**Thrombotic microangiopathy in a patient with primary dengue fever**

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**Introduction**

Thrombotic microangiopathy (TMA) is a broad term which includes thrombotic thrombocytopenic purpura (TTP), haemolytic uraemic syndrome (HUS) and other diseases characterised by common clinical and pathological features. Clinical features include thrombocytopenia, microangiopathic haemolytic anaemia (MAHA) and organ injury. Pathological features include arteriolar and capillary thrombosis due to abnormalities in the vessel wall and endothelium [1]. Bacterial infections like *Shigella dysenteriae* type I and *Escherichia coli* O157:H7 are well known to produce HUS. Viral infections including dengue virus leading to TMA are reported in the literature. We report a case of dengue fever presenting with TTP.

**Case report**

A 27-year-old sailor with acute febrile illness with headache and arthralgia for two days was admitted to Navy Hospital, Welisara. His white cell count was 4.6x10³/µl (N-63%, L-25%, E-2%, M-10%), haemoglobin (Hb) was 12.7 g/dl and platelet count was 32x10³/µl. Serum bilirubin was 3.4 mg/dl (normal range 0.2-1.2) with a high indirect fraction 2.75 mg/dl. Mild to moderate elevation of liver transaminases and albuminuria were detected. On fourth day of the disease schistocytes, polychromasia, thrombocytopenia and reactive lymphocytes were found in the peripheral blood smear and TTP/HUS was suspected. Purpuric patches were noted on the back of the chest. By the sixth day the Hb became 10.9 g/dl with a platelet count of 22x10³/µl. Packed cell volume (PCV) remained low. Serum creatinine rose to 2.25 mg/dl and LDH was 6250 U/l (normal range 230-460). There was no clinical evidence of active bleeding. Abdominal ultrasonography did not show any fluid leakage and non-contrast CT scan of brain was normal. He was haemodynamically stable. A diagnosis of TTP was made and the patient was transferred to National Hospital, Sri Lanka (NHSL) for plasmapharesis. In NHSL, fresh frozen plasma (FFP) was given and one cycle of plasmapharesis was performed. Since his blood picture indicated MAHA with reactive lymphocytosis, a viral aetiology was sought. On the eighth day of the illness, IgM antibody for dengue was detected. Dengue IgG antibody, and antibodies against CMV, Hanta virus and HIV were negative. On the fifteenth day of the illness, both dengue IgG and IgM antibodies were positive. The serum creatinine and platelet count became normal at the time of discharge from hospital.

**Discussion**

This young man had dengue fever and the clinical picture was complicated with microangiopathic haemolytic anaemia in the early phase of the illness. He had fever, headache, thrombocytopenia, MAHA, and elevated serum creatinine with albuminuria suggesting a diagnosis of TTP. Multiple viruses are capable of inducing thrombotic microangiopathy either through stimulation of endothelial cells or activating alternative pathways (complement mediated). In flaviviridae family dengue and hepatitis C are known to produce TMA [2]. Suggested pathogenesis of TMA in dengue fever includes direct endothelial injury, formation of ADAMTS13 inhibitors and immune complex mediation [3].

In this case acute seroconversion against dengue virus was shown by the initial presence of IgM antibodies followed by positive IgG during convalescence. There was no leakage of fluids and patient responded to plasma transfusions and plasmapharesis. When the Hb level and PCV are low in a patient with dengue fever, it would be appropriate to entertain the possibility of TMA and to perform blood picture and LDH level.

**Ethics**

Patient gave informed, written consent for publication of his clinical details.

**Conflicts of interests**

There are no conflicts of interest.

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A case of severe zinc phosphide poisoning

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Introduction

Zinc phosphide (ZnP) a rodenticide available over the counter, releases phosphine gas that impairs mitochondrial respiration. We report a case of suicide due to ZnP poisoning. She had severe metabolic acidosis, acute pulmonary oedema, acute kidney injury, acute liver failure and coagulopathy. She also had hyperglycaemia, which is rare and a poor prognostic indicator of phosphine gas exposure [1]. Although aluminum phosphide and ZnP poisoning are widely reported in the Indian sub-continent, fatal ZnP poisoning is rare in Sri Lanka [2].

Case report

A 14-year-old girl was admitted to the local hospital following ingestion of 5g of 25% ZnP (Run-rat) mixed with water. She developed profuse vomiting seven hours after ingestion of the rodenticide. After initiation of supportive care she was transferred to Teaching Hospital Anuradhapura. On admission to the emergency treatment unit, she was conscious with a Glasgow Coma Scale of 15, pulse rate of 80/minute and a blood pressure of 100/70 mmHg. Examination of lungs and abdomen were normal. She was commenced on intravenous normal saline at a rate of 50 ml/hour and was given metoclopramide 10 mg intravenously.

During the next one hour, she had repeated episodes of vomiting and gradually developed difficulty in breathing. The peripheral oxygen saturation was 92% breathing on air, pulse rate was 100 beats/minute, blood pressure was 100/60 mmHg and abdominal examination elicited right hypochondriacal tenderness. She was given oxygen 2 l/minute through a face mask. Arterial blood gas showed evidence of metabolic acidosis [pH 7.2 (normal range 7.35-7.45), PaCO2 11 mmHg (normal range 35-45), PaO2 111 mmHg (normal range 70-100), HCO3 3 mEq/l (normal range 24-34)] and a prolonged INR (2.05) indicating acute liver injury. In addition she had prolonged activated partial thromboplastin time [84.1s (normal range 24-34s)] and thrombocytopenia (123 000/μl) indicating coagulopathy. Fibrinogen and fibrin degradation product levels could not be performed due to unavailability. Serum creatinine (Cr) and blood urea (BU) were elevated [Cr 145 (60-120) μmol/l, BU 8.8 (2.6-7.7) mmol/l], and serum electrolytes were normal.

She was treated with soluble insulin infusion, intravenous sodium bicarbonate, intravenous vitamin K...