Table 1: Relationship between ABO blood groups and antibodies against *Toxoplasma gondii*.

Blood group	No. investigated	Percent	No. with positive test for *T. gondii*	Percent
A	835	47.7	190	22.8
AB	61	3.4	11	18
0	729	40.8	147	20.2
A + AB + 0	1.625	90.9	348	21.4[a]
B	163	9.1	47	28.8[a]

[a] $p < 0.03$ (chi-square, two-tailed).

gastrointestinal tract – down to the proximal part of colon – express ABH antigens on their surfaces (4). Thus, if the B antigen is a receptor of importance for the invasion of the *Toxoplasma gondii* protozoan, it is present on many cells lining the gastrointestinal tract.

As to why the protozoans do not also invade the erythrocytes (as the malarian plasmodia do), which express blood group antigens much more abundantly than do the intestinal epithelial cells, it might be relevant to focus upon some recent findings that show that ABH antigens on erythrocytes are carried exclusively on the so-called type 2 chains, while on gastrointestinal epithelial cells they are carried primarily on the type 1 chain (5). Consequently, the cellular receptors might be different. Whatever the mechanism(s) might be, however, more investigations should be carried out in order to ascertain the relationship between the presence of blood antigen B and an invasion made by the protozoan *Toxoplasma gondii*.

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Subdiaphragmatic Gas in Hepatic Hydatid Disease

The radiographic finding of subdiaphragmatic gas due to hepatic and subphrenic abscess is well recognised (1). It has not previously been reported in association with infected hepatic hydatid diseases.

A 48-year-old diabetic Malaysian woman was admitted to hospital after collapsing. On admission the patient was drowsy and reported a history of three days of general malaise with diarrhoea and leg and abdominal pains. She was pyrexial (39 °C) with right hypochondrial tenderness. X-ray demonstrated gas under the right hemidiaphragm. Two distinct fluid levels suggested more than one collection. Ultrasound showed a thick-walled cavity containing gas and fluid in the right hepatic lobe, in apparent continuity with a second collection located between the liver and the diaphragm. Laboratory investigations revealed haemoglobin of 10 g/l and a white cell count of 17.6×10^9/l. Renal function (urea 13.6 mmol/l, and creatinine 268 μmol/l) had not altered significantly since her last Diabetic Clinic consultation. Previously normal liver function tests showed raised hepatic enzymes and total bilirubin.

A presumptive diagnosis of a hepatic abscess was made, and intravenous antibiotic therapy was initiated, consisting of 500 mg of ampicillin four times a day and 60 mg of gentamicin three times a day. Blood cultures yielded an ampicillin-sensitive organism subsequently identified as *Streptococcus bovis* biotype II. The patient's geographical history raised the possibility of hydatid disease, and although the presentation had been atypical, therapy with 400 mg of albendazole twice daily was started.

As the patient's condition precluded surgery, the lesions were drained percutaneously. Four hundred milliliters of pus were aspirated, and Gram stain showed hydatid hooks and scolices with gram-positive cocci in chains and pleomorphic gram-variable bacilli.

Cultures yielded heavy growth of *Streptococcus bovis* biotype II and *Bacillus pumilis*. Serological investigations confirmed the diagnosis of hydatid disease (ELISA positive, complement fixation titer 1 : 8). Despite continued appropriate antibiotic therapy, the patient's condition deteriorated as she developed acute on chronic renal failure. She died six days after admission.

At autopsy there was generalized peritonitis with a pelvic collection of pus. The liver exhibited serositis. Within the right hepatic lobe a 10 cm cavity contained pus and a collapsed hydatid sac. Immediately above was a 6 cm diameter abscess from which arose two sinuses tracking through the hepatic capsule superiorly. Two separate cavities (3.5 cm and 4 cm diameter) containing membranous sacs were present in the left liver lobe. One was filled with straw-coloured fluid and the other pus. There was no subphrenic collection. The diaphragm was intact and although small bilateral, straw-coloured, sterile, pleural effusions were present, there was no evidence of lung involvement.

Infection is one of the commonest complications of hepatic hydatid disease, with large series recording suppuration as the presenting feature in 8–12.7 % of cases (2, 3). However, microbiological findings are poorly recorded, with only a few case reports detailing organisms (4, 5). Plain abdominal radiography in cases of hepatic abscess may reveal gas fluid levels, indicating infection with gas-forming organisms (1). However, an extensive literature review yielded only a single report of intracystic gas in an hepatic hydatid lesion (6). The microbiological details were not recorded, but the gas was attributed to infection.

Figure 1: *Left lateral decubitus x-ray showing two separate perihepatic fluid levels.*

In our case the presence of an infected hepatic hydatid cyst was proven. The atypical presentation and the isolation of organisms not normally regarded as gas-producers led us to question the origin of the the subdiaphragmatic gas and to consider the possibility of diaphragmatic rupture with lung involvement. Hepatopulmonary hydatid disease occurs in 1–9.5 % of cases and can be difficult to diagnose preoperatively, even using sophisticated techniques (4). It has been reported to occur in the absence of demonstrable diaphragmatic lesions (7). Although iatrogenic pneumoperitoneum has been employed diagnostically in this condition, there is no record of subdiaphragmatic gas as part of the disease process per se. Post mortem examination in our patient excluded lung and diaphragm involvement, and we therefore conclude that the gas was infective in origin. This case is obviously extremely unusual but stands as demonstrating an association between subdiaphragmatic and intrahepatic gas and infected hepatic hydatid cysts.

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