

*A Study Of The Prevalance Of Chronic
Salmonellosis In Cases Of Chronic
Cholecystitis*

Thesis Submitted

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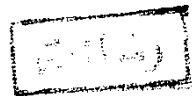
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INTRODUCTION

INTRODUCTION

Typhoid fever is a serious enteric infection in Egypt. (Farid et al 1975). Typhoid organisms can infect gall-bladder of man either through blood-borne or may even through the liver. (Christie 1980).

Chronic cholecystitis is a common disease in tropical areas. (Al-Hajjaimani et al 1986).

Chronic salmonellosis is a well known condition in tropical countries, either the patient is a chronic faecal carrier or a chronic urinary carrier (Farid et al 1979). In this work we try to detect the prevalence of chronic faecal carriers in cases of chronic cholecystitis. A twenty cases of cholecystectomized patients were chosen on the base of having a positive history of typhoid fever, at least since 12 months ago.

All the gall-bladders and also five stool specimens from five of the patients were subjected to repeated cultures and subcultures. The technique and results of the work will be discussed in the proceeding paper.

Aim Of The Work

To evaluate the prevalence of chronic salmonella carriers in cases of chronic cholecystitis.

TYPHOID FEVER

Introduction :-

Typhoid fever is world wide in distribution and is particularly prevalent throughout the tropics where it is one of the commonest causes of fever.

In Africa mainly, typhoid is present. Paratyphoid A occurs in Eastern Europe, U S A, Far East and India. Paratyphoid B occurs in North America. Paratyphoid C is wide spread in Guyana and is also found in Eastern Europe. (Manson et al 1982).

Hassan et al (1975) stated that typhoid fever is a highly significant endemic communicable disease in the Middle East and is one of the most serious enteric infections in Egypt.

The specific syndrome of typhoid fever is caused by the obligate human pathogen *Salmonella typhi*. Other enteric or paratyphoid fevers are similar to typhoid but are generally less severe. (Overturf et al 1981).

The salmonella are gram negative non sporing bacilli about 2-4 x 0.5 um, actively motile with numerous long peritrichous flagella.

They do not possess a capsule and most strains are fimbriated.

They grow well on ordinary media and form pale or colourless colonies on MacConkey's medium, since they do not ferment lactose. (Manson et al 1982).

The salmonella grow best at 37°C and can survive freezing, in still water and drying for several weeks. They are killed by a temperature of 60°C for 15 minutes and rapidly by boiling.

The typhoid bacilli has three antigens :-

O- Somatic or body antigen.

H- Flagellar antigen.

Vi- Coating antigen and responsible for virulence.

The bacteria may be in a smooth virulent phage or a rough non virulent variant, although this is not always the case. (Hackstep 1968).

Prevalance :-

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As the prevalence, the incidence rate in Egypt declined from 12.7 morbidity rate per 100,000 population in the year 1963 to 1964 to a rate of 60.9 per 100,000 population in 1965. (El-Akkad 1970).

Mortality rate of typhoid fever is about 1% in average. The incidence of typhoid fever is higher in towns and cities than in villages. One of the explanation is the fact that in towns people share others in the same source of food, milk and water supply, while in villages usually each house has its own food and milk supply so if any source is contaminated, infection will be limited to a very few number of cases.

Statistical records showed that while about 10,000 cases were discovered in Cairo, only two cases were discovered in Matruh in the year 1965. (Kent et al 1970, El-Akkad 1970).

Typhoid and paratyphoid occur all the year round but there runs a seasonal rise during May and it reaches its peak in August then it declines again. In years where summer months are prolonged a second wave of rise is expected in October as had happened in 1964 and 1966. (El-Akkad).

Source Of Infection :-

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The human population is the reservoir as well as the natural host for salmonella typhi. (Levine et al 1982). The source of infection is human excreta harbouring the organism but this is

probably true for the paratyphoids but other salmonella species for example salmonella typhi varium infect many animals which may act as a reservoirs. Every one becomes infected with enteric organisms and excrete them at the same time or other. Where no symptoms or clinical signs develop, the individuals are classified as symptomless excretors.

Most individuals who have clinical typhoid stop excreting the organisms within three months of the onset. Those who continue to excrete after three months are usually defined as carriers. (Macgregor 1953). In general 2-5% of all individuals who develop clinical or subclinical infection with S.typhi become chronic gall-bladder carriers and they lead to maintenance of the disease. (Levine et al 1982).

There are three types of carriers :-

- 1- Convalescent carrier, which passes bacilli in the excreta for up to six months after an attack of typhoid fever.
- 2- Chronic faecal carrier, which continues to pass the bacilli for more than one year in an intermittent manner. In the faecal carrier, the gall-bladder and the bile ducts being the seat of chronic inflammation, gall stones and chronic cholecystitis may occur.
- 3- Chronic urinary carriers especially with Schistosoma mansoni infection. (Hathout 1977, Hanson et al 1979).

Also if the patient continues to excrete the bacilli for more than a year of infection. He is called a temporary carrier. (Macgregor 1953).

Women are three times more likely than men to become carriers probably because of the compromises in the function of the gall-bladder secondary to the hormonal effects which occur during pregnancy. (Hanson 1985). Many examples of typhoid carrier state have been attributed

localisation of infection either in the gall-bladder or in the liver (18). Patients may be asymptomatic or present with a clinical picture similar to that of cholecystitis, suppurative cholangitis or liver abscess. Occasionally infection can persist in the gall-bladder for long periods. There is one report of infection persisting for 42 years. (Alberti et al 1979).

Spread Of Infection :-

The carrier is a danger to the community unless there is an efficient water carrying system of sewage disposal. Bacilli present on the hands of the carrier can be transferred by many vehicles, *S.typhi* spread mainly by water and *C.paratyphi A&B* by foodstuffs. All the enteric group can spread by ice cream. (Christie 1980, Manson et al 1982).

The organisms discharged in faeces and urine disseminated into water, milk or other food stuffs, infection follow ingestion of contaminated materials.

Typhoid and paratyphoid organisms will remain alive in water for days. If the water is clean they will not multiply but oxygenation and the presence of organic materials will assist their growth. Typhoid bacilli can persist for 2-3 weeks in sewage, also it have been recovered from sea water. (Goldman et al 1964, Macgrath 1984).

Outbreak of typhoid are often enter-borne although the organism is rarely present in small numbers except in gross infections. Shellfish consumption of sea water polluted may act as a source of infection because they concentrate water in the process of feeding. Many outbreaks results from milk and milk products consumption. Also flies may play a role by carrying the bacilli on their feet and proboscis. (Macgrath 1984).

It is stated that *S. typhi* may persist 38 days while *S. paratyphi B* for 21 days in sewage in spite of they are subjected to competition with other organisms but sewage is continually replenished with typhoid or paratyphoid organisms if there are carriers or cases in the area. If sewage from coastal towns is deposited crude & untreated into the sea, the beaches and coastal waters are subjected to pollution which may be heavy or light according to the current, tides & the site of the sewer outfall. In spite of all that there is no evidence that the percentage of infected bathers exceeds that of the inland inhabitants. Sea water is rapidly bactericidal to *S. typhi* but there may be to the amount of sewage pollution where our coastal waters can deal with. (Khalil 1970, Christie 1980).

Immunology:-

Humoral immunity is indicated by the presence of IgG and IgM antibodies which are not protective & cellular immunity by the granulomatous response in the typhoid nodule and the granulomatous pneumonia found in prologed typhoid.

An attack leads to some immunity which is not solid because another attack can occur but in a lesser extent than unimmunised persons. The earliest humoral response in acute typhoid fever is said to be a rise in the somatic O 'IgM' antibody titre. The flagellar H 'IgG' antibody usually develops more slowly but persists longer than the O antibody. At the end of the first week either antibody may be as high as 1/160. (Abdel Wahab 1970, Manson et al 1982).

Pathology:-

Acute salmonella gastroenteritis is most often caused by salmonella other than *salmonella typhosa*, it presents an acute gastrointestinal disorder 12-24 hours after ingestion of the organisms. The small bowel especially the ileum is most frequently affected but the stomach and colon may be affected. The small intestine is often dilated, the serosa is erythematous and fibrinous exudate may be

present, in severe cases the mucosa is erythematous, the lumen contains mucopurulent exudate, gas or bilious liquid chyme.

The pattern is that of acute catarrhal enteritis, acute mesenteric lymphadenitis and acute septic splenitis. This non specific pattern may show more histiocytes than might be expected, but this not sufficient to specifically diagnose salmonellosis. Diagnosis is certain only after immunofluorescent staining and or bacterial culture. (Smith 1970).

In enterica the course of the disease progresses through five stages :-

1- Period Of Incubation :-

The anatomic alteration consists of a mild inflammation of the lamina propria of the small intestine with mild mesenteric lymph node hyperplasia & lymphadenitis, these inflammatory infiltrates do contain neutrophils but other cells e.g. histiocytes, plasma cells & lymphocytes predominate.

2- Period Of Invasion :-

Mild endotoxin lesions of the liver & spleen hyperplasia of lymphoid tissue, histiocytic infiltration of the lymphoreticular system & catarrhal enteritis...

3- Fastigium :-

It is stage of necrosis though ulceration begins late in this stage. Zenker's degeneration of striated muscles may be severe and is almost often found in muscles which remain active while the patient lie in bed, " the intercostal diaphragm, rectus abdominis and the thighs. This degeneration may cause muscle rupture and haemorrhage which in the rectus abdominus muscle may simulate acute abdomen.

Microscopically the typhoid nodules are larger and may be centrally necrotic. It may be seen in the liver, bone marrow, spleen kidney, testis and parotid glands.

4- The Stage Of Lysis :-

It is marked sloughing of necrotic Peyer's patches, endotoxin effects and the early stages of tissue repair.

5- Convalescent Stage :-

The external appearance of the intestine return to normal, granulation tissue and reepithelisation occur, scarring and distorsion of the intestine rarely occur. (Spencer 1973, Smith 1970).

Infection is by ingestion, the organism pass from the small intestine via the lymphatics to the mesenteric glands and after multiplication they invade the thoracic duct. The liver, gall-bladder are infected and from the gall-bladder a further invasion of the intestine results. The gall-bladder and bile ducts which maintain the source of infection may be affected but the carrier state may be associated with chronic cholecystitis & gall stones , while chronic cholecystitis is not common in most tropical areas. (Manson et al 1982). The kidney shows cloudy swelling, the myocardium shows fatty degeneration also pyogenic meningitis may occur. Osteomyelitis of the long bones occur years after infection, the vertebrae may be affected with development of typhoid spine. Osteomyelitis complicating typhoid occurs more in children with sickle cell disease. (Edington et al 1976, McLeod John 1974). In paratyphoid fever the small intestine may be acutely inflamed throughout its length. In paratyphoid C septicaemia is common and deep metastatic abscesses are found. The organism may be recovered post-mortem from intestinal lesions, enlarged lymphatic glands, spleen, gall-bladder, heart, blood, and other tissues. (Huckstep 1962, Manson et al 1982, Christie 1989).

Clinical Features :-

Salmonella infections in man present a spectrum of clinical syndromes of

- 1- Enteric fevers "typhoid or paratyphoids" .
- 2- Acute gastroenteritis.
- 3- Bacteraemia.
- 4- Localized infection, which may occur at almost any site.

In addition asymptomatic intestinal carrier states are common. Occasionally a focus of infection persists in the gall-bladder or urinary tract to produce a chronic carrier state. (Thorn et al 1977).

The usual incubation period of all enteric infections is 14 days but may vary from 7-21 days. Also the severity of the disease varies much. (Manson et al 1982). There is some evidence that when the disease is water-borne the incubation varies from as little as 48 hours in food associated outbreaks to as long as 30 days in water-borne infections; this may be due to small number of organisms likely to be present in water-borne infections. (Christie 1980, Overturf et al 1981). Shorter periods of 4-5 days only are not uncommon especially when *S. paratyphi B.* is the infecting agent or vehicle a highly favourable one as milk or ice-cream. (Christie 1980).

Symptoms :-

The onset is normally insidious with malaise as a vague but typical presenting symptom. Chills are another indefinite but characteristic symptom. Rigors are not uncommon. The patient usually has nagging & persistent headache rather than severe but sometimes intense enough to suggest meningitis. Pain in the body, insomnia, malaise and anorexia may accompany it usually amount to discomfort rather than severe pain. (Manson et al 1982, Christie 1980). But the onset may be sudden with rigors and convulsions which resemble influenza, typhus and malaria. (Hathout et al 1979). Epistaxis is commoner in typhoid than paratyphoid. Abdominal discomfort is common but abdominal pain is rare, occasionally the patient has quite sharp abdominal