patients with dissemination of virus and seriously ill patients with herpes zoster ophthalmicus need intravenous acyclovir.[5] Possibly, the reversal of his immune-deficient state with stibogluconate resulted in the good recovery of our patient, even without treatment with acyclovir.

## Final diagnosis

Extensive cephalic herpes zoster with ipsilateral cerebellar involvement in a patient with visceral leishmaniasis.

**Keywords:** leishmaniasis; herpes zoster

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# Abdominal pain in a patient with falciparum malaria

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A 17-year-old native Central Indian man was hospitalised with constant abdominal pain accompanied by fever, chill, sweats and vomiting of 4 days duration. He was unemployed, and did not use tobacco, alcohol or illicit drugs. There was no history of biliary tract disease or lipid disorders, malaria or use of steroids or antimalarial drugs. On examination, he was found to be alert, febrile (38°C), jaundiced, and normotensive. Epigastric tenderness and mild generalised abdominal distension with reduced intestinal peristaltic sounds were noted. Laboratory studies showed: haemoglobin 9.8 g/dl, leucocyte count $11.8 \times 10^9$/l (polymorphs 73%), erythrocyte sedimentation rate 28 mm/h, and haematocrit 29%. Blood urea nitrogen was 7.2 mmol/l, creatinine 145 μmol/l, and serum electrolytes, cholesterol, and triglycerides were normal. Liver function tests showed a total bilirubin value of 31 μmol/l, and minimally raised hepatic enzymes (aspartate aminotransferase 84 U/l, alanine aminotransferase 70 U/l). Hyperamylasaemia (2132 U/l), hypocalcaemia (1.9 mmol/l), and hypoalbuminaemia (32 g/l) were detected. Urinary amylase levels were raised to 4011 U/l. A Leishman-stained peripheral blood smear demonstrated several ring-form trophozoites of *Plasmodium falciparum* (1.5% of parasitaemia). The electrocardiogram showed normal sinus rhythm. The red blood sickling, and antinuclear antibody tests were negative. An abdominal X-ray showed mild generalised ileus without air-fluid levels. Ultrasound demonstrated an oedematous, enlarged pancreas.

## Questions

1 What is the most likely diagnosis?
2 How does abdominal pain manifest in falciparum malaria?
3 List the infectious causes of acute pancreatitis.

## Answers

QUESTION 1

Acute pancreatitis due to falciparum malaria. The condition is characterised by severe pain in the upper abdomen and elevation of serum amylase. The diagnosis was supported by characteristic findings at abdominal ultrasonography and computed tomography of the abdomen which showed pancreatic oedema.[1]

QUESTION 2

Abdominal pain occurs in acute falciparum malaria, a great mimic of other diseases, as one of the localised symptoms with or without chest pain and arthralgias, as a component of the symptom complex of algid malaria and acute renal failure,[2] and rarely as a presenting manifestation of acute pancreatitis[3 4] (box 1).

QUESTION 3

Various infectious agents have been incriminated as aetiologic agents of acute pancreatitis. However, pancreatitis due to *P falciparum* is rarely encountered[3 4] (box 2).

## Outcome

The patient was treated conservatively with quinine, intravenous fluids, fasting, nasogastric suction, antibiotics, $H_2$-blockers, analgesics, and antacids. Over the next four days, he became afebrile, and anicteric; tenderness and distension of the abdomen decreased and vomiting stopped; the urinary amylase level decreased to 2411 U/l. On the fifth hospital day, clear liquids were begun by mouth. Ultrasound performed on the day of discharge (seventh hospital day) showed a marked decrease in pancreatic oedema.

Twenty-one days after hospitalisation, during follow-up examination, repeat ultrasound and CT scan showed a normal pancreas. The peripheral blood smear was negative for malarial parasite; serum albumin rose to 38 g/l, and calcium to 2.3 mmol/l. Blood haemoglobin, urea nitrogen, creatinine, bilirubin, and hepatic enzymes returned to normal values. The serum and urinary amylase levels were normalised. The patient is well two months after discharge.

**Causes of abdominal pain in falciparum malaria**

- localised symptom with or without chest pain and arthralgias
- component of symptom complex of algid malaria
- component of symptom complex of acute renal failure
- presenting manifestation of acute pancreatitis (rare)

**Box 1**

**Infectious aetiology of acute pancreatitis**

*Viral*
- mumps, hepatitis virus, coxsackie, Epstein-Barr, cytomegalovirus
- after immunisation with measles, mumps and rubella attenuated vaccine

*Mycobacterial*
- *M tuberculosis, M avium-intracellulare*

*Bacterial*
- leptospira, *Salmonella tophi*, mycoplasma, *Staphylococcus aureus*

*Fungal*
- *Aspergillus* spp, *Candida* spp, *Cryptococcus neoformans, Pneumocystis carinii*

*Parasitic*
- *Opisthorchis sinensis, Ascaris lumbricoides*
- *Toxoplasma gondii*, cryptosporidium, microsporidium, *Plasmodium falciparum*

**Box 2**

## Discussion

From mild disease to multi-organ failure and sepsis, acute pancreatitis is a disorder that has numerous causes, an obscure pathogenesis, few effective remedies, and an often unpredictable outcome.[5] Falciparum malaria causes many different syndromes.[6] Abdominal pain occurs in falciparum malaria as a presenting manifestation of acute pancreatitis.

Acute pancreatitis as a complication of falciparum malaria has been reported infrequently in the English literature.[3 4] Pancreatitis occurring in *P falciparum* infection was not mentioned in a recent review of pancreatitis,[5] or in an exhaustive list of aetiological agents of pancreatitis.[1] At least some of the current major medical texts,[7–9] do not include *P falciparum* as an aetiological agent of pancreatitis.

The accumulation of parasitized erythrocytes (occasionally causing thrombosis and infarcts) is more common in the small vessels of the spleen, liver, bone marrow, and brain, than in the kidneys, small intestine, pancreas, heart and lungs.[10] However, the erythrocytic part of the life-cycle of *P falciparum*, known as sporogony, occurs in the deep visceral capillaries throughout the body.[8 9 11 12]

In fatal *P falciparum* infection, autopsy studies demonstrated that the small blood vessels of the pancreas were packed with parasitized red cells and rosettes.[13] The parasitized erythrocytes bind to receptors on the endothelial cells by the formation of knobs (electron-dense structures),[7 12] and cause obstruction of capillary blood flow.[7 9 11] Tumour necrosis factor released from macrophages may be directly toxic to endothelial cells, favouring the accretion of thrombin, which may make the endothelial cells more adhesive for the surface of parasitized erythrocytes.[14] These vascular changes lead to anoxic damage to diverse organs, thus accounting for the protean clinical manifestations of falciparum malaria.[12]

This case report presents a patient with confirmed pancreatitis in the setting of acute falciparum malaria. While acute pancreatitis might be a rare cause of abdominal pain and vomiting in *P falciparum* infection, its serology is routine.

**Summary / learning points**

- acute pancreatitis can occur as a complication of falciparum malaria
- persistent and severe abdominal pain may be presenting manifestation of acute pancreatitis in *P falciparum* infection
- serology of pancreatitis should be ordered routinely in falciparum malaria
- pancreatitis should be considered in the spectrum of disease associated with *P falciparum* infection

**Box 3**

The list of identifiable causes of pancreatitis is growing and pancreatitis should be considered in the spectrum of disease associated with *P falciparum* infection.

### Final diagnosis

Acute pancreatitis complicating falciparum malaria.

**Keywords:** acute pancreatitis; malaria; *Plasmodium falciparum*

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# Metabolic acidosis

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A 60-year-old woman on haemodialysis secondary to end-stage renal disease, was brought to the emergency room with recent onset of weakness, decreased consciousness and shortness of breath. She has a history of diabetes mellitus, hypertension and coronary artery disease.

Her medications on admission to the emergency room were: hydralazine, quinidine sulphate, catapress patch, lorazepam, dipyridamol, propulsid, nifidepine XL, metformin HCl, promethazine, and zolpidem tartrate. Physical examination revealed the following: respiratory rate 26 breaths/min and shallow, pulse 104, blood pressure 120/70 mmHg, afebrile. Physical examination was otherwise remarkable for only mild abdominal tenderness. Laboratory investigation revealed: sodium 141 mmol/l, $HCO_3$ 9 mmol/l, blood urea nitrogen 36 mg/dl, creatinine 9.7 µmol/l, glucose 172 mmol/l, amylase 168 mmol/l, alanine transaminase 19 IU/l, serum ketones 1:4 dilution, lactic acid 17.4 mmol/l; whole blood count was normal. Arterial blood gas: pH 6.88, $pCO_2$ 9.5, $pO_2$ 153, $HCO_3$ 1.8, $SaO_2$ 97.2 on room air.

During her stay in the emergency room, the patient became more lethargic and tachypnoeic, eventually requiring intubation and mechanical ventilation.

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### Question

What is the most likely diagnosis: sepsis, diabetic ketoacidosis, drug-related side-effect, uraemic acidosis, or salicylate overdose?