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# VIRAL HEPATITIS IN HEAMODIALYSIS UNIT

#### THESIS

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# INTRODUCTION AND AIM OF THE WORK

#### INTRODUCTION

Viral hepatitis has been a particularly interactable problem among a number of infections which may complicate dialysis, it ranked third, 2-3% after septicaemia and bacterial pneumonia, in the infective causes of death in patients on regular dialysis treatment (Parson et al, 1971, Marion and Tonkin, 1972).

In patients with chronic renal failure undergoing regular dialysis, the liver is exposed to an abnormal chemical environment in which the metabolic functions are severely affected.

The high incidence of hepatitis in dialysis patients is related to the deranged metabolism and nutritional impairment of the liver and to the impaired immunological mechanisms of the uremic patients (Young and Parsonl 1970).

Dialysis patients aften require regular blood transfusions from time to time, the blood is frequently split during such procedures as manipulating the arterio-Venous shunt or fistula. (Sherlock, 1972).

Dialysis related hepatitis may be caused by many viruses including hepatitis A virus, hepatitis B virus, non - A, non-B virus, cytomegalovirus (Nakao et al, 1971) and Epstein - Barr virus.

Hepatitis B and non-A, non-B infection are the most predomenant (Marion and Tonkin, 1972, Galbraith et al, 1979).

The infection in the staff tends to differ from that occurring in patients. In the patients it is mild or asymptomatic, often anicteric illness with prolonged antigenaemia, so making them a continual source of infection. The staff tend to have a more acute illness, usually icteric and they rapidly clear the antigen from their blood as they recover. Mortality rates are higher among the staff (Sherlock, 1972).

Regular screening of H3 Ag by serological methods for both patients and staff help to prevent outbreaks, either by blocking the routes of infection or by identifying infected patients within the unit and isolating them as soon as possible (Polakoff, 1972).

### Aim of the Work

The aim of this work is to study the hepatitis problem in Ain Shams dialysis center during 1981-1982.

## Acute Viral Hepatitis

The term viral hepatitis refers to infections caused by at least 3 different viruses, hepatitis A (infectious hepatitis), hepatitis B (serum hepatitis) and the more recently identified type of hepatitis, non-A, non-B hepatitis which appears to be caused by more than one virus (Zuckerman, 1981).

In does not include hepatitis due to other viruses such as yellow fever virus, cytomegalovirus, glandular fever virus (Epstein-Barr) virus, adenovirus, herpes c simplex virus and Coxsackie virus (Stewart and Beswick, 1977).

Hepatitis A and hepatitis B viruses have been identified and characterized and the infection they cause can now be differentiated by sensitive laboratory tests for the specific viral antigens and antibodies.

Non-A, non-B is at present the commonest type of post-transfusion hepatitis in some areas of the world, it is also an important cause of speradic hepatitis in adults, although precise virological criteria and specific laboratory tests are not yet available (Zuckerman, 1981).

## REVIEW OF LITERATURE

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#### Viral Hepatitis A

Synonym (Infectious hepatitis, short incubation hepatitis).

Hepatitis A is an ancient disease, first described by Hippocrates. It was long known under such names "acute catarrhal Jaundice", "epidemic Jaundice", and "epidemic hepatitis". The fulminating form of the disease was called "acute yellow atrophy of the liver, (Murry, 1955).

Hepatitis A is endemic in all parts of the world, the exact incidence is unknown because of high proportion of asymptomatic and anicteric infections, but generally speaking, the prevalence in industrialized countries is decreasing and the infection is virtually universal. The highest incidence of hepatitis A is observed in school children, but in North America and in many countries in northern Europe most cases occur in adults. It appears to have a seasonal incidence, occuring during the autumn and winter manths (Zuckerman, 1981).

The occurance of both sporadic and epidemic hepatitis depends on the distribution of infected material and the susceptibility of the exposed population.

Thus poor sanitation, overcrowding and exposure of a non-immune population will contribute to an epidemic situation with a high frequency of infection in the young. Therefore, epidemics commonly occur in institutions that house small children - orphanges, schools and homes for mentally retarded. Adults without previous exposure to hepatitis A infection are particularly at risk when travelling in or moving to an area of high endemic infection with poor sanitation (Jeffries, 1979).

Transmission of the hepatitis A virus is mainly by faecal - oral route, usually by person to person contact. Outbreaks with a common source result most frequently from faecal contamination of drinking water and food. The source can often be traced to uncooked food or food that has been handled after cooking. The consumption of raw or inadequately cooked shellfish taken from sewage-contaminated water is also associated with a high risk of infection water borne epidemics can occur as the virus is relatively resistant to chlorine disinfection (Stewart and Beswick 1977).

Hepatitis A can be transmitted by blood or blood products and by parentral route with a more severe clinical course.

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Type A hepatitis maintains itself in human populations without either an extrahuman or human reservoir.

Intestinal carriers do not appear to be epidemiologically important, viraemic carriers have not been demonstrated (Mosley, 1975).

The incubation period is between 3-5 weeks with a mean of 28 days. Subclinical and anicteric infections are common, complete recovery is the rule. There is no evidence of peristence of infection nor of progression to chronic liver damage. (Jeffries, 1979, Suckerman, 1981)

Antibodies to hepatitis A virus appear in the serum within 1 month of onset of the clinical disease and persist for years. They may be responsible for persisting immunity to subsequent A virus infection and have no pathogenic role. No antigenic relationship could be found between hepatitis A and depatitis B antigens (Feinstone et al, 1973).

Only one serotype of hepatitis A virus has been identified in different geographical regions, but there is recent evidence that some differences in antigenic strains may exist (Zuckerman, 1981).

Viral particles 27 nm in diameter were identified in 1973 by the immune electron microscope in faeces obtained during the early acute phase of illness from adult volunteers infected wally or by the parentral route with hepatitis A virus. Viral particles are found during the incubation period, begining as early as nine days after exposure, also during the acute phase of illness, but the number of particles decreased rapidly after the onset of clinical jaundice.

Hepatitis A is an unenveloped virus containing a linear genome of single stranded RNA with a molecular weight of  $1.9 \times 10^6$ , and polypeptides with similar molecular weights to those of the four major polypeptides of the Enterovirus genus.

The virus is ether resistant, stable at pH 3.0 and relatively resistant to inactivation by heat. It is partially inactivated by heat at 60°C for one hour mostly inactivated at 60°C for ten hours and inactivated at 100°C for five minutes. Hepatitis A virus is inactivated by ultraviolet irradiation and by treatment with 1:4000 concentration of formaldehyde solution at 37°C for 72 hours. Also chlorine at a concentration of 1 mg/L for 30 minute can inactivate the virus.

Marmoset monkeys and chimpanzees appear to be the only susceptible animals. The propagation of hepatitis A virus in 1979 in primary monolayer and explant cell culture and in continuous cell strains of primate origin opens the way to the detection and assay of the virus in vitro and also to the preparation of hepatitis A vaccine, (Zuckerman, 1981).

Specific immune adherence and complement fixation tests, radioimmunoassay, enzyme linked immunoserbent assay for anti- hepatitis A IgM have been developed, using hepatitis A antigen (H.A.Ag) from the liver of infected marmosets.

Recently the solid - phase immuno-radiometric assay, can be used for the detection of hepatitis A.Ag and Ab.

Another method for the diagnosis of recent infection is by detection of hepatitis A virus in faeces if appropriate early samples are available (Hollinger et al 1975, Zuckerman, 1979, WHO, 1981).

In a dialysis unit, infectious hepatitis may present a hazard when precautions in handling contaminated objects such as bedpands or soiled sheets are ignored. Failure to wash the hands frequently or to wear gloves when indicated increases the danger of exposure. (London and Mckee, 1873).