy platelets peripherally, damage megakaryocytes, and inhibit platelet production in the marrow. An increased level of platelet-associated IgG antibody is observed in patients with idiopathic thrombocytopenic purpura (ITP) but is also found in a variety of diseases.

Acute idiopathic thrombocytopenia purpura (ITP) can develop 7 to 10 days after infectious such the cytomegalovirus, hepatitis-C virus, mumps, rubella, Epstein Barr virus, and parvovirus B19, among others, have been identified as the culprits behind this form of ITP.[9,10] It generally occurs at a time when the virus is cleared from the circulation. Platelets that are sensitized by autoantibodies are destroyed by cells of the reticuloendothelial system, particularly those of the spleen. These autoantibodies against glycoproteins of the platelet membrane can be identified in 80% of the patients.[11] The diagnosis of ITP is achieved by ruling out other possibilities.[12] However, other causes of low platelets should be investigated, such as systemic lupus erythematosus (SLE), HIV/AIDS, pregnancy, use of medications (heparin, sulfa and quinidine) and recent blood transfusion, among others. We reported a case of a lady who was presented in dengue season in 2025 with fever, epistaxis and bleeding gum and was diagnosed as a case of dengue hemorrhagic fever. Subsequently, she developed persistent low platelets which required treatment on the lines of immune thrombocytopenia and responded to steroids. Other causes of thrombocytopenia were ruled out.

Case report: A 30 year old Bangladeshi lady, not known to have any diabetes mellitus, hypertension, bronchial asthma or epilepsy presented to us with the history of high grade, intermittent fever, severe headache, body ache and retro orbital pain for 2 days, vomiting for several times for the same duration. She denied any cough, chest pain, palpitation, shortness of breath, abdominal pain or distension, burning micturition, joint pain. She had no recent history of travel of late. She lives on the 2nd floor of his apartment and has hobby of gardening. His elder brother just recovered from dengue 1 week prior his illness. On examination, she was conscious, febrile (temperature 103oF), with pulse 110 beats/min, with normal rhythm and volume, blood pressure was 90/70 mm of Hg. There was diffuse blanching erythema, more prominent over trunk. Other systematic examination revealed no abnormalities. On second day of admission, she became afebrile and developed gum bleeding, epistaxis and abdominal pain. Repeat clinical examination revealed blood pressure 80/50 mm of Hg, pulse 110 beats/min, capillary refill time 4 secs. There was evidence of ascites and bilateral pleural effusion on abdominal and respiratory system examination respectively. USG of whole abdomen showed ascites bilateral mild pleural effusion with edematous gall bladder wall suggestive of acalculous cholecystitis.

She was resuscitated with crystalloid solution and monitored closely for development of shock and major bleeding. CBC was done twice daily for any change in hematocrit and platelet count. On 9th day of illness, her general well-being was improved with stable pulse and blood pressure and good appetite with adequate urinary output. On CBC all parameters were improved except platelet count which was 30000/cmm. She was given discharge on request on 10th day of illness with the advice to follow up at outpatient door with repeat CBC on the next day.

Test names (Reference range)	Day 1	Day 2	Day 4	Day 5	Day 8
Hb (12-16 gm/dl)	12	12.8	13.6	11.8	11.9
TC WBC (3500-11000/mm^3)	3700	3150	2320	4600	5100
Platelet(150000-450000/mm^3)	130000	92000	13000	27000	30000
Hct(35-47%)	37.1	40	48	44	36.5
SGOT (10-45 U/L)	315	404	458		96
SGPT (10-50 U/L)	218	387	412		69
Blood urea (15-40 mg/dl)	21	40	26.5		28
S. Creatinine (0.6-1.2 mg/dl)	1.1	0.8	1.1		1.0
S. Sodium (135-145 mmol/L)	130	141	136		140
S. Potassium (3.555mmol/L)	4.2	4.1	3.7		4.3
S. Calcium (8.5-10.5 mg/dl)			8.8		9.1
S. Albumin (3.5-5.0 g/dl)			3.3		3.8

Blood glucose (<7.8 mmol/L) Non fasting cholesterol(mg/dl)	7	5.7	6.9 96		5.9
C-reactive protein (<6 mg/L)		18	28		5
S. LDH (140-280U/L) ICT for malaria Blood film for malarial parasite Blood Culture Dengue NS1 antigen	 Negative Negative No growth Positive	210			

Lab Investigations during hospital stay

During outpatient (OPD) follow up her CBC showed persistent thrombocytopenia without any evidence of overt bleeding. Immune thrombocytopenia as a part of expanded dengue syndrome was suspected. To exclude secondary causes of Immune thrombocytopenia, ANA, Anti-HIV 1 and 2, HBsAg, Anti HCV were sent and all came negative. There was no history of taking drugs responsible for immune thrombocytopenia. Patient refused for bone marrow examination. After explaining the possible diagnosis to the patient, she was started tab. Dexamethasone 40 mg daily for 4 days as per American Hematological Society guideline to treat immune thrombocytopenic purpura. The follow up CBC is shown below.

Test names (Reference range)	Day 10	Day 12	Day 15	Day 19	Day 21
Hb (12-16 gm/dl)	12.1	12.8	12.6	12.2	11.9
TC WBC (3500-11000/mm3)	4100	6150	9300	10600	10100
Platelet(150000-450000/mm3)	21000	23000	22000	114000	264000
Hct (35-47%)	37.1	39.7	40.1	37.5	36.5

CBC during OPD follow up and before (day 15) and after steroid treatment (day 19)

Last OPD follow-up was arranged in two months which showed completely normal CBC.

Discussion: Dengue is a worldwide public health problem and causes innumerous deaths. More than 40% of the world's population lives in dengue endemic areas, and the World Health Organization estimates that about 2.5 billion people in 100 countries are at risk of infection and that as many as 100 million people are infected by dengue viruses every year. In the majority of infected people, dengue is an auto-limited disease that resolves in 5–7 days. However, approximately 500,000 people develop a severe form, leading to about 20,000 deaths annually. Consequently, approximately 0.5% of dengue patients develop a severe form and requires specialized treatment.[2,13]

Dengue virus infection is a disease that found in children and adults with the main symptoms of fever, muscle and joint pain that usually worsens after the first three days. This disease is an acute febrile illness accompanied by bleeding manifestations with potential shocking and can lead to death in children<15 years, but not likely to attack adults.14 Signs of this disease are sudden high fever 2 to 7 days with no obvious cause, weakness, lethargy, anxiety, heartburn, accompanied by signs of bleeding in the skin (petechiae), bruising (ecchymosis) or rash (purpura).Sometimes there are other spontaneous bleeding manifestations such as nosebleeds, bleeding gums to dysentery. Severe symptoms can lead to decreased awareness or shock.[15]

Laboratory results in dengue fever are found in thrombocytopenia (20% of the baseline on dengue hemorrhagic fever is a sign of plasma. Serological tests results in dengue are influenced by the type of dengue infection, whether it is the primary/first, or secondary/reinfection. IgM antibodies are detectable by days 3–5 after the onset of illness, rise quickly in two weeks and decline to undetectable levels after 2–3 months, because this late appearance, the first five days of clinical illness are usually negative of IgM. In dengue secondary infection, the rise of IgM are not as high as primary infection, and sometimes absent / undetectable completely.[16]

IgG antibodies in primary infection, evolves relatively slow, with low titres 8-10 days after fever onset, increase subsequently and remain for many years, whereas in secondary infection it evolves rapidly, with high titres soon after fever onset and persist to a lifelong period. Hence, a ratio of IgM/IgG is commonly used to differentiate between primary and secondary dengue infections. Ratio of IgM/IgG titre less than 1.2 is considered a secondary dengue infection. But to be noted, titre ratio only could be validly use as data if the IgG/IgM serological test is using pure quantitative means, not by qualitative or semi-quantitative.[17]

NS1 antigen detection is widely used and cost-effective, NS1 could be detected from day 1-8 of fever onset, unaffected by a primary or secondary dengue infection. In conclusion, by combining the serological (IgG and IgM) and NS1 tests, clinicians could rapidly assess the dengue diagnosis with its types (primary or secondary infection) and apply the best treatment.[18]

In 2011, based on many reports of cases with dengue-related unusual manifestations and organ complications, WHO-SEARO further improved and revised 2009 WHO guidelines by adding a new entity, that is expanded dengue syndrome (unusual/atypical manifestation of dengue), these include neurological, hepatic, renal, cardiac and other isolated organ involvement, that could be explained as complications of severe, profound shock or associated with underlying host conditions/diseases or co infections.[8]

Low platelets and bleeding manifestations are consistent features of dengue fever.19 Typically, thrombocytopenia resolves by day 8 -10 of fever.20 However, few cases were reported worldwide having lengthy thrombocytopenia beyond day 10. The exact mechanism of thrombocytopenia is still clearly not known. Direct platelet destruction by dengue virus, immune mediated platelet destruction and even megakaryocytic immune injury have been proposed as underlying mechanisms. During a dengue endemic, ITP can go unnoticed. It is up to the clinician to have a high index of suspicion and identify patients who do not follow the usual course of dengue fever. An ITP can easily imitate and confound the diagnosis of a fever and thrombocytopenia syndrome. Therefore, the possibility of ITP must be entertained if thrombocytopenia persists beyond the expected period of platelet count turn around. A study by La Russa has demonstrated dengue mediated bone marrow suppression causing low platelets.[21] However, Wang et al, cited that anti-body coated platelets were cleared by immune-mediated reaction which responsible for thrombocytopenia in dengue fever.[22] In addition to that peripheral destruction of platelet also contributes to thrombocytopenia mainly by anti-platelets IgM serotype.[23] A study by Petaja J demonstrated that both coagulation and fibrinolytic paths are triggered in dengue infection leading to consumption of platelets.[24] demonstrated Furthermore, that a study cross-reactivity of antibodies against NS-1 antigen and the platelets proposes role of antiplatelet antibody as pathogenesis of thrombocytopenia during dengue virus infection.[25]

ITP is an auto- immune disorder characterized by low platelet count and skin-mucosal bleeding [26] ITP being a diagnosis of exclusion does not do justice to the threatening virulence it carries. It commonly, affects adults in an idiopathic and chronic manner, and it is found twice as frequently among women as among men. There are two major mechanisms paying to the development of ITP; increased platelet destruction by auto antibodies against platelet membrane glycoprotein and inadequate platelet production.[27] Persistent thrombocytopenia after dengue fever is a rare entity with only few reported cases. In our patient's fever was settled fourth day after the onset of fever, but persistently platelets were in the low side. In the accepted history of illness all the patients show recovery and platelet count recovers to the pre-illness level.[28] Although most cases are asymptomatic, very low platelet counts can lead to bleeding diathesis. Mucocutaneous bleeds such as petechiae, purpura, and easy bruising are expected in ITP. Older patients have more severe hemorrhagic manifestations ranging from gastrointestinal bleeds to intracranial bleeds [29]. The nature of the bleeding usually corresponds to the platelet count. It is known that ITP can be divided between primary or secondary as well as acute or chronic. In adults, ITP is primary, chronic and idiopathic. Acute, secondary ITP is usually seen in children after a viral infection and is self-limiting. The causes of ITP are given in Table 1. The mechanisms involved in the resulting thrombocytopenia are given in Table 2 [30]

Table-1: Cause of Immune Thrombocytopenic Purpura

Primary
Antibody mediated
Idiopathic
Secondary
Post Infectious: HIV, HCV, EBV, CMV, H. pylori
Drugs: Quinidine, Heparin
Lymphoproliferative Disorder: Leukemia (CLL), Lymphoma
Vasculitis/ autoimmune disorders: SLE, RA, APS, Evans syndrome
Others: Post MMR vaccination, Cirrhosis

Table-2: Mechanism of Thrombocytopenia in ITP

Increased platelet destruction
Breakdown in central and peripheral immune tolerance of T cells and B cells
Antiplatelet antibodies against glycoprotein IIb/IIIa, resulting in its destruction
Drug or inactivated/live pathogen binds to platelet surface recruiting antibodies, thereby triggering a

temporary immune response to platelets
Foreign antigens cross-react with platelet antigens (molecular mimicry)
Development of anti-platelet antibodies

Decreased platelet production

Anti-platelet antibodies also target megakaryocytes
Impaired function of megakaryocytes
Foreign antigen/virus induced bone marrow suppression
Insufficient thrombopoietin levels

Clinical indicators hinting at ITP are refractory, persistent, prolonged thrombocytopenia, and poor response to donor platelet transfusions. At present, there are no scientific recommendations or evidence available to predict the development of secondary ITP. Nor are there any specific criteria available to formulate a diagnosis of ITP. Currently, the diagnostic approach is based essentially on a process of exclusion[31] A failure of platelet counts to rise well after the illness should rouse the thought of an ITP. Unfortunately, as it may hold true for most diseases, a high index of suspicion and a search for certain clinical indicators may be the only approach to diagnosing such a predicament. To the best of our knowledge, only a few authors in the past have reported an association with dengue and ITP, all having a dramatic response with corticosteroids [32,33,34,35]

Our patient had a platelet count of 22000/mm3 on day 15 of illness and responded to dexamethasone promptly. There are few cases where reported that prolonged low platelets were well responded with steroids[36] ITP in adults commonly requires treatment using oral prednisone/ dexamethasone at the time when it is presented. Intravenous immune globulin (1 g/kg/day for 2 to 3 consecutive days) is used for treating internal bleeding when the platelet count is less than 5000/µl despite corticosteroid therapy, or when there is progressive or extensive purpura.[37] Patients who continue to show symptoms and who have severe thrombocytopenia (platelet counts of less than 10 000/µl) after this time can then be assessed for the possibility of splenectomy.

Conclusion: Thrombocytopenia following dengue is common but is usually transient. Persistent thrombocytopenia is rare but not uncommon in dengue fever and usually responds to steroid therapy. Primary and secondary ITP can be a masquerader as well as an offspring of dengue fever. An ITP can mimic and go undetected and unnoticed during a dengue endemic. The physician should intently be on the lookout for certain telltale signs and features and signs that indicate an ITP in a patient presenting with a fever and thrombocytopenia syndrome, particularly during a dengue outbreak. Early detection and a prompt initiation of treatment need no clarification of its benefits. A careful assessment for clinical indicators of an ITP especially during a dengue endemic is vital to timely diagnosis.

Conflict of interest: None declared

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