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CASE

OF

ULCERATIVE ENDOCARDITIS WITH PYÆMIA; DEATH FROM PERFORATION OF THE HEART.

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[Read June 5, 1878.]

M. S., æt. 34 years, was admitted to the Medical Ward of the Philadelphia Hospital on a Monday in April, 1878.

The patient had the appearance of being well nourished; was a washerwoman; was married; had had two children; and stated that she had never been sick before in her life. Rheumatic history was carefully searched for, but none obtained; she denied ever having had syphilis, and no eruption nor scars were found to disprove her statement. She complained of obscure pains in the joints, and of muscular pains, but when questioned closely stated that the feeling was one of stiffness rather than pain, the symptoms all being of very moderate intensity. There was a rise in temperature. She was seen that evening shortly after admission by the resident physician, who, thinking the case to be one of muscular rheumatism, ordered her a dose of jaborandi. The following morning she expressed herself as feeling perfectly well, she had sweat profusely, and the temperature was nearly normal.

Owing to my absence, my colleague, Dr. Edward T. Bruen, who had temporary charge of the ward, and to whom I am indebted for the following notes of the case, after instituting

rigid inquiries which elicited the above history, made a careful examination.

Upon percussion the heart was found in its normal position, and not hypertrophied; the apex beat was normal. The pulse was regular, rather small, resembling that found in cases of mitral regurgitation. Auscultation revealed a basic, systolic murmur, but this murmur was transmitted downwards to the ensiform cartilage, and was heard louder at that point than at the base. There was also a murmur at the apex, and this was slightly transmitted to the axilla. The murmur was not found in the arteries of the neck, nor was either murmur heard in the back. The first murmur was blowing, low in pitch, and occupied the whole of the systole. At times the radial pulse became more feeble for one or two consecutive beats, and then the murmur was faint or entirely absent. The venous system was moderately engorged throughout, but there was no venous pulse. This moderate but still marked engorgement was more than could be accounted for by the apparently slight valvular lesion. The second sound of the heart was impaired; it was but feebly accentuated, and had not the clear, defined characteristics of health.

The lungs were normal throughout. There was no dyspnœa. There had never been hemorrhages. There had never been dropsy, nor had the patient suffered from headache. The urine was not albuminous. The patient stated that she had of late suffered at times from gastric irritability, and that this still remained. She was ordered the Tinctura Ferri Chloridi, and a pill of Digitalis gr. ss, Quiniæ Sulphat. gr. ss, Extract. Gentianæ gr. j, thrice daily.

The report on Thursday morning stated that the patient had vomited all through the night. The temperature on the previous evening had been very high, but was now, as will be seen by the temperature chart (Fig. 1), lower than normal. There was slight joint pain, no tenderness, and no other bad symptoms. Heart the same as before. Medicine discontinued.

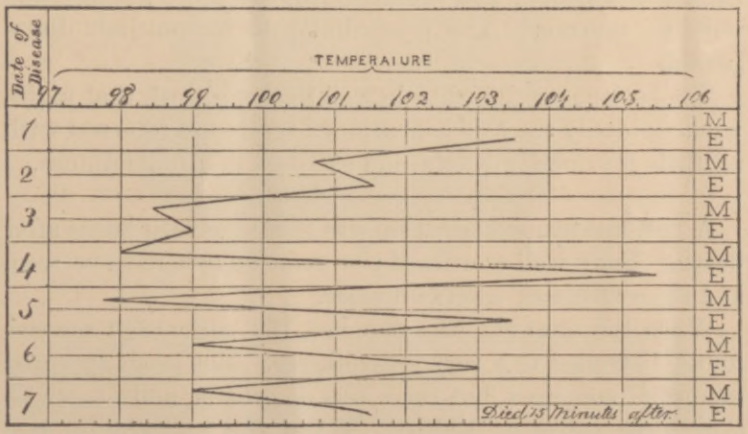
During the day the vomiting ceased, the pain disappeared,

and the patient felt comparatively well; in the evening the thermometer again showed a rise.

Friday morning.—Patient seems perfectly well. She asked to get up, but this was forbidden. In the evening the temperature again rose. She was unusually bright.

Saturday morning.—The patient sat up in bed about six o'clock to arrange her hair, when she complained of sudden and great dyspnœa, and intense pain. Shortly afterwards she became almost collapsed; the feet and hands were cold, and the body blue and covered with sweat; the facial expression anxious; and the radial pulse almost imperceptible. The respirations were very rapid.

FIG. 1.



When seen by Dr. Bruen, at ten o'clock, these symptoms continued unabated. He at once examined the heart, and found upon percussion a dulness reaching from the second to the sixth rib on the left side, and from the right sternal border four inches to the left. This broad area of dulness extended over the whole region of the pericardium. Surrounding this area of dulness, pulmonary resonance was clearly and sharply defined. Palpation revealed no apex beat. The sounds of the heart were scarcely audible, and were distant; but when distinguished, as they occasionally were, an absence

of murmur was noted. The respiratory murmur was harsh, but was heard distinctly everywhere. The diagnosis of extensive pericardial effusion of course was clear. The very sudden onset explained the gravity of the symptoms.

It will now be seen that as the temperature and vomiting indicated pyæmia, and the murmurs revealed an endocardial roughening, the diagnosis of ulcerative endocarditis with rupture of the heart seemed plausible. On this account paracentesis was deemed unnecessary. During the day the severity of the symptoms decidedly ameliorated, and at 5 P. M., when again seen, the patient expressed herself as feeling better; she was, and had been, perfectly conscious; the pulse could be felt at the wrist; the general surface of the body was warmer; and the heart sounds more audible, and with no murmur. The precordial pain had entirely disappeared.

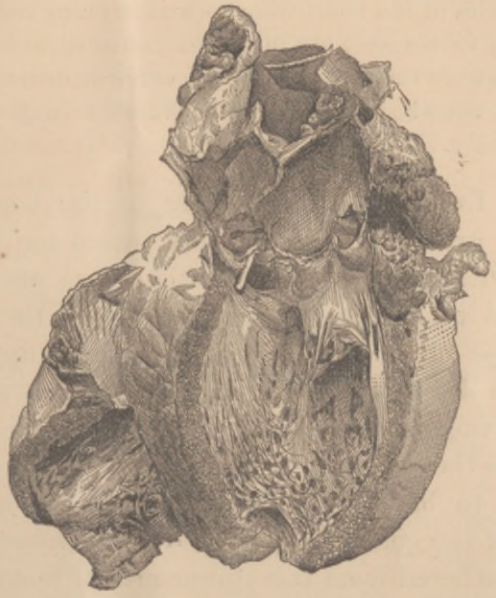
The patient continued to do well till ten o'clock that night, when suddenly the above-mentioned symptoms returned with all their previous intensity, and she died in a few minutes.

A post-mortem examination was made fourteen hours after death. Body well nourished; no scars anywhere to be seen, and no evidence of specific disease.

When the sternum was removed, the pericardial sac was found distended to a very great degree. The lungs appeared somewhat engorged, but there was no œdema, and otherwise they were perfectly healthy. When the pericardium was opened, about twelve ounces of fluid blood were removed, with a large amount of clots of various sizes. Adherent to the heart was a stringy clot, which, when traced to the point of its apparent origin, was found attached to a process springing from the base of the aorta posteriorly, at the junction of the aorta and the cardiac muscle. An opening was carefully searched for at the place where the clot was adherent, and one was found so small as scarcely to deserve the name, being merely large enough to allow the passage of a No. 8 needle. The process mentioned above was teat-like in

shape, and about half the size of an ordinary nursing-bottle nipple; it was composed of the outer sheath of the aorta.

FIG. 2.



The left ventricle being opened, presented a healthy appearance as far as the size of the muscular structure and its condition were concerned. There was no fatty degeneration, and no hypertrophy. The mitral valves were healthy; the aortic were sufficient; and the general appearance of the endocardium was healthy with the exception of a spot below the origin of two of the aortic valves, where there was a crater-like mass about the size of a dime, which with jagged edges surrounded an orifice communicating by a narrow sinus with the teat-like process before mentioned. This ulcer opened by a larger opening, probably big enough to transmit a lead-pencil, into the right auricle, just inside the attachment of the internal leaflet of the tricuspid valve. A mass of fleshy granulations, resembling in size that found in the

left ventricle, surrounded the opening here also, and that portion of it nearest the right ventricle had no doubt interfered so with the proper closure of the tricuspid valve as to give rise to the systolic murmur there heard. Otherwise the right cavities of the heart were normal in every respect. The pulmonary valves were not in the least affected, even by thickening, nor were the tricuspid valves at all diseased. No atheroma was anywhere visible. All the other organs were in perfect health.

Death from perforating ulcer of the heart is certainly of great rarity. But few cases are found on record, with an ante-mortem history so complete as to permit of, at least, a supposition of the originating cause of the occurrence. The symptoms recorded in the many reports of cases of heart-rupture, are very vague and obscure, relating as they all do simply to the rupture, not to the cause; the data that we have to base our clinical studies upon, are, the verdict of the coroner's jury and such information as can be gathered from the statements of friends.

Intrinsic diseases of the heart,[1] such as fatty degeneration, etc., are the usual causes of rupture, and as such pathological changes are found in persons past middle life, rupture of the heart is usually found at that age. Markham's[2] report of twelve cases of heart rupture, excluding those of traumatic origin, taken from the first seven volumes of the London Pathological Society's Transactions, shows us that the youngest patient was 52 years of age, the oldest 79. Seven were men, five women. In nearly all, if not in all, the coronary arteries were diseased. Quain[3] reports

[1] Schroetter. Ziemssen's Cyclopœdia of Medicine, vol. vi.
[2] Medical Times and Gazette, 1859.
[3] Lancet, vol. i. 1872.

88 cases—63 in persons over sixty, 33 between sixty and seventy, and the remainder between seventy and eighty years of age. He states that in many cases no symptoms whatever were noticed antecedent to the death agony. Out of 100 cases, death occurred in one or two minutes in 71; one patient lived eight days; one six days; one three days; and five over forty-eight hours. The heart was fatty in 77 cases, and in 6 "softened." In one case there was bursting of an aneurism, and in *one* the rupture was due to an abscess. Acute disease of the endocardium or of the heart substance may lead to the same result, by the formation of an aneurism through the yielding of the endocardium at any point, in the former case, or by localized myocarditis with the formation of an abscess, in the latter. In interstitial inflammation of the heart, the pus formed, following channels, may burrow in various directions. As the foci of inflammatory action are occasionally discrete, and oft-times limited in number, they have been called cardiac abscesses.[1] When they ulcerate through the endocardium or begin in that tissue, they form ulcers.

Senac,[2] in 1749, first pointed out that ulcers or abscesses of the heart were more frequently found at the base, referring at the same time to diseases of the pericardium and neighboring organs as their immediate cause. Deitrich in 1852 made the same observation. They usually occur between the ages of 20 and 40.[3] According to Schroetter, "*primary* disease of the heart substance is very rare. There are only a

[1] See an interesting article on this subject in Hayden, Diseases of the Heart and Aorta.

[2] Schroetter, loc. cit.

[3] Ibid.

few cases described where no possible cause could be ascertained." Syphilis is stated by the same writer to be a cause. The acute fevers, especially typhus, are among the many causes that have been assigned for abscess of the heart and ulcerative endocarditis, but rheumatism, according to most authors, is the most frequent. The puerperal state has an equal tendency to this result, and the so-called metastatic abscesses are found in all active muscular organs having poor nutrition. Pyæmia is then the most prolific cause.

But ulcerative endocarditis, according to Lancereaux,[1] is not always an off-shoot of the rheumatic process; the high temperature, vomiting, and chills, with sudden syncope, which may disappear or may be followed by death, are really the symptoms of the concurrent pyæmia. Lancereaux believes that purulent ulcers of the endocardium may be the result of intense malarial poisoning, but that they are then never situated on the valves themselves, but occupy the heart lining at the valvular attachments, and produce death either by perforation or by pyæmia. In support of his view he quotes the observations of Winge and Heiberg, verified in one case by Virchow, where vibriones of filiform shape were found in the ulcers.

It may be interesting to glance over the cases reported that bear upon this intricate subject, and endeavor to gather what we can to complete its etiology.

As an example of secondary abscess of the heart caused by metastasis or thrombosis, may be cited the case reported by Dr. Moxon,[2] of a child at Guy's Hospital with suppurative periostitis, where death resulted

[1] De l'Endocardite végétante ulcereuse; Archives Générales, 1873.

[2] Medical Times and Gazette, vol. ii. 1872.

from multiple cardiac abscess. There were abscesses in the kidneys also.

Dr. Inman[1] reports the case of a man of 35, who had complained of "ague-shakes" for three or four hours daily, during seven or eight months. There was no malarial history. Nothing wrong could be detected with the heart or lungs. The patient's intellect seemed affected as in the early stage of typhoid fever, and he had a yellow, jaundiced complexion. The man died suddenly. After death there was found an extensive abscess at the base of the pulmonary artery, and this abscess communicated with the right ventricle behind one of the valves of the artery. Around this opening a fleshy vegetation existed about the size of a horse-bean. The lungs contained diffused and infiltrated pus. This patient's case was evidently one of primary suppuration of the gland in that vicinity, from which pus had been thrown into the venous current for some time.

The most interesting case, and the only one that I can find analogous to that which I have had the honor to report, is one given us by Heslop,[2] of a girl aged 18. There was no heart murmur, but the action of the organ was tumultuous and irregular. On the patient's admission to the hospital, she stated that she had had rigors, followed by flushes of heat, but never any rheumatism. She was a weak, anæmic girl, and died in convulsions. The part of the endocardium immediately beneath the semilunar valves (in the left cavity[3]), presented an

[1] Ibid. vol. i. 1862.

[2] Medical Times and Gazette, vol. ii. page 245, 1856.

[3] Hanska (Medical Times and Gazette, 1855) is quoted to have shown, by 300 examinations of hearts, that there was in the normal septum a spot varying in size from a bean to an almond, entirely destitute of muscular

irregular, ecchymosed surface, and had the appearance of being undermined, leading to the base of the aorta. At the attachment of the middle and anterior segment of the valves, was a mass of fibrinous deposit the size of a small walnut, surrounding a cavity containing a recently formed coagulum, pus, etc.: it did not perforate. It was noticed that two days before death a continuous blowing sound was heard, accompanying and masking both first and second sounds. The patient had vomited at the commencement of the attack, and there was epigastric tenderness; rigors set in early, and the skin was jaundiced. Death took place in all probability from pyæmia.[1]

Greenfield notes, for Dr. Murchison,[2] a man aged 56, in whom the temperature from May 7 till June 1 varied, with nightly exacerbations, from normal to 105° Fahr. The rigors occurred at irregular periods, sometimes more than once daily. The mitral valves were thickened, and the aortic slightly affected; the tricuspid valves also were thickened. There was a granulating, white mass, an eighth of an inch thick and a quarter of an inch long, on the border of one flap; the heart was the seat of fatty degeneration.

substance, the two chambers being there separated only by the layers of endocardium that line them. Examining the septum from the left, after slitting up the aorta, we may remark a thin diaphanous spot under the angle formed by the convex borders of the right and posterior semilunar valves of the aorta, being closed above by a thin musculoid bundle, coursing along the contour of the ostium arteriosum sinistrum. In the right ventricle, the deprivation of muscular substance is covered by the end of the tricuspid valve, and so thin is the duplicature of the endocardium that the lines and markings of the fingers held under it can be seen through.

[1] In Watson's Practice I find it stated that "a Duchess of Brunswick died of rupture of the heart. In her case an ulcer penetrated the parietes of the right ventricle, which in other respects was healthy."

[2] Lancet, vol. i. 1873, page 909.

Cases have been recorded where friction was suggested as a cause of endocardial ulcer, by Dr. Hodgkin, Dr. Hilton Fagge, and others. Coupland[1] records a case with an aneurismal pouch starting from a nodule on an aortic valve which directly faced it.

In this short *résumé* of a subject about which really so little has been written, it will be seen that ulcerative endocarditis is looked upon as *secondary* to various affections and diatheses; as a *primary* disease it must be looked upon as rare, particularly when limited to one spot, as it was in Heslop's case and in that which I have reported; in fact, in the above cases it may even be attributed to a cachexia, where a focus of inflammation started either in the connective tissue of the heart, or in the endocardium, resulting in an ulcer which, small as it was, induced pyæmia. Suppurating processes near the heart may involve it in their progress, as we have already seen; again there is no reason why metastatic infiltration or infectious embolism, may not occur and give rise to phenomena so intense as to mask the primary irritation; or thrombosis of the cardiac veins or of atheromatous coronary arteries, may be a cause, in persons advanced in years. Malaria, from pigmentous deposit or otherwise, may give rise to embolism, or, if the germ-theory be accepted, the emboli may be charged with bacteria. But notwithstanding all these varied causes assigned by authors, undoubtedly some cases exist unaccounted for, and that which I have had the honor to report stands boldly forth as an example.

In a strong, healthy woman, bearing evidence of no

[1] Lancet, vol. ii. 1875.

previous disease, with all the other organs in perfect health, a small ulcer situated in healthy tissue demands an explanation which none of the cases I have cited throw light upon. The process without doubt had been going on for some time. Could some congenital malformation, some previous strain, or the puerperal state, have laid the foundation of disease at a point where the blood current was directly and forcibly impinging; or shall we attribute the rupture to an abscess of a lymphatic gland within the heart substance? But take it as we may, it serves to prove that, as far as we know, such cases can occur spontaneously, or rather idiopathically.

Among the many points of interest in these cases, we have the temperature. A small quantity of pus, measured by the drop alone, gave rise in this case, and in others I have cited, to the most marked symptoms of pyæmia, viz., hectic, vomiting, and, in some, jaundice, chills and convulsions.

In the patient spoken of by Dr. Inman, the pyæmic symptoms had lasted many months, and the fleshy vegetation at the cardiac opening of the abscess showed that pus had been intermingling with the blood for some time. The same existed in Heslop's case, and also in mine.

The only symptom then that marks this disease is pyæmia—pyæmia coming on suddenly, and usually associated with some cardiac disturbance, or else with embolic infarction of other organs. Of course the symptoms of pyæmia are severe[1] in proportion to the amount of pus entering the circulation, or to the non-resisting power of the nerve centres to its poisonous

[1] Medical Times and Gazette, October, 1877.

action. Chance[1] speaks of a boy aged 13, who, apparently in perfect health but with a scrofulous diathesis, was attacked one day immediately after eating with nausea and vomiting. The next day he became drowsy, and complained of feeling very sick with pain in the stomach. Finally, complete coma set in, with rapid and fluttering pulse, and occasional convulsive movements. He died in two days from the beginning of the attack. There were multiple abscesses of the heart, with perforation and pericarditis. Here nothing was observed until perforation into the pericardium had taken place. But in all cases where ulcerative endocarditis has been found, a rise in temperature has been noticed.

Jaundice may be said to be dependent on the length of the attack; in those where pus was pouring into the circulation for some time, it was always noticed, but not otherwise.

Vomiting has always been present, both in the very acute and in the more prolonged cases, though usually it has been more frequently seen in the former, as tolerance seems to have been established in the latter cases. I would call attention to the fact that in Heslop's case there was no stupor characteristic of a typhoid state, nor septicæmia, but that the patient died in convulsions. In the case reported by me there was consciousness to the very last. Patients dying of puerperal pyæmia also exhibit this peculiarity.

As regards physical signs, there is nothing that will aid us in diagnosis until perforation takes place. If the abscesses are numerous or confluent, irregularity

[1] Lancet, vol. i. 1846.

and tumultuous action of the heart may aid us. If ulceration takes place, murmurs will be produced, differing in character and position from those of valvular lesions, unless the action of the valves should be interfered with. It may be well to state here that old valvular deposits, from rheumatism or other causes, may become caseous and break down,[1] causing at times pyæmia or embolic infarction in other organs (*secondary* ulcerative endocarditis). The murmurs heard in such cases would of course be limited to the valves affected. The corporeal endocarditis of acute specific fevers, such as puerperal or scarlet fever, or pyæmia, may lead to ulceration, and ulcers will then form on the papillary muscles, often eroding them.[2]

In Greenfield's case, there was heard at the ensiform cartilage and over the lower part of the sternum, a loud, systolic murmur which became fainter towards the left, but was replaced on that side by a rough, systolic murmur, apparently distinct and conducted to the angle of the scapula; all the valves, as we have seen, were affected more or less, and hence the mitral regurgitant murmur; the tricuspid murmur was aided by the fleshy mass spoken of. In Heslop's case there

[1] See case by Pepper, Transactions of the Philadelphia Pathological Society, vol. iv.

[2] Dr. Harrison Allen, in answer to a note from me, kindly gives me his opinion on this subject, as follows: "The researches of Schweigger-Seivel show that lymphatic vessels are in abundance beneath both pericardium and endocardium, and from these two localities freely communicate by irregular spaces in the muscular structure of the heart-wall. I presume that an analogous arrangement exists in the septum between the endocardial surfaces of the right and left hearts. The glands are outside the cardiac figure, at the base. I could not localize an abscess at the base of the ventricular septum by reason of any known disposition of lymphatic glands or vessels."

was no murmur, but the action of the heart was "tumultuous and irregular." In my case, the aortic valves showed no insufficiency, but at times there was slight systolic roughening; the tricuspid murmur was well marked, but the feeble mitral-systolic murmur was not transmitted to the back, and may have been due to the granulations changing the course of the blood current.

In conclusion, then, I think that we can safely assert that *primary* abscess of the heart, or primary ulceration, if it proceed far enough, independent of general myocarditis, is occasionally found.[1] I cannot offer any explanation except that it may be due to disease of the lymphatic channels or the glands. I have no doubt that many of the cases recorded as secondary ulceration, and attributed to multitudes of causes, were in reality cases of this kind occurring in persons of strumous diathesis. Can we proceed farther, if we accept this theory, and give credit to small abscesses of lymphatic origin, in the heart or arteries, in early life, for the starting point of many of those obscure cases of aneurism where the absence of atheroma is noted?

Of course little is to be said under the head of treatment. When the cases come to us they are usually beyond our aid. Cases have gotten well, and, after death from other causes, calcareous nodules have, it is said, been found imbedded in the heart muscle.

[1] Hayden, Diseases of the Heart and Aorta; Article on Myocarditis.