day 7 doxycycline was added. Patient had defervescence of fever on the 13th day and subsequently was discharged with normal renal functions on day 15. The follow up echo and chest X-ray were normal.

Comment. Falciparum malaria is one of the most common parasitic diseases causing much morbidity and mortality in the tropics. Pericardial involvement in malaria is rare, described only in a few reports worldwide [2,3]. It is usually in the form of pericarditis. This is an unusual case where a patient of documented falciparum malaria and acute renal failure not only developed periodically but also pericardial effusion. While the acute renal failure improved, the pericardial involvement worsened and he developed tamponade, which responded to anti-malarial therapy.

The issue is whether the effusion was due to malaria or due to uraemia per se. The indirect evidence for the association with malaria include, the development of pericarditis and subsequent effusion during the recovery stage of acute renal failure. The azotaemia was for a short duration and hence unlikely to be solely responsible for development of pericarditis and effusion. There was resolution of effusion with clinical defervescence of malaria. The patient had no peripheral oedema and thus fluid overload is also not a likely contributing factor for effusion. The confirmatory evidence for this association comes from the isolation of the falciparum antigen from the pericardial fluid. We are not aware of any previous published case in literature reporting this association.

The mechanism of the development of pericardial effusion is not very clear. One possible explanation may be the locally elevated cytokine levels. Day et al. [4] have shown that cytokines produced locally in particular tissue during malarial infection may be regulated by the degree of regional parasitic sequestration. Probably regional sequestration of parasites in pericardium may explain the isolated pericardial effusion in our case. Documentation of parasitic antigen in the pericardial fluid confirms the association of malaria and pericardial effusion.

We conclude that development of pericardial effusion may be seen with falciparum malaria. This is crucial, as tamponade if not diagnosed and managed promptly may be a potentially fatal complication, in what is otherwise a curable disease.

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Acute renal failure and oligohydramnios induced by magnesium dypirone (metamizol) in a pregnant woman

Sir,

A 21-year-old equadorian woman, previously healthy, was admitted to our hospital because of acute renal failure in her 30th week of pregnancy. Ten days before admission, she developed acute back pain with no urinary or intestinal symptoms. Without medical advice, she started treatment with oral magnesium dypirone (Nolotil®), 1.5–3 g/day for 10 days. Physical examination revealed a pale woman with
generalized cutaneous rash and oedema. Supine blood pressure was 110/70 mmHg and abdominal palpation revealed that the size of the uterus corresponded to gestational week. Laboratory studies reported haemoglobin 8.9 g/dl, haematocrit 26%, creatinine 3.7 mg/dl, urea 117 mg/dl, white cell count 10 000 µl, with eosinophils 200 µl and platelet count 190 000 µl. Serum glucose 90 mg/dl, creatinine 3.7 mg/dl, urea 117 mg/dl, total proteins 5.6 g/l, bilirubin 3.2 mg/dl, AST 241 IU/l, ALT 9 IU/l, LDH 414 IU/l, CPK 111 IU/l and total alkaline phosphatase 311 IU/l. Antinuclear antibodies, c-ANCA, p-ANCA and anti-MBG were negative. Tests for serum antibodies to hepatitis C virus, hepatitis B surface antigen and anti-HIV were also negative. Serum levels of complement C3 and C4, serum immunoglobulins and TSH were normal. A renal ultrasound revealed normal sized and symmetric kidneys. Blood smear did not reveal schizocytes or eosinophilia. Urinary protein excretion was 0.8 g/24 h and the sediment revealed three red blood cells and leucocyturia. Obstetric ultrasound scan showed oligohydramnios with an amniotic fluid index of 2 (normal value 5–24.0 cm [1]). With parenteral fluids and biochemical data as well as after a detailed history, tubulointerstitial nephritis was made according to clinical and laboratory findings as well as the increased creatinine. The mechanism of oligohydramnios with these drugs is unknown. It could be related to fetal oliguria due to the increase of arginine vasopressin in the fetal collecting duct induced by prostaglandin inhibition, as has been shown in animal models [7]. In our patient, the dose of self-administered magnesium dyprone was high, and probably part of the drug entered the fetal circulation transplacentally.

Magnesium dyprone can induce two different forms of acute renal failure: (i) reversible renal ischaemia secondary to inhibition of prostaglandin synthesis, appearing 3–7 days after initiation of therapy when drug levels inhibit prostaglandin synthesis [5]; and (ii) acute tubulointerstitial nephritis [9]. In our patient, the diagnosis of acute tubulointerstitial nephritis was made according to clinical and biochemical data as well as after a detailed history record, but both forms of renal disease associated with magnesium dyprone were probably present. Moreover, withdrawal of dyprone was associated with a dramatic improvement of renal function and the oligohydramnios, as has been described previously [6].

We conclude that magnesium dyprone may induce reversible acute renal failure and oligohydramnios in pregnancy. It should be used with caution, with monitoring of renal function and amniotic fluid volume.

Conflict of interest statement. None declared.