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MALIGNANT ENDOCARDITIS.

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THE following cases are illustrative of that form of endocardial inflammation which it seems best to speak of under the title given above. After the masterly studies of Bouillaud and Kreisig, making the recognition of acute inflammation of the endocardium possible, there was recognized but one form of endocarditis, though, of course, the appearances of the lesion were known to vary. Kirkes, however, in 1851, first pointed out the existence of a variety, differing from that most commonly observed, and which has since been variously known as ulcerative, mycotic, necrotic, diphtheritic, and malignant endocarditis. The last name is preferable because it expresses very well the essential features by which this form of endocarditis differs from the ordinary simple or benign. The careful pathologic and bacteriologic work of recent years has shown that there is no sharp dividing line by which the benign cases can be separated from the malignant; and the coincident increase in our knowledge of the clinical features of these important diseases has similarly shown that in this direction also there is no separating line. Those terms which are based upon supposed anatomical differences fall short of satisfaction; and particularly must we set aside the name ulcerative, which is, perhaps, the most commonly used, in that it indicates but one form, or manifestation, under which malignant endocarditis may present itself. It is improper, for the same reason to speak of necrotic or diphtheritic endocarditis and the etiologic term mycotic must fall, since micro-organisms have been found in cases which are unquestionably of the simple or benign type.

The following cases are of particular interest in illustrating a clinical type of malignant endocarditis which is more common than is usually supposed, the form to which German authors have given the name chronic recurrent endocarditis. It is a form in which an acute process becomes engrafted upon an old disease of the valves. It may result, as does primary malignant endocarditis, from infection in the course of pneumonia, diphtheria, scarlet fever, small-pox, or other diseases of a similar nature; but it may also occur in an insidious manner and

without any definite discoverable cause. The onset, therefore, may be either abrupt or it may be gradual. In the cases reported here the gradual type is illustrated. In none of them was a distinct cause detectable; in each an old endocarditis was known to exist. The symptoms were few, but sufficiently marked to make it possible to establish a proper diagnosis. Autopsy was performed in two of them, and a demonstration of the suspected morbid lesions thus obtained.

The features by which this form of endocarditis is recognized are the gradual development of irregular fever, for which no adequate explanation can be detected, and which is usually accompanied by excessive sweating, and oftentimes by chills. The patient's general condition more and more assumes the appearance of a septic state. There may be enlargement of the spleen; there may be purpuric eruption; there may be diarrhea; albumin, blood, and even tube casts may appear in the urine; sometimes the joints are swollen and red; finally and conclusively, as was the case in two of the three here reported, embolism not infrequently supervenes. Of course, embolism does occur in cases of simple endocarditis, but where there is a general septic state with irregular fever and sweats the occurrence of embolism and infarction is sufficient to direct suspicion upon the endocardium as the primary focus from which the general infection and intoxication have taken origin. The course of cases of this description is exceedingly variable; sometimes they rapidly grow worse and worse and death may occur after but a few days or weeks; in others the duration is stretched over a period of months; and cases have been observed in which the final termination was delayed for over a year. In these prolonged forms there is constantly a considerable degree of variability of the symptoms and, from time to time, such subsidence of the latter that there may seem to be an arrest or even recovery. Indeed, it is not impossible, and cases have been reported to sustain this position, that the final outcome may be the restoration of the condition of the patient to that present before the attack began, or with but slight increase of the chronic valvular disease. From our present views regarding the pathology of acute endocarditis it would naturally be expected that border-line cases exist which, though presenting to an extent the clinical features of malignant endocarditis, are of sufficiently benign character eventually to terminate in recovery. In other words, these cases exemplify what was said above,—viz., that no sharp dividing line exists separating the simple from the malignant forms.

CASE I.—W. A., aged 25 years, was admitted to the University Hospital May 3, 1892. The patient was born in Poland. His father, mother, and three sisters were in good health. He had had small-pox when quite young and scarlet fever, but no other illness until six

months before, when he had a severe attack of rheumatism with implication of the joints, sore throat, and considerable fever. The course of this attack was very irregular; one joint after another being involved at intervals of a few days or weeks. He denies a specific history entirely; has never been a hard drinker; does not use tobacco. But his occupation—that of a coal-miner—exposed him to cold and dampness, and was doubtless the immediate cause of the rheumatic attack. Immediately following the latter, he began to have uncomfortable feelings about his heart with, sometimes, severe palpitations. There never was any edema of the feet or hands.

The note made on the day of his admission reads: The patient is pallid; has decidedly club-shaped fingers, which he says have been present since childhood. The apex-beat is displaced outward an inch beyond the mammary line, is forcible and somewhat irregular. On auscultation there is found at the apex a presystolic followed by a sharp systolic murmur, the systolic being transmitted to the axilla. At the aortic region there is a loud diastolic murmur. The patient is somewhat short of breath, but examination of his lung reveals no abnormal physical sign. The temperature is normal; the pulse somewhat collapsing.

During the first few days after admission, he complained of vague pains in the muscles and joints, and at the end of the first week there was a sharp rise of temperature. After this time, and until his death, September 5, there was at no time normal temperature, the range being from 99° to 103° F., higher in the evening than in the morning. The daily excursions averaged about 2° to $2\frac{1}{2}^{\circ}$ F., and were sometimes as great as 4° F. He continued to complain of vague rheumatic pain, of headaches, and of excessive sweating now and then. The heart-action continued excitable and dyspnea was constantly present, increasing and decreasing with the temperature, though out of proportion to the latter. The range of the respiratory curve was from 20 to 40 inspirations per minute; and shortly before the termination the number reached 50. There was little change in the patient's condition from day to day, but his strength gradually decreased; his color grew more and more pallid; and the heart-action less vigorous. In the latter end of August, there began to be mild delirium in the evening and he complained that other patients were talking about his approaching death, and generally he grew more and more suspicious and irrational. Physical examination revealed a little dulness at the base of the lungs posteriorly and there were a few soft râles. This increased towards the end. He died, on September 5, of heart-failure.

The diagnosis in this case was attended with some difficulty at first. At his entrance to the wards, and for a week thereafter, there

was neither fever nor any other indication of general intoxication ; but simply dyspnea and the ordinary indications of lacking compensation in chronic valvular disease. At the end of the week, however, fever set in, and the man became evidently more ill. In the mean time he had been in bed, there had been nothing to induce the infection of the valves that afterwards became so clearly evident. There was, however, some complaint of pain in the joints and members, that not unnaturally suggested recurrence of rheumatism, though at no time did any of the joints exhibit evidences of active inflammation. Not, therefore, until time had made it clear that these articular manifestations were too slight to justify the belief in their being the cause of the patient's general state was it recognized that some underlying cause existed which had thus far eluded discovery. Physical examination did not furnish any data upon which an opinion could be founded ; for the valvular murmurs continued exactly the same as they had been ; and the other organs seemed entirely healthy. Meantime, however, the continuous septic state of the patient persisted ; the heart-power grew weaker and weaker, and thus the conclusion was forced upon us that the underlying lesion was a malignant endocarditis. This may have been of rheumatic origin, or in some way dependent upon the exposure his occupation necessitated, but the subsequent rheumatoid articular pains, which persisted throughout the entire duration of the case, were more likely of the nature of the joint-pains that not infrequently associate themselves with septic conditions of various kinds. The duration of the case, four months, was remarkable, but, as has been before noted, much more protracted cases are recorded.

Autopsy.—(Only the points of interest in relation with the cardiac condition are extracted.) The pericardial sac contained 400 cubic centimetres of clear serous fluid. There were a few old and some recent fibrinous adhesions binding the two layers of the pericardium to each other. The heart was very greatly distended and weighed 770 grammes. The hydrostatic test showed the aortic leaflets to be incompetent. The left ventricle was greatly hypertrophied and dilated. The mitral valve segments were markedly thickened and the orifice narrowed. On the ventricular face of the anterior mitral leaflet there was a large ulcer covering it almost completely and extending upward to the aortic valve. Two of the segments of the latter were more or less completely destroyed, leaving jagged stumps. There was deep ulceration in one of the sinuses of Valsalva. The auricular surface of the mitral valve was not involved. Several of the chordæ tendineæ of the left ventricle were destroyed by ulceration, but there was no involvement of the right side of the heart. There was, therefore, old valvular thickening affecting the aortic and mitral leaflets, and recent malignant endocarditis of the same valves and of the neural endocardium.

Old scars were found in the spleen and kidneys, and in the right kidney also recent infarction.

CASE II.—J. H., aged 19 years, was admitted to the University Hospital March 27, 1893. His *family history* furnished no important data.

The notes on admission to the hospital read,—*Previous history*: He had measles at 5 years of age, after which he was healthy until he was 12 years old, when he got a severe cold from working in the wet as a plumber's boy. This ended in an attack of rheumatism in his knees and ankles, and kept him in bed eight days. About a week after this he noticed a bulging of his left chest over the precordial region. He had never noticed this before, and there was no pain in that region. About six months later, while lifting something, he struck his chest and suffered considerably from this, so much that he consulted a doctor, who first told him he had heart-trouble. He had grippe three years ago, and after it for a short time had shortness of breath and palpitation on any exertion. About four months later he had severe epistaxis, which lasted six hours and required plugging of his nares for three days. After this he was in a hospital for two weeks, and at this time his feet were swollen and sore and weak. Following this and until last October he has been fairly comfortable.

The *present illness* began last October with another attack of rheumatism, which lasted nearly six weeks (three weeks in bed). Ever since then he has had occasional slight attacks of rheumatic pain and continual headache and shortness of breath. Phosphenes appear before his eyes, and when he lies down he hears his heart-beats. It sounds like running and dropping water to him. He has an occasional sharp pain over the heart, but not very often or severe.

Physical examination showed a marked bulging over the precordia, with a very extensive apex-beat; practically the whole chest wall anteriorly pulsated with each heart-beat. On auscultation there was found a distinct, loud, systolic murmur at the apex, transmitted into the axilla. At the aortic interspace there was a slight systolic murmur, but it was difficult to determine whether this was transmitted or whether it was followed by a diastolic murmur. The patient's temperature was normal; the pulse varied from the normal to 100; the respirations from 20 to 30 or occasionally as many as 40 in a minute. There was slight cough. The patient remained in the hospital about three weeks, during which time his condition was never bad, and he was repeatedly used for class demonstration. He left in very good condition.

On July 14 he returned to the hospital, complaining of increased pain over the region of the heart and epigastrium, and he had decided dyspnea. The temperature when he was admitted was 101° F.; the

pulse 112; respirations 30. There was found on physical examination a somewhat loud, mitral, systolic murmur, and a diastolic bruit over the aortic region, and particularly over the lower part of the sternum. After a few days there was detected a to-and-fro friction-rub at the base of the heart, which, however, was only occasionally present. The temperature remained between 100° and 101° F., but sometimes dropped suddenly to the normal and as suddenly rose again to its previous height. The patient constantly complained of drenching sweats, which were particularly annoying when he awoke from sleep. Several times there was slight chilliness, but no pronounced chills. On July 21 it was noted that there was a sudden painful swelling on the inside of the ring-finger of the right hand, which was hard and somewhat purpuric, the size of a small marble. At the same time a similar lesion more advanced was discovered on the inside of the left foot. There was considerable epicardial pain. On the 22d the patient complained of great pain over the region of the spleen, which was found considerably enlarged. There were looseness of the bowels and albumin in the urine, but no tube-casts. The great toe of the right foot showed a small infarction in the same way as the finger previously noted. On the 24th there was great pain in the abdomen, particularly in the splenic region; the abdominal muscles were tense and tender. On the 26th the temperature fell to subnormal, and there was every evidence of collapse, in which condition the patient remained until the 31st, when he died.

In this case the diagnosis offered less difficulty than in the last. The patient had been under observation with definite indications of old-standing cardiac disease in March, and had then presented neither fever nor other evidences of acute disease. At his next admission to the hospital there were decidedly irregular temperature, great sweating, and the general appearances of a systemic intoxication. A loud, diastolic, aortic murmur was audible. This had never been heard during the first period during which he remained in the hospital, though aortic regurgitation was then thought present and careful examinations repeatedly made. No lesion of any other organ was discovered; but the occasional pericardial frictions now entered as discordant facts to confuse the diagnosis. It is true the fever of pericarditis is not of the type found in this case, nor would a fresh pericarditis be apt to cause other systemic symptoms such as were seen in this case; but still there was unquestionably an acute process here which was capable of inducing fever. Much light was therefore shed upon the case by the occurrence, a week after the patient's entrance into the ward, of a distinct embolic lesion of the finger, and the discovery of an older one on the foot. There could now be little doubt but that an acute inflammatory lesion

existed within the heart; and this grew practically to a certainty when the evidences of splenic and renal infarction supervened. The previous history and the general constitutional symptoms further made it almost positive that the endocardial lesion would prove malignant.

At the autopsy the following conditions were discovered: Pleuro-pericardial adhesions bound the pericardium in all directions, and rendered its detachment from the chest-walls exceedingly difficult. A moderate quantity of pericardial effusion was noted, but the epicardium and the pericardium were so intimately attached in places that their separation was difficult. Both layers were greatly thickened, and recent fibrinous exudation covered their surfaces. The heart, pericardium, and enclosed clots weighed 1830 grammes. The cavities were enormously dilated and the walls hypertrophied, but the muscle was soft and flabby and pale in color. The weight of the heart alone was 990 grammes. There were fresh granulations on the mitral, pulmonary, and aortic valves, and thickly set in the wall of the left auricle. A large eroding ulcer was found on the aortic segments involving two of the leaflets. Ulcerations were also noted on the mitral valve and in the left auricle. There were none in the aorta. The aortic and mitral valves were thickened and sclerotic. The spleen was enlarged (weight 500 grammes) and contained a large fresh infarction, with the remains of an old infarct showing softening. The right kidney contained several large and small recent infarctions; the left was free. The liver was congested and enlarged.

CASE III.—P. L., aged 24 years, was admitted to the University Hospital on February 1, 1892, when the following notes were made: The patient was born in Germany. He has been a healthy person until last summer, when the present illness began, and he has since been unable to continue his work. He has been in this country seven years, working first as a driver in the mines, where he was considerably exposed to cold and dampness, but lately has had more suitable occupation as a mason.

It is difficult to obtain a satisfactory history, but he asserts that last summer he had fever, pain, and swelling of the ankles, and excessive sweating.

On admission to the hospital he was found to be extremely anemic and suffering with vague rheumatoid pains in the ankles, knees, and hands. There were no evidences of acute inflammation of the joints, though they were somewhat enlarged. There was no fever. The ends of the fingers were club-shaped and cyanosed.

On physical examination there was found great hypertrophy of the heart, the apex-beat being displaced far to the left and the impulse being strongly marked. There was a long, rumbling, presystolic

murmur at the apex, and coincidentally with this a palpable thrill. Immediately following this murmur there was a short, puffing, systolic bruit, best heard at the apex, but also quite distinctly in the axilla and posteriorly. At the aortic region there was a loud systolic murmur, and the second heart-sound was faintly audible. No diastolic murmur could be heard. The lungs were normal to percussion, but on auscultation dry and moist râles were audible in all parts, particularly at the bases posteriorly. The liver was slightly enlarged. The urine was scanty, highly acid; its specific gravity 1025, but it contained neither albumin nor sugar. Examination of the blood showed 4,500,000 red corpuscles and 50 per cent. of hemoglobin. There was no leucocytosis. The patient's condition remained unchanged during the first week he was in the hospital.

On February 7 the records read: There was an attack of sudden and severe pain in the splenic region this morning; but there is so much tenderness the spleen cannot be felt or outlined. There has also been some increase of the cough and bloody expectoration. Physical examination of the lungs reveals nothing but some fine liquid râles at the bases posteriorly. The air seems to enter all parts of both lungs with uniform ease. The patient's general condition now remained stationary for several weeks; but it was noted that the evening temperature was generally elevated to 100° or 101° F. On February 29 there was another attack of pain in the splenic region, and the spleen was now palpably enlarged. Cardiac excitement and dyspnea accompanied the attack. After a few days the splenic enlargement seemed to have subsided entirely, but the patient complained that he could feel a constant sense of pulsation in that region. About the middle of March attacks of excessive vomiting and of epistaxis are noted, and the patient's condition had steadily grown worse. The blood examination showed 4,300,000 red corpuscles and 45 per cent. of hemoglobin. There were many nucleated red corpuscles. The irregular mild fever continued, and there was profound prostration. The latter part of March there was considerable complaint of pain in the chest. The right base was dull on percussion, and on auscultation the breath-sounds in this region were exceedingly weak. The patient constantly expectorated bloody sputa. In the early part of April the patient died. Autopsy was not permitted.

Though the lesions in this case were not demonstrated post-mortem, the great similarity of the clinical features in this and in the first two will be recognized, and the diagnosis can therefore scarcely be doubtful. For some time after his entrance to the hospital there were no evidences of an acute disease. Suddenly infarctions developed, and the man showed evidences of a general toxemic state. The duration of the case was at least two months.