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CATAANHAL JAUNDICE

University of Nebraska
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CATARRHAL JAUNDICE

Catarrhal jaundice is an acute form of jaundice, the cause of which is not definitely known. It is characterized anatomically by jaundice of the skin and mucous membranes, and by enlargement of the liver; clinically by malaise, anorexia, and headache, which precede the presence of jaundice. The course is usually mild in character and lasts from three to six weeks.

Catarrhal jaundice is a common disease and is observed by the practitioner more frequently than any other liver disease. Of particular interest at the present time is the controversy as to pathology and new principles of treatment of this disease. Osler speaks of duodenal catarrh and a plug of inspissated mucous in the common bile duct where it enters the duodenum as being the pathology of catarrhal jaundice. The literature of the last fifteen years is in sharp conflict with the older views concerning the etiology, pathology, and underlying physiology of catarrhal jaundice. The study of this disease has been hampered in the past by absence of autopsy material, but a series of accidental deaths occurring in patients having catarrhal jaundice has stimulated investigation and literature concerning this disease.

I shall show that a new conception of the pathology of catarrhal jaundice has appeared since the writing of the older descriptions such as were given by Osler. In fact, the present tendency is to consider the name "catarrhal jaundice" a misnomer as recent evidence does not show a catarrh of the common biliary duct but instead, consistently finds intrahepatic pathology. Some form of "hepatosis" or "icterus simplex" are terms suggested as substitutes for the term "catarrhal jaundice."
New forms of therapy which substantially decrease the distress and the length of the disease bring this subject to worthy consideration at this time.

It is of importance to be able to make a differential diagnosis of catarrhal jaundice, for a diagnosis of this disease gives a much different prognosis and method of therapy than most other forms of jaundice.

The first literature on this subject was reported by Cleghorn in 1749, describing a number of cases in England. In the United States Faulkner wrote a description of a number of cases in 1812, and again in 1860.

This disease is found the world over, but principally in temperate climates. It is typically a disease of adolescents and young adults, although it affects individuals of every age. It is rare in infants but Reber has collected from literature and private practice more than a dozen cases of catarrhal jaundice in patients less than one year old. It is not uncommon in elderly patients and increases the difficulty of diagnosis in these patients, as there are so many cases of jaundice in older individuals.

Both sexes are affected equally, according to the large majority of authors, although some claim that men are more frequently affected. Catarrhal jaundice occurs both as isolated cases and as epidemics. Formerly the epidemic form was believed to be a mild form of "Weil's disease", which is caused by a spirochaete, but extensive clinical work by many men: Grover, Bard, and Wilson, Blumer, Morgan, Ross, and others, has definitely disproved this. Work done by these investigators included search for bacteria and spirochaetes in blood, faeces, and urine; blood serum agglutination tests for typhoid and paratyphoid organisms; blood Wasserman tests, and dark field examinations for spirochaetes. Animal inoculations were made to rule out possibilities of spirochaetes. All these tests have been consistently negative for any indication as to the etiology of catarrhal jaundice.
The clinical symptoms, course, et al., were the same for all these cases as for the isolated cases, and the syndrome has been found in epidemic form to be the same as for the isolated case, so that practically all students of the disease agree that they are the same disease.

A large percentage of cases have an associated upper respiratory infection, most commonly illustrated by a reddened pharynx. The logical deduction, then, is that droplet infection from the throat or nasal secretions is the causal agent. Whether this works directly or through a toxemia is an unsettled point.

The tracing of epidemiology with no upper respiratory infection, when there has been no known exposure, and in isolated cases where there is almost certainly no exposure to other cases, is a problem which has not been settled nor explained satisfactorily. Research has been extensively done to rule out foods, rats, and water supply as etiological agents, and no connection of these agents to the disease has ever been found.

Davis\(^8\), Bronson\(^9\), Guthrie\(^10\), Calvert\(^11\), and others have had a considerable number of cases following or associated with influenza, Davis reporting eighteen cases during an epidemic of influenza, Bronson twelve cases, and Calvert five cases in one family, but the large majority of cases with no such associated history indicates that this is not a frequent etiological factor. The presence of a toxic condition in association with the jaundice may be of some significance, as will be discussed later.

Blumer\(^5\) classifies cases as (1) isolated, (2) family outbreaks, (3) institutional outbreaks, (4) city outbreaks, and (5) country outbreaks. These are self-explanatory, the most common being the isolated and family outbreaks. Outbreaks have occurred in hospitals and accurate records kept which establishes strong proof of the contagious nature of certain types of catarrhal jaundice.
Incubation period in these cases appears to be quite long, investigation by several authors placing it from eighteen to thirty-eight days, several agreeing at about twenty-six days.

The ordinary case does not seem to be contagious enough to worry about the spread of the disease unless it is epidemic at the time.

As stated before, the older theory as to causation of this disease is a spread of catarrh from the duodenum into the common bile duct. Osler's conception of etiology and pathology was a congestion, swelling, edema, and increased production of mucus at the lower end of the common bile duct and the duodenal mucosa adjacent to the diverticulum of Vater. In some cases, he believed this swelling itself sufficient to obstruct the flow of bile, but in other cases a plug of inspissated mucus more effectually blocks the common bile duct. He admits that he had only seen one such case found at autopsy and explains the fact that this had not been found at more post mortem examinations to a subsiding of swelling and congestion after death. Quoting from Osler:

"In other cases there is an infection of the finer ducts within the liver which may be due to an infection from the intestine. There is evidence of disturbance of the liver function, and some degree of hepatitis is probably always present. Catarrhal jaundice is occasionally seen in infectious fevers."

It is interesting to note here a fact that many authors: Carr, Hopkins, and others, have noticed, that catarrhal jaundice is rarest in the summer months when digestive disturbances are the most frequent. There has been no explanation of this fact by adherents to the theory that catarrh of the bile ducts occurs following digestive disturbances.
Eppinger\textsuperscript{16}, in 1908, autopsied a case of catarrhal jaundice with accidental death on the eighth day of jaundice, which showed hyperpasia of the lymphoid tissue of the mucosa of the part of the common biliary duct which runs in the duodenal wall; this had occluded the duct and led to the dilatation of the duct and retention of bile. He concluded that a duodenitis would lead to the swelling of this lymphoid tissue and thus result in catarrhal jaundice.

Eppinger continued to study this disease and during the world war had opportunity to autopsy several cases of soldiers with mild catarrhal jaundice killed in the war. In none of these cases was a mucous plug or any evidence of any inflammation of the common bile duct or of duodenitis present. The liver in each case was enlarged; otherwise grossly normal. Microscopically, the cells were distorted and poorly stained, and the nuclei were deformed and poorly stained. The lymph vessels were enlarged and in close proximity to the blood vessels. There was no round cell ed infiltration or other evidence of any active inflammatory process. Bauer took a section from the liver of a case which had been incorrectly diagnosed and a laparotomy done and found the same microscopic picture present.

Lindstedt\textsuperscript{18} of Sweden reports a typical case with accidental death, with parenchymatous degeneration of the liver and no other finding at autopsy. S. Corinaldesi\textsuperscript{19} reports two cases with exactly similar history and findings.

Eppinger considers this pathology as acute atrophy of the liver in miniature. There are several cases reported of deaths of acute yellow atrophy of the liver following what appeared to be typical catarrhal jaundice. This will be discussed later in this paper.

There is no recognizable pathology in other organs of the body in this disease except occasional splenomegaly and the deposit of pigment in the skin and mucous membranes causing jaundice.
In almost every case there is evidence to show actual liver damage and disfunction. This will be more fully discussed. This is another point in favor of pathology in the liver itself, rather than in the bile duct.

It is entirely possible that all cases of catarrhal jaundice are not alike, that is, that some of the cases are due to cholangitis and duodenitis but these are probably the exception rather than the rule.

The symptomatology of catarrhal jaundice is well established but varies in the individual case to some extent.

In the typical case there is a three to six day period of anorexia, malaise, lassitude, and headache, with possibly occasional nausea and vomiting. Accompanying this there is usually bad taste in the mouth, coated tongue, and foul breath. There is epigastric distress especially after eating, which may be felt as a sensation of soreness over the liver area. Constipation is the rule, but the bowel movements may be normal or there may be a diarrhea. The malaise and lassitude often amount to an actual mental depression. Vertigo often accompanies the headache, and the lassitude may be an actual sleepiness. Quite frequently there is an upper respiratory infection and cough to which the symptoms are attributed. The temperature is normal or slightly increased, 100° representing a rather high temperature for catarrhal jaundice.

The patient usually presents himself to the physician in the stage of beginning jaundice. The yellow tint may be overlooked in artificial light, is present in the sclera of the eyes before the skin is jaundiced, and is most marked in the sclera even after the jaundice has developed. The jaundice is of slow and insidious onset. The skin and mucous membranes become diffusely yellow without intense areas of localization. Although the jaundice usually appears three to six days after symptoms are present, occasionally jaundice is the first
symptom and there are no subjective symptoms until after the jaundice is present.

With the appearance of the jaundice, or even before, the stools become pale and are soon clay colored, and the urine becomes darker. The urine becomes very dark as the jaundice increases, scanty, with a heavy sediment. Yellow foam appears on shaking the urine. The stools become lighter until they are even lime colored.

It takes four or five days after the jaundice first begins for the condition described above to reach its height and it then continues for a few days, followed by a gradual clearing up of all symptoms over a period of about two or three weeks. The jaundice is the last thing to clear up and may remain some time after all other symptoms have subsided.

During the disease the pulse rate is usually slow, around sixty per minute. This bradycardia is usually attributed to the action of bile retention on the circulatory system. The blood pressure is usually normal or slightly subnormal.

The pruritis associated with jaundice is often quite marked and sometimes precedes the noticeable change in color of the skin. Cases of severe eczematous lesions resulting from uncontrollable scratching sometimes result unless careful treatment is given.

In many cases the liver is palpably enlarged and tender, although it may be entirely normal. Rarely the spleen is enlarged so that it may be palpated.

The patient loses weight and strength, is usually irritable toward the latter part of the disease.

The temperature is usually below normal, or normal, in the latter part of the course of the disease. There may be an initial slight leukocytosis but a leukopenia is the rule in this disease throughout its course.
Typical forms have been reported in which the condition described lasts as long as three months. With proper and early treatment it is sometimes cleared up in a few days, as far as subjective symptoms are concerned.

Even before the jaundice occurs, excessive amounts of bile pigments are present in the blood serum, and at the first clinical indication of jaundice the icterus index is increased and the Van den Bergh test is strong and immediate direct and strong and high indirect. The icterus index is usually fifty to one hundred times the normal at the height of the jaundice, and the indirect Van den Bergh goes as high as one hundred milligrams per liter.

The red cell count and hemoglobin determinations are not affected by this disease. Jones and Minot\textsuperscript{20} and Thewlis and Middleton\textsuperscript{21} made intensive study of the white cell count and found that early in the disease there may be a slight leukocytosis, not over ten thousand cells, but the count quickly drops until there is a leukopenia. A white cell count somewhere about four thousand, five hundred cells is to be expected a few days after the jaundice has appeared.

The color of the urine is dark, as has been stated, and the color deepens as the jaundice increases. Yellow foam appears on shaking, indicating the presence of bilirubin. Hay's test for bile salts in the urine is positive in an early stage. This consists of placing a pinch of flowers of sulphur on the urine, the sulphur sinks when bile salts are present, as in this case, and floats if bile salts are absent. Urobilinuria is a constant symptom in catarrhal icterus, Simici and Popesco\textsuperscript{22}. This is explained by the fact that since the obstruction to bile flow is not complete, some bile finds its way into the intestines. The urobilinogen formed in the gut is carried back to the liver but the diseased parenchymal cells of the liver are unable to utilize it. The liver therefore shunts the urobilin into the general circulation and it is then eliminated in the urine in large amounts.
As the jaundice increases, the stools become lighter in color, until at the climax of the disease they are clay colored. This might infer that a complete lack of flow of bile into the intestine might be assumed, but this is not the case, as proved by duodenal drainage. The bile obtained, however, is very poor in biliary pigments and bilic acid. This is a very strong argument against the "duct catarrh" or cholongitis theory of etiology of this disease, as bile that gets through such a condition would be normal, and this points to liver cell pathology. The whitish color of the stools is partly due to the presence of fats, which, in turn, is due to the inability of the intestine to digest fats in the absence of bile.

The above described laboratory data is true for the climax of the disease and lasts for a few days, and is followed by a gradual return to normal limits and normal finding of all laboratory procedure. The variation in degree and length of time abnormal laboratory findings are present of course varies with each case. The blood, faeces, and urine return to normal for several days before the jaundice clears up.

A galactose test for liver function which has been particularly worked on by Bauer and Lepezne\(^2\), has been found to be positive in this disease persistently as well as in other diseases with liver damage. It is constantly negative in true obstructive jaundice. This test has been found to be positive in patients for more than a month in practically all patients, and sometimes much longer.

The diagnosis of catarrhal jaundice is not always easy but is suggested by the history and the symptoms preceding the appearance of the jaundice. In epidemics the diagnosis is suggested at once. The occurrence in young individuals is suggestive of this disease rather than other liver pathology. Since luetic jaundice
closely resembles catarrhal jaundice, a Wasserman test should be made if there is any suggestion or possibility of lues as an etiological factor. Toxic jaundice due to arsenical treatment of lues should be ruled out by history, as should any possibility of mushrooms, phosphorus, arsenic, and chloroform as etiological agents.

In elderly patients obstruction of the common bile duct by stone or carcinoma should be seriously considered. A history of gall stone colic would indicate a stone and the galactose test is negative in obstructive jaundice. In case of carcinoma duodenal drainage is always negative for bile, while some bile can always be obtained in catarrhal jaundice.

Weil's disease is an infectious jaundice caused by a leptospira and is characterized by high fever and marked albuminuria, neither of which is present in catarrhal jaundice. Jaundice due to septic conditions or to abscess of the liver would not be confused with this disease.

If the jaundice persists, a gall bladder drainage (Lyon-Melchior) should always be done and a Grauer-Coyle gall bladder visualization is indicated to rule out other pathology. The material aspirated by the gall bladder drainage should be analyzed as to bile content and pus cells. Presence of pus cells may indicate a cholangitis present at the ampulla Vater. The immediately positive direct Van den Bergh reaction rules out hemolytic jaundice.

The prognosis of catarrhal jaundice is so uniformly good that many practitioners do not know that there may be complications. Although complications are rare, it must be remembered that many cases are protracted and the patient is often below par for weeks or even months after the jaundice has disappeared. Kalk and Heinz, Taylor and Rood, and others. This can be shown by bile index tests, galactose test, and bilirubin tolerance test. Also the clinical course of the patient may be that of a run-down, weak, and possibly nervous and irritable individual.
The most common complication reported is acute atrophy of the liver. Morgan and Brown, Blumer5, Jones17, Findlay, Dunlap, and Brown25, have each had at least one case ending fatally following typical cases of catarrhal jaundice and have mentioned at least ten other cases in foreign literature. If the liver is enlarged and palpable, and suddenly decreases in size, this should be regarded as an ominous sign, as it may be due to acute liver necrosis. In eleven years preceding 1927 in Sweden there were twenty-seven deaths reported from acute necrosis of the liver but in 1927 there was a great epidemic of catarrhal jaundice and there were forty-two deaths from acute liver necrosis in Stockholm alone that year. This is pointed to as an association of catarrhal jaundice and acute liver necrosis.

Abortion in pregnant women has been reported following acute catarrhal jaundice. Relapse and exacerbation of jaundice and clinical symptoms has been reported as an occasional occurrence. This is quite rare and usually lasts only a few days.

The acute necrosis of the liver, abortion, and relapse are so rare as to be almost negligible, but the protracted case which is jaundiced for several weeks longer than usual, or even if jaundice disappears and the patient remains below par should be emphasized. These cases are very common and should be borne in mind when treating a case, and all efforts should be made to assist the liver to recuperate from damage done to it. Sometimes the weight loss is severe in these protracted cases, requiring several months' convalescence.

Since, in many cases, catarrhal jaundice represents serious damage to the liver, it should be given serious treatment. The patient should be in bed, quiet, comfortable, and protected from changes in temperature. The protein intake should be reduced to no more than the minimal need, and in severe cases should be entirely omitted for a few days.
Anything that can possibly injure liver parenchyma must be avoided, especially alcohol, salvarsan, etc. No general anesthesia should be given unless absolutely necessary. Any gastritis, colitis, or other intestinal disease should be cleared up to avoid liver irritation by absorption.

Any measure tending to prevent further resorption of poisons or infectious material from the gastro-intestinal tract may be regarded as therapy against a possible causal agent. Evacuation of the bowels is the chief concern, even if diarrhea is present. Calomel, magnesium sulphate, and sodium phosphate all seem to be cathartics and at the same time stimulate bile flow in this disease.

Occasionally one of the greatest difficulties of the treatment of this disease is to persuade the patients that they must stay in bed for a time, and to be restful and away from work for an additional period. This should be insisted upon even if the course is a light one. Heat should be applied to the liver region either as hot compresses, hot water bottle, or electric pads.

Proper diet is one of the most important items in treatment of catarrhal jaundice. The proteins which charge the liver most severely: meat and fish, eggs and milk, must at first be eliminated entirely and later be taken very sparingly. The intake of fat should be restricted, to rest the liver and prevent foul smelling stools. The diet should consist chiefly of carbohydrates. Thickened soups, porridge, cream of wheat and other cereals, rice or starch puddings, noodles, macaroni, oat flakes, mashed potatoes, white bread, cookies, fruit juices, stewed fruits, apple sauce, etc., should make up the diet. Alcohol, spices, pepper, and excess salt are to be strictly forbidden.

The foregoing are time-tried remedies, used by all, but two new advances in treatment have been introduced which thus far have been found to reduce materially
the morbidity of this disease. These are the use of the Lyon-Meltzer duodenal tube and grape sugar and insulin therapy.

The technique of the Lyon-Meltzer drainage is to pass a duodenal tube (Reynuss tube is usually used) on a fasting morning stomach, say at seven A.M. The residuum of the stomach is withdrawn and the stomach washed clear with warm water. The patient is then given a glass of water to drink, turned on the right side, and by slow swallowing the duodenum should be reached in fifteen to forty-five minutes. This can be told by the different appearance and the alkaline reaction of the aspirated fluid. Then fifty cc. of warm twenty-five per cent magnesium sulphate is introduced, the tube clamped for fifteen minutes, and then allowed to siphon until one P.M. A. H. Hopkins found that he got an increased flow of bile by the addition of one and one-half grains of calomel by mouth after siphoning the contents following the magnesium sulphate injection.

Large quantities of bile and quick amelioration of symptoms and disappearance of jaundice is the rule following the above therapy, according to Hopkins, Lyon, Howard, Lepegne, and others. These men say that this not only stimulates the flow of bile, at the same time acts directly on the secreting function of the liver cells. Lyons treated sixteen cases in succession, eight with duodenal drainage and eight without, and in the group given duodenal drainage the jaundice averaged seventeen days and in the other group thirty-five days, until absence of jaundice. Hopkins found gastrointestinal symptoms to leave within forty-eight hours by use of duodenal drainage as against several days for those untreated by this method. He cut the average length of jaundice from thirteen days to six and one-half days in a large series of cases by this method.

It is claimed that the duodenal tube is distressing to the patient but all were grateful for the amelioration of symptoms by using this method. Painting
the pharynx with cocaine solution is advisable for some patients before the tube is passed.

An additional form of therapy used extensively in Germany is to feed grape sugar to the patient, an effective method being to use fifty to one hundred cc. of a thirty per cent solution of grape sugar in the duodenum just before the tube is removed. Also, small amounts by mouth at other times. This, with the use of five units of insulin three times a day is supposed to build up the glycogen reserve in the liver and thus fortify the liver against damage. This was first brought out by Richter and Umber and is quite new, but is widely acclaimed by foreign investigators. Thirty to forty grams of grape sugar per mouth is the usual amount given per twenty-four hours.

Treatment of course should be based upon the condition of the patient, and as the jaundice recedes, the stools darken, and the urine lightens, the diet may be eased up somewhat, but a period of at least a month should elapse before the patient should get back to a general diet. The bowels should be watched and kept loose during this month.

A somewhat distressing symptom associated with catarrhal jaundice is a pruritis. Not always present, it is sometimes the chief complaint of the patient and attempts at relief may be given with calamine lotion with phenol, powdering, washing with lemon juice, water and vinegar, or a one per cent mentholated alcohol. Bran baths may be tried or any favorite antipruritic of the physician treating the disease.

In conclusion, I wish to emphasize the newer concept of pathology and treatment of catarrhal jaundice. The generally accepted concept of the pathology is that there is a parenchymatous involvement of liver cells rather than the
older theory of the common bile duct being closed by congestion and edema and mucous. The possibility of a cholangitis accounting for a few cases of catarrhal jaundice should be considered. The newer form of therapy by use of the Lyon-Meltzer drainage, by use of duodenal tube and magnesium sulphate solution, seems to so materially relieve symptoms and decrease the duration of jaundice. The possibility that the use of grape sugar and insulin may still further reduce morbidity of catarrhal jaundice is well worth more experimental work.
Case Histories:

Case I:

Patient 27 years old, male, medical student. About January 11, 1933, patient began to have epigastric distress and felt badly generally, and did not care for food. This lasted a little more than a week when symptoms cleared up for four or five days. During this period his bowel action and stools were normal. Then about the twenty-fifth of January the distress returned to his epigastrum in an aggravated form and he felt nauseated but did not vomit. He felt an actual distaste for food and did not notice his stools but noticed his urine was very dark about the first day of February, and on February second he noticed his skin was turning yellow. He went to a physician who had him admitted to the University Hospital on February third. The physical examination showed only the jaundice and tenderness under the right costal margin as positive findings. Laboratory work done on February third showed urine normal except for a very dark color, red blood count and hemoglobin normal, and 5,900 white blood cells. The icteric index was 94, Van den Bergh strong and immediate direct and indirect strong and 91.4 mg. per liter. He was placed in bed on a fat free diet, forced fruit juices, and given sodium phosphate two ounces twice a day. On February eighth he was started on Caroid and Bile salts, tablets, one three times a day. On February sixth an attempt was made to do a duodenal drainage but the patient could not swallow the Reyfuss tube, so he was given two ounces of thirty per cent magnesium sulphate solution by mouth. Temperature was
always normal or subnormal, and pulse 60 to 80 per minute. He did not show much improvement until February seventh when symptoms began to be relieved and the jaundice was definitely lessened on February eighth. He was discharged from the hospital on February thirteenth in a much improved condition, with only faint jaundice, and went on to an uneventful recovery.

Case II:

From A. H. Hopkins:

Patient, a university student, male, twenty-two years old. Nine days before admission to the hospital he developed, after exposure to chilling, what he calls an attack of the "grippe", this being characterized by slight fever and pains in the back and legs. Three days later his throat became sore, and a day or so after that his friends called attention to a yellow discoloration of his skin. His bowels were constipated and after taking blue mass, he vomited. The jaundice had gradually grown more intense, so that at the end of two weeks it was very marked. He had some generalized abdominal discomfort, especially during the second week. He is usually constipated. Past history is negative. Physical examination on admission, i.e.: nine days after the onset of the illness, shows the skin and sclera deeply jaundiced, the liver extends to 3 cm. below the costal border in the nipple line, and is distinctly tender to palpation. Temperature 99°F., and pulse 60. The patient was put to bed and given a liquid diet and calomel .065 grains. followed by magnesium sulphate the next morning. Duodenal drainage was instituted over a period of two hours
with one and one-half grains of calomel given by mouth along side the tube and fifty cc. of 25% solution of magnesium sulphate administered over a period of nine days, with return of 50 to 250 cc. of fluid, consisting chiefly of yellow bile with lessening jaundice and symptoms, until on the ninth day of treatment he was both jaundice free and symptom free.

Case III:

From M. Reber:

Patient four months old, normal birth, weight and development.

No icterus neonatorum. On September 30, 1930, parents called a physician because the child had been jaundiced for several days, had clay colored stools, and had suffered from slight vomiting. Physical examination showed icterus, palpable liver, and palpable spleen as only positive physical signs. Urine positive for bile but otherwise negative. Patient fed on skim-milk. Temperature normal to 99.5 rectally first few days and thereafter normal. Wasserman and Von Pirquet reactions negative. Not much change until October 15th, gradual lessening of jaundice from the fifteenth to the nineteenth and jaundice absent by October 23, 1930. Followed for some time, the child was perfectly normal in every way, without return of the jaundice.


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