Clinical

Hepatitis B infection following deployment to Angola

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Abstract
We describe the clinical illness and long-term follow up of two British personnel who acquired hepatitis B infection during a 3-month UN mission to Angola. Medical officers need to be familiar with this viral illness, which may present in military personnel after any exercise in or deployment to the tropics or subtropics.

Key words: UN, infectious disease, hepatitis B, Angola, immunisation.

Introduction
In May 1995 a force of 642 British personnel deployed to Angola for 3 months, as the British contribution to the United Nations Angola Verification Mission (UNAVM). The British deployment was codenamed Operation Chantress and its purpose was to establish an enduring logistics infrastructure to support the later deployment of a 7,200-strong UN force.

In the 6 months prior to deploying the British personnel at readiness to move were offered monovalent vaccination against hepatitis A, meningitis (polysaccharide vaccine against Neisseria meningitidis serogroups A and C) and typhoid fever. These mission-specific vaccines supplemented the standard UK military immunisation regimen at the time, which comprised BCG, and polio, TABT (inactivated tetanus toxoid combined with vaccines against typhoid fever and against paratyphoid fever A and B) and yellow fever vaccines. The deploying personnel were prescribed chloroquine 300 mg once weekly and proguanil 200 mg once daily, as malaria chemoprophylaxis.

The deployed British force consisted of a logistic battalion group, based on 9 Support Regiment RLC. The group included elements of 13 other Army units or detachments, and an RAF air movements cell. During the deployment, the British logistic battalion group managed, built and maintained transit accommodation, produced clean water, unloaded UN ships, categorised and stored large quantities of UN stores, distributed rations, and moved stores by convoy along newly opened roads.

UNAVM was a difficult mission owing to the huge size of Angola (Figure) and the fact that intense mistrust persisted between the previously warring factions. Banditry was rife, most roads were closed and the threat of mines was ever present. Notwithstanding these dangers, there were no fatalities or significant injuries in the British force, and air recovery to the UK took place successfully on 9 August 1995.

Some weeks later, two members of the Operation Chantress force developed hepatitis B infection. We describe the course of their clinical illness, and their subsequent follow up.

Case A
The first case was a 35-year old serviceman who had a static role with the
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infantry guard company, based at the port of Lobito. 5 weeks after returning to UK from Angola he presented to his unit medical officer with a 4-day history of loss of appetite, lethargy, loin discomfort and nausea. Clinical examination was unremarkable, urine and MSU were normal and a viral illness was diagnosed.

On review 3 days later he was obviously jaundiced, and now complained of malaise, vomiting and passing dark urine. On abdominal examination he had a mildly tender right upper quadrant and a palpable liver. In addition, abdominal ultrasound scan showed an enlarged spleen. He was admitted to the local civilian hospital where he was found to have a raised total bilirubin of 131 Îmol/l and a hepatic pattern of liver function test results (ALT 228 U/l, AST 183 U/l, alkaline phosphatase normal). Malaria films were negative and hepatitis serology was HBV positive with HBsAg >10 000 WHO units. He was also HbcAg positive and HBeAg positive. A diagnosis was made of acute hepatitis B. He was discharged after two days to rest at home, with outpatient follow up.

On review a month later he was still jaundiced and still HBV positive, though now with anti-HBeAG, indicating reduced infectivity. His total bilirubin had fallen to 46 Îmol/l, his ALT to 145 U/l, and his AST was now within the normal range.

He continued with monthly outpatient follow up, and 3 months later he had seroconverted and was HBsAg negative. He was no longer considered an infection risk, and was discharged from the clinic.

The patient denied any sexual contact while in Angola, and had no involvement with blood spillage, emergency first aid or contact sport.

After his acute episode of hepatitis B his general health remained good. He had no relapse of his viral infection, and he completed his military service in August 2000.

Case B
The second case was a 24-year old driver. He too was stationed at Lobito but in contrast to Case A he travelled widely throughout western Angola, and frequently spent the night away from his home base. 7 weeks after returning to UK from Angola he presented to his unit medical officer with malaise, fever and jaundice.

He was admitted to a UK military hospital where he was found to have significantly raised hepatocellular enzyme levels (ALT 326 U/l, AST 173 U/l), with slightly elevated alkaline phosphatase. His prothrombin time was prolonged and urobilinogen was present on urinary analysis. HBsAg and HBeAG were both positive and hepatic imaging showed a moderately inflamed liver. No malaria parasites were seen on thick and thin blood films.

He was diagnosed as having acute hepatitis B and was treated symptomatically as an inpatient for one week, then discharged to unit sick leave and light duties. During subsequent follow up his laboratory parameters all returned to normal levels, although HBsAg persisted in the serum for 6 months. A year following his infection, he had returned to normal health.

This second patient admitted to putting himself at risk while in Angola. He had sex with an African prostitute on two separate visits, and was a witness to a road traffic accident where he administered first aid to an African child who was bleeding profusely, with the result that he became covered with the child's blood.

He had no relapse of his infection, and at time of writing (September 2008) he is still in full-time military service and is classified as fully deployable.

Discussion
Hepatitis B is a global public health problem. Approximately 400 million people are chronically infected and in the year 2000 around 620 000 people died worldwide from the disease (1). Of these, 6% died from acute hepatitis B, and 94% died from cirrhosis or hepatocellular carcinoma, secondary to infection with hepatitis B virus (HBV) (2). Carriage rates of HBV in the population show wide geographical variation, and are notably high in the Far East and in
southern Africa (10–20% carriage) and low in northern Europe and North America (<1% carriage) (2).

Man is the only reservoir of HBV. Transmission is from person to person by a number of blood-borne routes, summarised in the Table. The incubation period is 3–6 months. Specific HBV genotypes can be identified using PCR techniques. Genotypes B and C appear to have more aggressive disease that responds less well to viral therapy (3).

Clinically, hepatitis B is indistinguishable from other causes of viral hepatitis (4). After a non-specific prodromal illness with fever and malaise, jaundice appears and the fever stops.

In 90–95% of adults, full recovery from acute HBV infection occurs within 6 months, and is characterised by the appearance of antibodies to viral antigens (3). The remaining 5–10% of adults develop a chronic infection which typically persists for life, and is characterised by detectable hepatitis B surface antigen (HBsAg) and/or detectable e antigen (HBeAg). These patients become chronic carriers (5).

Approximately 25% of all patients with chronic hepatitis will progress to cirrhosis and about 20% of those with cirrhosis will go on to develop hepatocellular carcinoma (6). Between 1971–1990 there were 36 cancer registrations for cancer of the liver (ICD-8: 155) among male UK Armed Forces personnel, and it is likely that some of these cancers will have been long-term sequelae of primary HBV infection.

There is no specific treatment for acute hepatitis B and for chronic infection the treatments are still limited, with no drug able to eradicate HBV completely (3). Commonly-used agents include alfa-interferon, lamivudine and adefovir (7).

Hepatitis B is prevented through vaccination. The primary immunisation schedule for adults consists of three doses of vaccine given at 0, 1 and 6 months. For more rapid protection this can be accelerated to 0, 1 and 2 months (or to 0, 7 and 21 days), in which case a 12-month booster dose should be given as a fourth dose (8).

The two cases described here illustrate the common incubation period, the normal presenting features and the typical clinical course of hepatitis B infection. Medical officers need to be familiar with this ubiquitous viral illness, which may present in military personnel after any exercise in or deployment to the tropics or subtropics.

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Table: Common routes of transmission of hepatitis B virus (adapted from Reference 4)
Declaration of Interests
The authors have no conflicts of interest.

References