

Intracranial Haemorrhage in a Patient Diagnosed with Idiopathic Thrombocytopenic Purpura and Diabetic Ketoacidosis

The Editor

Sir,

A 15-year old girl presented with rapid respiration and excessive water consumption and urine production. At physical examination, her general condition was average, and she was lucid, oriented and cooperative. Respiratory acidosis was present. There was no purpura on the skin. At laboratory tests, urinary glucose was 4⁺ and urinary ketones 4⁺. Serum glucose was 459 mg/dL, sodium 137 mmol/L and potassium 4.7 mmol/L. At full blood count, haemoglobin was 11 g/dL, white cell count $12.2 \times 10^3/\mu\text{L}$ and thrombocyte number $78 \times 10^3/\mu\text{L}$. Thrombocyte clustering, albeit minimal, was seen in the peripheral smear. Blood gas pH was 7.14, bicarbonate 7.4 mmHg and pCO₂ was 22.2 mmHg. Prothrombin time was 11.9 seconds, active partial thromboplastin time 26.3 seconds and international normalized ratio (INR) 1.12. Factor levels, lupus anticoagulant levels, protein C, protein S, antithrombin 3 activity, activated protein C resistance, fibrinogen and D-Dimer levels were within normal ranges. Serum osmolarity on arrival was 319 mOsm/L. Glycated haemoglobin (HbA_{1c}) was 12.3%. Type 1 diabetes mellitus-related diabetic ketoacidosis (DKA) was diagnosed, and a DKA protocol initiated such as to be completed in 36 hours was started. No haemorrhage was observed, and the patient was placed on daily thrombocyte monitoring. At the end of 36 hours, blood ketones and urinary ketones were negative, and the patient was fed by mouth. When the patient emerged from DKA, serum osmolarity was 294 mOsm/L. Headache began on the 2nd day of hospitalization. Sinusitis was determined from examination findings and Waters' view of the sinuses. The patient was started on intravenous ampicillin. Headache worsened on the 4th day of hospitalization. Thrombocyte values reached $42 \times 10^3/\mu\text{L}$. Nuchal rigidity was identified. Haemorrhage was determined in the right parieto-occipital region at tomography of the brain performed following detection of papillary oedema (Figure).

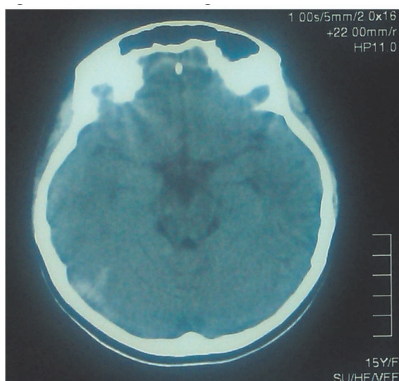


Figure: Intracranial haemorrhage in the right parieto-occipital region at tomography.

Cerebral magnetic resonance imaging (MRI) and MRI angiography revealed an area of lesion not significantly restricting diffusion, hyperintense on fluid-attenuated inversion recovery (FLAIR) and T2-weighted images and hypointense on T1-weighted images, with a lobulated margin, in the cortical and subcortical region. This was assessed as subacute stage haematoma. The brain surgery department reported no need for surgery. The patient was started on dexamethasone, and thrombocyte suspension was administered. Phenytoin was started following episodes of absence. Convulsions increased in intensity on the 10th day of monitoring, and tomography revealed resorption of the area of haemorrhage. Phenytoin and dexamethasone therapy was reduced and then stopped with no further episodes. Thrombocyte number at subsequent follow-up was $224 \times 10^3/\mu\text{L}$. The patient was discharged in a healthy condition. The case exhibited thrombocytopenia and was subsequently assessed as idiopathic thrombocytopenic purpura (ITP).

Less than one per cent of intracranial haemorrhage develops in paediatric ITP patients (1, 2). Diabetic ketoacidosis and its treatment result in a prothrombotic state and activation of the vascular endothelium and thus predispose to cerebrovascular outcomes (3). Haemorrhagic stroke is a rare presentation of juvenile DKA. Intracerebral haemorrhage has been reported in two adolescent girls who had juvenile DKA (4). The initial presentation of paediatric stroke can be subtle, with nonspecific changes in behaviour, new-onset seizures or altered level of consciousness. Asymptomatic or silent infarcts are believed to occur more frequently in diabetic patients (5).

The patient exhibited a clinical picture of thrombocytopenia and DKA on first arrival and was monitored in the absence of any finding of haemorrhage. Haemorrhage was determined at computed tomography (CT) of the brain, performed due to headache, nuchal rigidity and papilloedema. We wish to emphasize that DKA can cause hypercoagulability, and activation of vascular endothelial and intracranial haemorrhage may ensue.

Keywords: Diabetic ketoacidosis, idiopathic thrombocytopenic purpura, intracranial haemorrhage

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Ultrasound Imaging for Prompt Monitorization of the Treatment Response in Patients With Multi-joint Involvement

The Editor,

Sir,

A 23-year old male patient who was previously diagnosed with peripheral spondyloarthritis (SpA) presented to our clinic. During physical examination, pain and swelling were observed bilaterally in his knee and ankle joints. In this patient, musculoskeletal ultrasound imaging showed grade II synovitis [greater than grade 1 to < 50% of the intra-articular area filled with colour signals representing clear flow] (1) at both regions (Fig. 1A–1B).

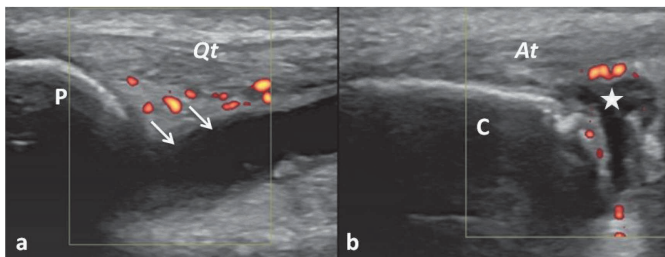


Fig. 1: Ultrasound imaging (longitudinal view with power Doppler) for knee joint synovitis (a) and enthesitis of the Achilles tendon (b). The suprapatellar (arrows) and retrocalcaneal (star) bursae are distended with anechoic effusion. Peribursal synovitis is observed at both sites. P, patella; C, calcaneus; Qt, quadriceps tendon; At, Achilles tendon

Since his current medical treatment (indomethacin 150 mg/day and sulfasalazine 400 mg/day) was considered insufficient, infliximab (5 mg/kg every 8 weeks) was commenced. On the 4th week of follow-up visit, the patient was found to be significantly improved both clinically and ultrasonographically (Fig. 2).

Ultrasound can visualize a great spectrum of pathologies regarding peripheral SpA involvement [*ie* enthesitis, bone

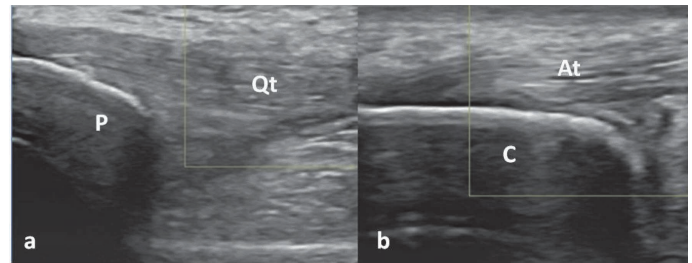


Fig. 2: A repeat imaging (4 weeks after treatment) reveals the disappearance of the effusions and the power Doppler signal at knee (a) and ankle (b). P, patella; C, calcaneus; Qt, quadriceps tendon; At, Achilles tendon

erosions, synovitis, bursitis and tenosynovitis] (2). Further, keeping in mind all of its advantages (handy, has high resolution, avoids radiation, provides dynamic imaging), ultrasound imaging of the joints and entheses can reasonably be incorporated as a complementary procedure into the overall assessment of disease activity and response to therapy (2–4). Apart from its great convenience for the clinician during patient follow-up (being as the ‘stethoscope’), the recent literature suggests that ultrasound images can be used as a visual biofeedback for the patients as well (5). Yet, it is not uncommon to have the patients comment and say, “*It is not burning any more*”, even while Doppler imaging is being performed.

Keywords: Anti-TNF treatment, enthesitis, ultrasound, synovitis

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