Original Articles.

OBLITERATIVE PERICARDITIS A CAUSE OF HEPATIC ENLARGEMENT AND ASCITES, WITH REPORT OF A CASE.¹

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E. H., a boy of eighteen, entered the Massachusetts General Hospital, February 2, 1893, in the service of Dr. F. C. Shattuck, by whose kindness I am permitted to report the case. There was nothing significant in the boy's family history. He had measles at seven, and says he has never been as well since. Six years ago a physician told him that his liver was enlarged and four years ago he noticed swelling of his belly.

It reached its present size (see illustrations) in a few weeks; there has been but little change in it since. Occasionally his feet have been swelled for short periods.

All these symptoms have troubled him but little. The reason for his coming to the hospital was cough, which had troubled him on and off for three years, but worse of late. Cough was usually dry. Appetite, digestion, bowels and sleep were all right, and until very lately he had worked in his father's store.

Physical examination showed a boy very backward in his development, which corresponded to that of most boys of twelve. He was considerably emaciated. The great prominence of the belly (see illustrations) was due partly to enlargement of the liver, which extended a hand's breadth below the navel and reached to the fourth rib above; partly to a large accumulation of fluid which made the belly flat on percussion except over a small area situated about the navel when he lay on his back, and shifting to whichever side was uppermost when he turned over. The surface of the liver

was apparently smooth, its edge easily felt. No glands, nodules or areas of increased resistance were palpable elsewhere. There was no tenderness anywhere. No enlargement of the spleen was demonstrated.

There was evidence of considerable fluid in the left chest — dulness, and absent breathing and voice-sounds up to the third rib in front and midscapula behind. This made it impossible accurately to determine the size of the heart, especially as the apex beat could be neither seen nor felt. The apex sounds were loudest in the fourth interspace, one-half inch inside the nipple — the heart not being pushed to the right by the pleuritic effusion, so far as could be determined. The sounds were feeble but clear, the pulmonic second sound somewhat accented.

The left lung was apparently compressed by the fluid in the pleura; for there was bronchial breathing and increased voice-sounds over that part of the chest not occupied by fluid. Whether there was any fluid in the right chest was hard to determine, on account of the (apparent) enlargement of the liver upwards. Over the upper half of this lung the breathing was puerile, with many medium, moist râles — signs interpreted as compensatory emphysema and edema.

There was no edema of the legs.

The urine showed the characteristics of renal congestion.

The boy could not breathe except in a sitting position; and in this position he sat, uncomplaining, either in a chair or in bed until his death on March 29th, eight weeks from the time of entering the hospital. He had some diarrhea — three to five loose movements daily — during the whole of this period despite various efforts to check the increased peristalsis. Cough was at times very troublesome and never absent. He often spat up four or five ounces of nearly pure serous fluid during the night.

The abdomen was tapped three times and the left

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chest once. About four quarts were obtained each time from the belly and two from the chest. The fluid was about the same at all the tapplings — approximately 1.008 in gravity, with a relatively small percentage of albumin, evidently a transudation.

An interesting feature of the case was the occurrence of transient swellings in the parotid region lasting three to eight days. Such swellings have been noticed in connection with a variety of abdominal diseases.

Towards the end of life his breathing grew quicker and shallower, and the evidences of pulmonary edema increased. Pain was absent throughout. The treatment was purely palliative and expectant. Death occurred apparently from pulmonary edema and exhaustion.

The *diagnosis* at the time of death was entirely uncertain. The absence of any history of alcohol and of any history or lesions of syphilis, congenital or acquired, made cirrhosis of the liver unlikely. There did not appear to be sufficient evidence of cardiac or renal disease to explain the signs. Tubercular peritonitis seemed unlikely in view of the nature of the fluid obtained by tapping and the absence of nodules or masses in the abdomen after tapping. The sputa was repeatedly examined for tubercle bacilli with negative results. The possibility of a new growth in or near the liver and pressing on the portal radicles or otherwise obstructing the portal circulation could not be excluded, and was considered on the whole the most likely diagnosis.

Autopsy, by Dr. W. F. Whitney, showed the following:

Heart of normal size; muscular substance presents nothing abnormal.

Pericardium entirely obliterated by firm fibrous adhesions; in the upper part a small, cheesy nodule.

Left lung retracted against chest wall into a mass the size of the fist and covered with dense fibrous adhesions. On section airless, dark red, looking like a spleen.

Right lung also somewhat compressed. Considerable fluid in both pleural cavities.

Liver greatly enlarged; nutmeg section surface

Kidneys dark red in color, with evidence of abundant blood.

Diaphragmatic muscle fibres in a state of marked fatty degeneration.

Diagnosis: Chronic obliterative pericarditis, probably of tubercular origin; chronic pleurisy, with double hydrothorax, and splenification of the left lung; chronic passive congestion of liver and kidneys.

Pick² reports three similar cases.

CASE I. Waiter, forty-seven. June, 1889, pericarditis. March, 1890, left pleural effusion; enlarged liver, ascites. Abdomen tapped six times in 1890 to 1892. Heart negative except for faintness of the sounds. *Diagnosis*, cirrhosis of the liver. Death, November, 1892, three and a half years after his pericarditis. *Autopsy*: Obliterative pericarditis; left lung compressed against chest wall and splenified; pleural cavity obliterated (exactly as in Dr. Shattuck's case); nutmeg liver, showing microscopically signs of chronic passive congestion with secondary increase of connective tissue; no evidence of primary cirrhosis.

CASE II. Man of twenty-six. Swelling of abdomen for eight years, supposed to be obesity. In 1888 began treatment for this at the Marienbäder, but the size of the belly increased and cyanosis and dyspnea supervened, so that the treatment was hastily abandoned. Veins of *upper and lower* extremities dilated; liver enlarged; ascites. Tapped repeatedly. Heart not enlarged; no murmur. In

the next three years increasing dyspnea, ascites, edema and hepatic enlargement. The diagnosis was at first cirrhosis of the liver, later tubercular peritonitis. A few months before death, which occurred in December, 1892 (twelve years from the first enlargement of the abdomen), a paradoxical pulse and an apex-presystolic murmur were noted; yet at the time of death the diagnosis rested: "cirrhosis of the liver." *Autopsy* showed obliterative pericarditis, with extensive calcification of the exudate; liver of chronic stasis; double hydrothorax.

Both these patients were moderate drinkers — not excessive — so that the etiological suggestion was equivocal. In both something closely resembling a primary cirrhosis was finally established by the prolonged stasis and irritation in the liver due to the insufficient cardiac power.

In the third case, Pick was enabled by his experience in the other two, to make the diagnosis during life.

CASE III. A man of twenty four had complained for two years of dyspnea, palpitation and swelling of the abdomen. Seen in February, 1894, with considerable enlargement of the liver, ascites, edema of the scrotum and feet. Heart-sounds clear; apex beat not to be seen or felt. *Diagnosis* at first, hepatic cirrhosis. Repeated tapplings necessary. In 1895 occasional friction sounds over the heart suggested the diagnosis of pericarditis. Death, September, 1895. *Autopsy*: Total obliteration of the pericardial sac, with calcification, so that the heart was almost cased in stone; double hydrothorax; secondary connective tissue overgrowth in liver.

Pick also mentions that he has seen two other cases not yet fully reported.

Most text-books and works on the heart make no mention of such a connection between chronic pericarditis and secondary interstitial hepatitis with enlargement and ascites, but a certain number of isolated cases are recorded.

Weiss³ mentions the case of a girl of fourteen with enlarged liver and spleen, slight jaundice, and marked ascites, but without edema of the legs, in whom physical examination showed nothing wrong with the heart, and in whom the diagnosis of cirrhosis of the liver was made. *Autopsy* showed a chronic obliterative pericarditis and the remains of old pleurisy and peritonitis. The liver was enlarged and firm, but not cirrhotic to the naked eye. Chronic renal congestion was present.

Wagner⁴ records a case of marked ascites in a man of thirty-five — not a hard drinker and without venereal history. The liver was enlarged. Heart-sounds feeble but clear. Tapping required four times in six months. The diagnosis made was: "Cirrhosis (?)" (*vielleicht Lebercirrhose*). *Autopsy*: Chronic tubercular pericarditis (fibrinopurulent), miliary tuberculosis. No evidence of tubercular peritonitis. Both pleural cavities obliterated. Pigmented scars and small cheesy patches in the lungs. Liver reaching a hand's breadth below the ribs; "nutmeg" section-surface, with a few fresh miliary tubercles. No cirrhosis, macroscopic or microscopic. Here the obstructed pulmonary circulation doubtless contributed largely to the weakness of the heart.

Liebemeister describes a case of chronic pericarditis with dilatation of both ventricles, in which edema of the extremities accompanied the enlargement of the liver and the ascites, but in which the edema of the extremities disappeared under the use of digitalis, while the ascites persisted.

Weinberg⁵ narrates two cases of tubercular pericarditis with obliteration, and large ascites, one tapped fifteen times. In both the liver was greatly enlarged. In one an explor-

³ Wien. med. Jahrb., 1876, p. 1.

⁴ Deutsch. Archiv. f. klin. Med., vol. xxxiv, p. 536, 1884.

⁵ Munch. med. Woch., 1884, Nos. 46, 47.

² Zolt. f. klin. Med., vol. xxix, 1890.

atory laparotomy was done, and the surface of the liver is noted to be "not quite smooth."

Most⁶ has reported two and Rumpf⁷ one similar case; and Unverricht,⁸ in a review of Most's article, speaks of the association of obliterative pericarditis and ascites as not uncommon.

Neusser⁹ mentions a case of obliterative pericarditis with ascites and edema, the ascites preceding the edema by some time and leading to a diagnosis of hepatic cirrhosis, which autopsy did not confirm.

V. Schrötter¹⁰ mentions a similar case in a young girl.

Rosenbach¹¹ mentions the case of a boy of ten with a puzzling enlargement of the liver and ascites, without any edema elsewhere, in whom at autopsy the only lesions were an obliterative pericarditis, with a nutmeg liver and perihepatitis.

Curschmann¹² refers to a woman of fifty, in whom ascites of unknown origin developed and persisted between tapings for six and one-half years. Autopsy showed obliterative pericarditis and obliterative right pleurisy with perihepatitis.

In the eighteen cases here collected the absolute latency of the pericarditis is important, but not on the whole surprising. I have watched a number of cases with Dr. F. C. Shattuck in which, during the course of a pneumonia, he was on the lookout especially for the advent of a pericarditis and examined carefully for it each day in vain; yet at the autopsy the pericarditis was found to have crept in.

The weakening of the apex impulse was the only sign present in the case of adhesive pericarditis here presented; and such weakening is in no way distinctive, but occurs in various other conditions.

The diagnosis can hardly be made from physical examination of the heart, unless systolic retraction at the apex, paradoxical pulse or a pericardial friction is present. The history of previous pericarditis and the absence of alcohol or syphilis in the antecedents might help us.

That weakness of the heart brought on by its adhesions to the pericardium should produce hepatic stasis and ascites without edema of the legs or anasarca is strange, but not at all without precedent in literature and experience. It has been repeatedly recorded in cases of valvular or parietal disease, but most frequently in adhesive pericarditis. I stated at the outset that there was little or nothing in the text-books upon the topic of my paper. This statement referred to the association of hepatic enlargement with adhesive pericarditis. That the latter often produces *ascites* is mentioned by Strümpell and by Leube in their text-books. The most acceptable explanation of this special localization of the passive congestion in the portal system is that the stasis in the liver gives rise to permanent tissue changes there, namely an increase of the stroma with contraction, over which the influence of rest and drugs has no effect. Other manifestations of passive congestion can be removed by treatment, but in the liver they are self-perpetuating and grow in a vicious circle.

Diagnosis.—Bearing in mind the possibility that any unexplained enlargement of the liver with portal congestion and ascites is due to adhesive pericarditis, we may be led to keep a daily watch upon the heart, and so happen upon some positive evidence of this

most obscure malady. This should be particularly insisted upon in cases occurring in young persons and in those where there is no evidence of excessive alcoholism, syphilis or malignant disease, or where there is a history of symptoms which sound like those of pericarditis, pneumonia or acute multiple arthritis. As most cases of chronic pericarditis are tubercular in origin, the presence of tuberculosis elsewhere in the body in connection with unexplained portal stasis should make us think of the pericardium as a possible cause for the stasis.

Prognosis.—The chief importance of correct diagnosis is that the course of cases of portal stasis due primarily to pericarditis is considerably longer, as a rule, than that of primary hepatic cirrhosis, for which they are often mistaken. The case here reported seems to have lasted at least six years; and a duration of eight to twelve years is apparently not uncommon.

Treatment is in any case simply palliative.

CONCLUSIONS.

(1) Some cases of hepatic enlargement with ascites and other evidence of portal stasis appear to be due to chronic obliterative pericarditis.

(2) Appreciation of this possibility may lead to the correct diagnosis through careful and frequent examinations of the heart, and close scrutiny of the previous history.

(3) The disease appears to be relatively frequent in persons under thirty years of age, and usually runs a course of six to twelve years, that is, a longer course than most cases of primary alcoholic cirrhosis. This fact is of importance in prognosis.

ON THE TREATMENT OF CERTAIN FORMS OF CERVICAL LYMPHADENITIS BY THE INTRODUCTION OF MEDICINAL SUBSTANCES INTO THE CRYPTS OF THE FAUCIAL TONSILS.¹

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The following paper is intended as a preliminary communication regarding a new method of treating some forms of cervical glandular enlargements which has suggested itself to me as the logical deduction from certain clinical facts and experimental investigation.

The cervical glands, to the enlargement of which I refer, are those situated in the neighborhood of the angle of the lower jaw. It should be emphasized that the other cervical lymph-glands, such as the occipital, jugular, and submental, are here left entirely out of account.

The angular glands, to which our present consideration is limited, become enlarged, as is well known, in a variety of acute and chronic pathological processes affecting the fauces and the posterior portions of the mouth, such as, for instance, diphtheria, epithelioma of the tonsil, dental caries, etc.

In the present connection such possible causes as acute inflammation, malignant disease and dental irritation are excluded. There will, then, remain a class of angular glandular enlargements which possess particular interest and importance. These are the chronic

¹ Read before the Boston Society for Medical Improvement, January 24, 1898.

⁶ Practitioner, February, 1887.

⁷ Deutsch. Archiv. f. klin. Med., 1895, p. 272.

⁸ Fortschritte d. Med., 1887, p. 380.

⁹ Beiträge zur Leberkrankheiten, 1864, p. 19.

¹⁰ Cited from Pick, loc. cit.

¹¹ Deutsch. med. Woch., 1882, p. 601.

¹² Loc. cit., 1884, p. 564.