INFECTIVE HEPATIC JAUNDICE *

BY

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During the last few years sporadic cases of the infective hepatic jaundice type have been common in South Derbyshire, and several epidemics have been described in this or adjacent districts. Montford (1934) recorded forty-five cases in Castle Donington (population 2,674) in 1933, only two of which were in adults. Frazer (1935) recorded jaundice in twenty-five children all attending the same day school in Newhall. Richards (1933) described an epidemic among adults attending the Derbyshire Royal Infirmary for anti-syphilitic treatment, of which there were 120 cases. It is true that all except one had received, at some time, an arsenobenzol compound, but the epidemic was due to close contact in the waiting-room, and died out when this was remedied.

If we exclude spirochaetal jaundice and discuss simple infectious jaundice we have a subject suitable for a clinical society, because any one of us may have the opportunity of studying the natural history of the condition more fully and the chance to add something to medical knowledge. It was Pasteur who said, "In the scientific world chance only comes to the mind that is prepared."

Nomenclature

Our subject will include: (1) Catarrhal jaundice proper in epidemic and sporadic form; this is essentially a gastritis passing on to the duodenum, whence inflammation spreads up the bile duct, leading to obstructive jaundice. (2) Infective hepatic jaundice, also occurring in epidemic and sporadic form, which is an acute hepatitis, giving rise to toxic jaundice. From this second group arise some cases of acute or subacute necrosis of the liver, which is a better-defined name than acute yellow atrophy or the clinical label icterus gravis.

Much has been written under the title epidemic catarrhal jaundice since Cockayne (1912) distinguished the milder type of epidemic jaundice from that due to the spirochaete. In recent years, although the name catarrhal has been retained, the trend of opinion and much of the evidence has been toward a mild toxic jaundice as the explanation of the cases. For this disease infective hepatic jaundice is the name of choice. Hurst and Simpson (1934) have published convincing evidence that catarrhal jaundice and infective hepatic jaundice are two distinct diseases which have been confused. We may return to this under the appropriate headings below when discussing the features about which further clinical observation is needed. A paper recently published, entitled "Non-spirochaetal Infectious Jaundice," illustrates the difficulties of nomenclature. It is probable, however, that in the future we shall group most of the cases, both epidemic and sporadic, under the title of infective hepatic jaundice, with an occasional group attributable to the rarer condition catarrhal jaundice.

Some Features of the Epidemics

In the Wensleydale epidemic, reported by Pickles (1930), there were 250 cases of jaundice in a population of 5,700 people. The majority of those infected were elementary school children, and close contact in school was a definite source of infection. There were no fatal cases. A few children had pain suggesting some acute abdominal lesion. The incubation period was twenty-five to thirty-five days and the period of infectivity short.

Glover and Wilson (1931) observed an epidemic in a country town of 6,000 people in 1930. There were two or three hundred cases in the town. In one family jaundice began in four children on the same day. There were two large boarding schools in the town. In a girls' school with 250 boarders there was no jaundice, although one of the teachers was affected during the holidays. In a boys' school with 400 boarders there were ninety-five cases of jaundice. It was thought that infection was spread by the nasopharynx, and as evidence of close contact we have seven cases of jaundice out of nine boys in one dormitory and five out of nine in another.

Bashford (1934) recorded forty-eight cases of jaundice during the months of June and July, 1934, in workers in the General Post Office in London. In one building, where there were 1,900 workers, there occurred forty-three of these cases, but they were spread over four different rooms. This would suggest that the condition is highly infectious, but that the majority of adults may have acquired an immunity. None of them carried infection to their own homes, where they remained during the illness, and all recovered.

A study of the epidemics described will show that they tend to occur in rural districts and in institutions. It is most infectious to children, is the result of close contact, and the stage actually infectious is short. The incubation period is a long one, probably between three and five weeks.

Clinical Features of Infective Hepatic Jaundice

The onset is fairly sudden, with perhaps one attack of vomiting; there is an early rise of temperature, with jaundice usually appearing next day. Malaise is pronounced. The liver is enlarged, and the spleen appears to vary in size in different epidemics. There is some bile in the stools and the van den Bergh reaction is biphasic, being characteristic of a toxic condition. In a number of cases there is only jaundice, with the other symptoms reduced to a minimum. Exceptionally there may be severe abdominal pain. A few cases have a good deal of vomiting. In one or two epidemics the illness has begun with a sore throat. The jaundice lasts a variable time, usually one to three weeks. Convalescence is slow.

Prognosis in Infective Hepatic Jaundice

The great majority of the patients get perfectly well. Some liver efficiency tests carried out by American observers (Soffer and Paulson, 1934) suggest that frequently the organ does not recover fully. The observations, however, are few and the tests themselves not of proven value. The clinical evidence is all in favour of complete recovery in most cases, although a follow-up over several years by those who have observed epidemics will be the most valuable evidence. There is no doubt that convalescence may be very tedious. A colleague of mine in a neighbouring country town, where there has been an epidemic, tells me of an adult who felt miserably ill for three months after what appeared to be quite a mild attack.

Though death is an exceptional event it is necessary to treat these apparently simple cases of jaundice with respect, because there may be much damage to liver tissue without symptoms arising to give warning of the fact. This latency of symptoms in hepatitis is borne out by the experimental evidence of giving sublethal doses of chemical toxins to animals.

* From an address to a combined meeting of the Nottingham and Derby Medical Societies on April 21st, 1936.

As an example in clinical medicine I have observed the following case.

A boy aged 8 years was admitted to the Derbyshire Royal Infirmary. Fourteen days previously he became feverish, with loss of appetite, and slight jaundice developed. The day of admission, without any warning, he became very ill, was admitted delirious, with vomiting, muscular spasms, and incontinence of urine and faeces. The pupils were widely dilated; jaundice became intense, the liver was enlarged, but the spleen not palpable. The van den Bergh reaction gave a biphasic reading. He was treated with insulin and glucose, and after being very ill for two or three days recovered completely. Eighteen months afterwards he was reported as having remained in good health.

This would appear to be a clear case of necrosis of the liver as the result of infective hepatic jaundice, which stopped just short of a fatal issue. The sudden onset of the serious symptoms was dramatic.

Since putting these notes together a fatal case in a child has been admitted to the Derbyshire Royal Infirmary. The clinical history was similar. He died soon after admission. There was no evidence of duodenitis or inflammation of the bile ducts, but the liver cells were necrosed.

Relation to Necrosis of the Liver

These two cases may be accepted as necrosis of the liver cells arising in infective hepatic jaundice and giving rise to serious symptoms.

It is reasonable to conclude that acute hepatitis is present in all cases of infective hepatic jaundice. Gaskell (1933) had the opportunity of observing this post mortem in a child who died from secondary haemorrhage three days after tonsillectomy, and two days after jaundice with pyrexia had developed. Findlay and Dunlop (1932) recorded "a fatal case of acute necrosis of the liver associated with epidemic catarrhal jaundice." They refer to other fatal cases recorded, and summarize the evidence from Sweden of Wallgren and Bergstrand, who recorded that from 1914 to 1925 there were only two cases per year of acute necrosis of the liver in Stockholm. In 1926 and 1927 epidemic jaundice was common. In the latter year there were forty-two cases of acute necrosis of the liver. Most of these were adults, suggesting that the power of tissue regeneration was less than in childhood.

If infective hepatic jaundice is due to a virus, which sets up acute hepatitis; if it is highly infectious in children, but well resisted by them; if most adults have acquired immunity, but those who become infected have a liver less capable of regeneration than the child, the natural history of epidemic and sporadic cases may be explained.

Catarrhal Jaundice

Hurst and Simpson have discussed the features which distinguish true catarrhal jaundice from infective hepatic jaundice. Post-mortem evidence of a bile duct obstructed by catarrhal inflammation is hard to come by, but there are well-authenticated examples. The early symptoms of gastritis should be pronounced; and if this be the essential lesion, we should expect in an epidemic that a number of cases of gastritis without jaundice would be met with. There appears to be little evidence of this, but Hurst and Simpson record the observation of one doctor, who had under his care at the same time a brother and sister, the one with uncomplicated gastro-enteritis and the other with the same symptoms followed two days later by catarrhal jaundice.

I have observed two epidemics of simple jaundice in an institution. In the one case there was definite gastritis preceding the jaundice and the incubation period appeared to be seven or eight days. In the other epidemic, which was typical of infective hepatic jaundice, the incubation period was about twenty-five days.

On theoretical grounds the van den Bergh reaction should help in differentiation, by means of a direct positive result in catarrhal jaundice giving rise to obstructive jaundice, and by obtaining a biphasic reaction in toxic hepatitis. The readings, however, are not sufficiently convincing, and are probably of less value than the presence or absence of bile in the stools.

Summary

Simple epidemic jaundice is discussed with the conclusion that most cases labelled catarrhal jaundice are really due to an acute hepatitis and should be called infective hepatic jaundice. An isolated case may be regarded as a sporadic example of what is met with in epidemic form.

When opportunity arises to observe an epidemic it is suggested that further clinical observation is required with regard to the incubation period, which appears to be three or four weeks in the hepatitis cases and may be shorter in true catarrhal jaundice. In the latter condition in epidemic form we should like to obtain evidence of an infective gastritis which passed off without jaundice developing.

There is considerable evidence that infective hepatic jaundice may be a serious condition requiring careful treatment, with rest and glucose (with perhaps insulin) to protect the liver cells. In view of the doubtful value of our present liver efficiency tests there is need for a careful clinical study of the after-histories in those districts where epidemics have occurred.

There is recorded evidence of several children beginning the illness at the same time. Recently two medical officers have come under observation who both became ill within a day or two. This would suggest the possibility of a carrier, or more probably infection from an abortive unrecognized attack.

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M. A. Sisti (*Policlinico*, Sez. Prat., November 23, 1936, p. 2099), who records four illustrative cases in patients aged from 19 to 56, states that he obtained successful results by treating bronchial asthma by pneumoperitoneum when other methods had failed. The technique is the same as that used in the treatment of tuberculous enteritis. A puncture is made with a bevelled needle three fingerbreadths below and to the left of the umbilicus and oxygen is injected with an artificial pneumothorax apparatus. The quantity of gas introduced at a time varies from 250 to 350 c.cm.; refills should be given at first every two or four days, and later every eight or ten days. The duration of the treatment will depend on the clinical results. The method does not involve any risks. The patient has only to stay in bed in a supine position for two or three days with the pillows slightly raised to relieve the feeling of pain and dragging on the hypochondria and shoulders. He can then resume his ordinary occupation.