EDITORIAL

Why viral hepatitis?

Readers may well ask this question upon realising that this issue of the *SAMJ* is entirely devoted to viral infections of the liver. Indeed, some may be moved to discard it in the belief that these infections are caused by esoteric viruses which they need not know much about. For those tempted to do so we would counter that almost every South African adult has evidence of previous exposure to the hepatitis A virus; roughly 60% have been infected with hepatitis B and between 1% and 10% are infectious carriers of this virus; 0,16 - 0,75% have evidence of hepatitis C infection and as many as 8 - 14% hepatitis E.[1-5] At this point a few diehards may argue that hepatitis is usually subclinical and that when it occurs it belongs to the realms only of general practitioners, paediatricians and physicians. Wrong again! The hepatitis viruses are a major cause of morbidity and mortality. Apart from classic hepatitis, which may progress to acute liver failure, they can cause cirrhosis, carcinoma of the liver, glomerulonephritis, polyarteritis nodosa, peripheral neuritis, cryoglobulinaemia and bone marrow aplasia.[6] They not infrequently occur in pregnant women, where some, like hepatitis E, may result in 20% mortality[7] and others like hepatitis B may be transmitted from mother to child during delivery.[8] They may be confused with surgical conditions and apart from being an important cause of bleeding oesophageal varices may pose a far greater threat to the surgeon than HIV infection.[9] Anaesthetists will attest to the fact that such patients often require special care. The fact that many of these viruses are transmitted as a result of poor socio-economic conditions and the advent of vaccines for hepatitis A and B have made these diseases of great interest to community health physicians.[10,11] Those responsible for the provision and funding of health care will no doubt be interested in better ways of diagnosing or preventing these conditions, the optimal frequency of often expensive liver function and serological tests, and cost/benefit analyses of various means of preventing these diseases. Hepatic encephalopathy may bring these patients to the attention of psychiatrists, while accompanying rashes and the intractable itch of cholestasis may call for a dermatologist. Finally, ethicists, workmen's compensation commissioners and medicolegal buffs may have to pronounce their verdicts concerning the transmission of hepatitis from patient to health care provider and vice versa. If we've left you out read no further!

The identification of the hepatitis B virus marked the beginning of a new era in virology. From the apparently humble observation that serum from an Australian Aborigine contained an antigen which cross-reacted with serum from persons who had required multiple infusions of blood products and the profound insight which led to the hypothesis that this Australian antigen was related to a hepatitis virus[12] flowed advances in immuno-electron microscopy, molecular biology, immunology and epidemiology which allowed the identification of hepatitis viruses A to E. The results of these studies led to increased knowledge of the clinical syndromes associated with each virus, to relatively simple methods for their exact diagnosis, to advances in the therapy of chronic hepatitis[13] and finally to vaccines for viruses B and A.

Readers may well next ask 'why viral hepatitis at this time?'. As South Africa is being born to the world of democracy, unique opportunities are becoming available as this country's health system is transformed into one which emphasises a primary health care approach.[14] In particular this policy framework is ideal for an immediate concerted government-led programme for the control of hepatitis B (and therefore hepatitis D as well) in the short term and eradication of hepatitis B in the medium to long term. Such a control programme, which is now possible, requires a three-pronged approach:

1. A policy of universal childhood vaccination against hepatitis B introduced by government. The political will to ensure implementation of this policy is even more important and the anticipated district-level service[15] may be well placed to make hepatitis B widely accessible as part of a basic package of primary health care services.
2. An information system is essential to manage a control programme. Discussions are currently underway to create a new health information system, including infectious disease surveillance, in South Africa.[16] The problems experienced in hepatitis B data[17] need to be addressed, while both laboratory and notification sources of data need to be developed to serve control efforts and programme management needs such as the identification of target areas where vaccination coverage may need to be improved.
3. An ethos which promotes the notion of public health and clinical medicine as parts of a continuum, where each supports and strengthens the other, is essential. This ethos, which is now gaining prominence with the participation of medical schools in the development of schools of public health,[18] is well illustrated in the context of hepatitis B control, e.g. counselling of hepatitis B patients to use condoms, introduction of single-use disposable syringes and the importance of universal precautions for potentially infectious body fluids. Building on this, control measures targeted at household contacts and sexual partners identified through patients presenting with clinical hepatitis will become increasingly important as vaccine coverage reduces childhood transmission. The value of these steps in concomitantly preventing HIV infection is obvious.

We believe it is timeous to rekindle an interest in viral hepatitis lest the opportunity to control the principal viral cause of liver disease passes us by. A childhood vaccination programme together with a health information system to support the implementation of this programme, with efforts linking clinical care and public health measures as the essential ingredients, are now becoming realities. This will make control of hepatitis B a realisable objective.

This issue of the *SAMJ* contains articles on the clinical and laboratory diagnosis of viral hepatitis, a review of each of the viruses, the diagnosis and treatment of chronic hepatitis, the relationship of hepatitis B and C to hepatocellular carcinoma, the role of blood transfusion services in limiting spread of these diseases by blood and blood products, the medicolegal consequences of hepatitis, and, perhaps most important for our proud new nation, an article on prevention which holds the promise of eradication not only of acute hepatitis but of many cases of chronic disease including cirrhosis and hepatocellular carcinoma.

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ARTICLES

Clinical and biochemical features of acute viral hepatitis

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Viral hepatitis is a major cause of mortality and morbidity worldwide. Acute viral hepatitis, although a generalised systemic infection, presents with clinical manifestations relating directly to inflammation of the liver with hepatocellular dysfunction and jaundice.

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The most important causes of acute and chronic hepatitis are the five hepatotrophic viruses, hepatitis A, B, C, D and E. There are 2 other, as yet unidentified, hepatitis viruses, F and G.

The clinical features of acute hepatitis caused by these hepatotrophic viruses are similar and only minor features of the clinical disease, together with the incubation period and epidemiological history, help to distinguish the different acute hepatitides. Specific diagnosis requires serological testing.

The clinical severity of acute hepatitis varies. Most infections are asymptomatic, subclinical or anicteric with mild gastro-intestinal symptoms only. Occasionally infection results in acute fulminant hepatitis with an associated high mortality.

Clinical features of acute viral hepatitis

A full clinical history is important. Particular emphasis should be paid to a recent history of travel, high-risk sexual practices, blood transfusions and the use of drugs.

Symptoms

The most common symptoms experienced during the prodromal phase include: malaise and fatigue, myalgia, anorexia, nausea and vomiting, right upper quadrant discomfort and fever.[1] Between 5% and 15% of patients with hepatitis B may develop a serum-sickness-like syndrome. Other extrahepatic manifestations, which may occur in acute viral hepatitis, include urticaria and angioneurotic oedema, arthritis, vasculitic and renal lesions, myocarditis and cardiomyopathy, pancreatitis and CSF abnormalities.[2] The prodromal symptoms usually last a few days to 2 weeks and are followed by the development of jaundice. However, jaundice may occur in the absence of any prodromal symptoms. Frequently, the systemic symptoms improve with the development of jaundice. Occasionally there is a prolonged cholestatic phase with associated pruritus. If there are minimal prodromal symptoms it is often difficult clinically to distinguish this form of viral hepatitis from extrahepatic cholestasis or drug-induced cholestasis.

During recovery, the nausea improves and the appetite returns. General malaise may persist for some time and relapses associated with an exacerbation of symptoms and of jaundice occur in up to 5% of patients. These may be precipitated by too early a return to work or vigorous exercise programmes.

Signs

The most common physical signs in acute hepatitis are jaundice, right upper quadrant tenderness and mild hepatomegaly. Splenomegaly occurs in 5 - 10% of patients and lymphadenopathy is occasionally seen.[1] Skin rashes may be present and in children with acute hepatitis B, a papular acrodermatitis involving the arms, legs and face may be found.

Fulminant viral hepatitis occurs in a small proportion of patients. This is associated with the development of