HEPATITIS A and B

Prof Julius Ogeng'o

University of Nairobi

E-mail : jogengo@uonbi.ac.ke

Hepatitis A and B — A real threat to Life

By Dr. Julius A. Ogeng'o

Nancy had been to my house twice before — the first time to see our new born baby, and the second time to drop my Insurance policy certificate. They lived two flats from us and she had gone to school with my kid sister. So, when Betty rang to say Nancy was very ill, my memory of this 22 year-old lovely girl was quite fresh. Yes, she had to be very sick for me to be called. I advised Betty to drive her to the casualty department and I would be there shortly.

When I got there, a huge crowd, which my arrival dispersed, surrounded Nancy. "Thank you for coming Doctor. I have been feeling hot for over a week now, general weakness, feeling like vomiting, loss of appetite, a vague abdominal ache and my urine has been deep yellow, since two days ago. This morning I had convulsions and auntie Betty thought I had cerebral malaria but other things are equally important, occasionally with a lot less predictable outcome." She summarised.

I asked her a few relevant questions and then examined her. She had lost some weight and her eyes, lips and tongue had a yellow tinge. She was mildly dehydrated and obviously had high temperature. The liver was enlarged and painful to touch. I immediately suspected viral hepatitis and admitted her to the female medical ward. When my diagnosis was confirmed by laboratory findings, I settled her symptoms and three days later, I discharged her to be seen in the liver clinic as an out patient. I did not see her again, as while she was away on holiday, I was transfered to a surgical ward.

One afternoon, as I was passing through the liver clinic, to see a friend of mine, a weak voice said, "Good afternoon doctor." When I turned, it took me sometime to place Nancy. She had lost weight, was dehydrated but had swollen legs and looked quite yellow. On Sister Maneno's request, I accepted to see her and admitted her to the surgical ward. Two days later, she underwent successful surgery but unfortunately, on the third post — operative day, she went into frank liver failure and died.

The recorded history of viral hepatitis finds its origins in accounts which date from ancient times. Nonetheless, serum was accurately documented following the yellow fever vaccine in Brazil, in the 1930s. At the start of the second world war, there was an epidemic of serum hepatitis among American soldiers given the yellow fever vaccine; and those young Americans overwhelmingly exposed to epidemic infectious hepatitis in North America, and Italy. These led to the first sizeable burst of knowledge.

Today, viral hepatitis is well known all over the world. This viral infection of the liver is worldwide in distribution but is more prevalent in tropical than temperate regions, and especially in the developing countries, where material standards are lower and blood screening facilities not as good.

Dr. Ogeng'o: Those who receive repeated blood transfusions are at higher risk of getting Hepatitis B.

Several viruses affect the liver but the best known ones include Hepatitis A, Hepatitis B; Hepatitis Non A-Non B; Cytomegalovirus; Infectious mononucleosis and even measles virus. In this article, I shall discuss the first three and in the next; liver failure, a commonly lethal outcome of viral hepatitis. Before I go into the details of the disease, let me highlight very briefly the main functions of the liver, the principal organ affected by the viruses.

The liver is the second largest organ in the body, found in the right upper abdomen, just below the diaphragm. It is responsible for the following major functions;

* Protein synthesis. Apart from the proteins for its own use, the liver produces albumin, a protein

responsible for keeping the blood within the blood vessels and prothrombim and fibrinogen, which are important in blood clotting.

* Secretion of bile — the yellowish bitter liquid which is poured into the intestines where it facilitates the digestion and absorption of fats; and thus all the important fat soluble vitamins like A, D, E and K.
* Storage of fat and carbohydrate, which material it releases for body use during starvation. It is also able to convert protein and fat product into glucose for use in energy production.
* Inactuating, usually by breakdown, of drugs and other material, which are either poisonous (toxic) to the body or are not needed. Let us now turn to the diseases of the liver.

Hepatitis A

This is a highly infectious liver disease caused by hepatits A virus, spread in faeces. It is worldwide in distribution and has an incubation period of 2-3 weeks. Infected persons excrete the virus in faeces 2 weeks before and 5-7 days after onset of the illness. It affects children more and is promoted by poor sanitation and overcrowding. Outbreaks are occasional and are usually traced to infected milk, water and shell fish.

It may however be spread by blood transfusion or homosexual activity though these are uncommon modes. In a community, the sources of the virus are usually either persons incubating the disease, or those suffering from it. No carrier states has been described.

Although the disease mainly affects the liver, it is generalised, affecting the heart, joints, pancrease, spleen and the Gastro intestinal tract. The disease presents with chills, headache, general weakness, loss of appetite, distaste for cigarettes, feeling like vomiting; vomiting and diarrhoea and abdominal pain. The urine gets darker as the mucous membranes i.e. tongue, lips, eyes and eyelids get yellow. This is called jaundice. The stools get pale and then the liver enlarges and is painful to touch. In children, especially, the lymph glands and spleen also enlarge.

No specific treatment is known, and the diagnosis rests on clinical judgement supported by urine, stool and blood tests. Within 3-6 weeks, there's usually spontaneous recovery. Doctors may, however, advise bed rest, plenty of sweet fluids and low protein diet. Antibiotics, when given are usually to clear the gut of bacteria, whose metabolic products may be bad for the sick liver. Almost all patients recover fully though 5-15 per cent may show relapse during the recovery phase. This relapsing hepatitis also subsides spontaneously. There may be debility for 2-3 months, associated with prolonged general weakness, poor appetite, nausea and abdominal discomfort but again, usually these eventually subside. Death is uncommon in young adults, claiming about 0.2. per cent but with advancing age, death increases. As is expected, other intercurrent illnesses are bound to worsen the outcome.

Hepatitis B

This has a longer incubation period, varying between 6 weeks and 6 months. Man is the only important reservoir of infection, although it has been described in chimpanzees and a few domestic animals. It is predominantly blood borne, infection being transmitted by blood transfusion, injections, and innoculations, tribal sacrifices, ritual circumcision; insect vectors etc. In a few cases however, infection could be spread sexually, or through the air or in contaminated food and drink. The virus has been described in other body fluids such as tears, saliva and urine, but transmission by related modes is unknown! About 20-30 per cent of children born of infected mothers develop hepatitis B positivity.

While it is possible to transmit the virus to the unborn baby through the placenta, the virus may be transmitted at or soon after birth. It is important to reiterate here that with a few exceptions, man is the only important source; those who are suffering from the disease, those who are incubating it, and asymptomatic carriers. The latter group constitute the most dangerous population in epidemiology of this disease. Indeed, there are over 200 million infectious carriers worldwide and a further 250,000 new cases are diagnosed yearly. Those who receive repeated blood transfusions are at higher risk and coagulation factor concentrates which cannot be sterilised are a particular hazard and account for the higher incidence of hepatitis in Haemophiliacs.

The presentation is the same as that of hepatitis A, but is a lot more severe. In addition, there may be transient rashes, including urticaria; and joint affliction. It has a higher death rate which depends on the seriousness of the virus, the age of the patient and the presence of any underlying disease. Surprisingly, some individuals with no history of acute hepatitis and lacking clinical evidence of liver disease are chronic carriers of the virus. These are usually detected when their blood is screened.

Diagnosis is based on good history such as of travel, blood transfusion, acupuncture; tattooing,

To page 15