MALIGNANT ENDOCARDITIS, WITH NOTE OF A CASE IN WHICH DEATH WAS CAUSED BY EMBOLISM OF THE RIGHT CORONARY ARTERY.

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In addressing myself to the subject of malignant endocarditis I shall not presume to do much more than offer some commentary on a few cases which have recently come under my notice.

I shall first ask your attention to the clinical and pathological phenomena of a case which has lately been under my care.

Case I.—M. B., a single woman, twenty-seven years of age, a woollen weaver, was admitted to the Whitworth Hospital on the 8th September, 1890. She stated that two years ago she had had pains in her knees, and had been laid up for three weeks. Three months before admission she again began to suffer from pains in the arms, legs, and body, unaccompanied by any joint swellings. The menses, previously regular, had been absent for four months. She was of slight, but not thin, build, of fair complexion, and anæmic appearance. On examination she was found to have a loud blowing systolic apex murmur, with accentuation of the second sound in the pulmonary area, and the apex beat was displaced slightly downwards and outwards. She complained of pains in her feet, which were very slightly œdematous behind the malleoli. Within a week after admission the pains and œdema disappeared, but it was easily seen from her aspect and temperature that her freedom from discomfort was no measure of her freedom from disease.

On Sept. 25th there was noticed over the trunk a small dark-red papular rash, which disappeared in a few days.

Sept. 29th.—An apex presystolic murmur was noticed.

Oct. 14th.—The systolic murmur can be well heard in the aortic area and at the back.

Oct. 26th.—She has had purpura on the right gluteal region and on both legs and feet for the last few days. She tells me that for a month past she has had frequent momentary chills. She was of such patient temperament that it was only by questioning that we elicited facts which would have been grounds of complaint in a less grave case.

Oct. 27th.—The purpuric eruption is on abdomen to-day. There is tenderness in the epigastric region. The spleen can be felt below the ribs to the extent of two fingers' breadth during inspiration. Liver dulness extends higher than usual by about a rib's breadth.

I may summarise the further history of the case. The patient was kept in bed from her admission till her death on November 18th. Until the last two or three days she had no œdema, but such slight fulness of the lower eyelid as one not uncommonly sees in anæmia. She made no complaint of pain; disliked soups, and preferred to take a little meat. She had no diarrhœa, no sweats, no jaundice, no difficulty in breathing, no cough, and no delirium. She became of a somewhat chlorotic aspect, while her lips gradually assumed a bluish pallor. The temperature throughout was of irregular pyrexial type, and will be seen recorded in the accompanying chart. She died by exhaustion ten weeks after admission and over five months after the onset of her illness. I was never able to get any account of an external wound or internal lesion forerunning this illness; but it is obvious that the channels by which septic infection can enter the body are so numerous that it would be impossible to assert of anyone, no matter how healthy-looking, that he was free from such a danger.

Autopsy, fourteen hours after death.—A well-marked purpuric rash all over the body, thickest on the left side of thorax and abdomen, the side on which the patient had lain constantly for some days preceding death. There was slight anasarca. There was a little fluid in the pleural cavities, and a somewhat more than normal quantity in the pericardium. The lungs were congested, and showed a small area of induration, with one or two caseous spots in each apex. The liver was somewhat enlarged and nutmeggy. The spleen, enlarged and soft, showed a few infarcts. The kidneys contained also a few small infarcts. The stomach showed several spots of submucous hæmorrhage. The heart muscle

was œdematous; all the cavities somewhat dilated. The right side endocardium normal. The left auricle has a large patch of soft vegetations on its postero-external wall. This patch is continuous below, with a similar patch on the posterior mitral valve. The anterior mitral valve is thickened, with large, soft, greyish vegetations over all its auricular and the lower half of its ventricular surface; it is apparently divided in two right up almost to its base. It has very few chordæ tendineæ attached to it, and these are thickened. Several other chordæ are hanging free from the valve; some are beaded and some are attached to one another.

This case was diagnosed as one of malignant endocarditis from the period when we found that, notwithstanding the disappearance of all pain, the irregular pyrexia, as seen in the chart, persisted. The changing character of the cardiac murmur strengthened our opinion. The frequent rigors added further confirmation, and the term malignant was amply justified by the issue.

The foregoing case set me thinking how far malignant endocarditis is a morbid entity, how far it is definable by the pathologist, and how far recognisable clinically by the physician.

Taking the clinical question first, I may ask—How is one to know a case of malignant endocarditis? I shall attempt an answer. If a patient with a suppurating wound, a bone necrosis, a pneumonia, a puerperal metritis, an intestinal ulcer, or such like cutaneous or mucous lesion, presents a cardiac murmur, a pyæmic temperature curve, frequent rigors, embolisms which set up suppurations, and if it can be ascertained that the cardiac lesion has occurred subsequently to the peripheral lesion, and if the murmur shows any tendency to change, whether in position, in time, or in character, then such patient probably has a malignant or septic endocarditis. But the physician who should wait till a majority of these signs had presented themselves would soon have his doubts as to malignancy set at rest. The question is, how to recognise the disease before it has loomed completely out of the fog. By what lights shall we know the patient's proximity to danger? Most of the characters I have detailed would fit the diagnosis of pyæmia without cardiac lesion, and moreover most cases of malignant

endocarditis lack one or many of these characters. Which of them then are the essential ones? I would say the cardiac murmur and the irregular pyrexia—any of the others may be undiscoverable. Nay, even of these two I venture to suggest that perhaps one may be absent throughout, as we now recognise that it may be for a time—I mean the cardiac murmur. Diagnosis under such circumstances will not be easy, but it may be possible. The peculiar pyrexial curve will probably be the ultimate essential in the diagnosis. Or to make a practical rule:—The occurrence of a pyæmic temperature curve in the absence of evidence of other causation ought to hint at the possibility of the existence of this form of endocarditis, more especially if we know that the patient has previously had any disease which might have induced sclerosis of his heart valves.

But, it may be asked, what is the use of retaining a term like malignant, or any of the other names applied to this form of endocarditis, if the condition is but a part of a systemic pyæmia? In the present state of our knowledge the term is still of use. We are unable to make a natural and scientific classification of the various forms of endocarditis. Let us for the present have a convenient clinical one, and in such this or some similar name will have a place. A multiplication of names does no serious harm. What would be harmful would be that a name such as this should be taken as conveying facts which have yet to be proved or disproved. For instance, in this very case we have at least two such pitfalls. One is inclined to assume that this form of endocarditis is a pathological unity, which, I think, is far from being proved; and, what is of far more importance clinically, one thinks of the disease as necessarily fatal, a proposition which assumes that there are no cases of this disease in which early diagnosis and appropriate treatment would just turn the scale in favour of life. If we use the term *malignant* in the sense in which it is applied to small-pox or scarlatina—namely, as signifying very considerable gravity indeed—I do not think the name does any harm.

I submit to you to-night a heart (No. 2) which bears all the marks of having had an acute proliferative and necrotic endo-

carditis, which was in all probability the product of septic poisoning; yet the owner of this heart did not die of the endocarditis, but of a single subsequent embolism in a vital part. The brief history I am able to present to you is this:—

Case II.—A man between twenty and twenty-five years of age, about 5ft 7in. high, spare but muscular, lodged for a month in a street near the Whitworth Hospital. Of his previous history nothing could be ascertained. During that period he went daily to his work as a labourer, made no complaint of ill health, and was not noticed by his fellow-lodgers to be anything but a fine, healthy young man. One night after he had gone to bed he suddenly complained of being very ill, and he died in a quarter of an hour afterwards.

Post-mortem.—The right heart was found normal. In the left auricle were numerous minute vegetations on the endocardium. In the ventricle the chordæ tendineæ formed a cauliflower-like mass of cream-coloured oval lumps with calcareous cores, as is not uncommon in malignant endocarditis. The whole mass sprang from the papillary muscles, and was free from the mitral valves. The anterior valve was quite free at its margin. Death was caused by one of the oval lumps getting washed into the circulation and being deposited in the posterior (descending) branch of the right coronary artery.

The specimen is of great interest as a case of coronary embolism, but I venture to adduce it as an example of a previous malignant endocarditis which had got well. I regret for the sake of the pathological completeness of the specimen that I removed the embolic plug during the examination of the heart, and ground it up in search for bacilli, and I subsequently removed another of the oval masses from the chordæ in order to make sections. The *sacra fames* of the histologist devours the choicest bits of the best glass-jar preparations.

Malignant endocarditis may affect an endocardium previously, so far as we can judge, healthy, and an endocardium previously diseased, and for this latter it has a special affinity. That is to say, that a valve previously sclerosed by rheumatism is a favourite locus for subsequent septic infection. Almost as a corollary from

this, it may be assumed that some cases of cardiac disease, more especially of recurrent disease, are due to septic poisoning, but are not recognised as such because they are not fatal. We tend to forget that in septic poisoning the issue depends not only on the nature of the poison, but on the quantity absorbed.

I contend that we have to recognise that death is not an absolutely necessary consequence of infective endocarditis—possibly not even necessary in the majority of cases in which it really occurs. We want a name badly for the group of cases of endocarditis which are not of diathetic (for instance, rheumatic) causation or due to degeneration. "Septic" or "infective" would be fairly satisfactory both to the pathologist and the clinician if it could be shown that no infective organisms had to do with other forms of endocarditis. "Ulcerative" is objectionable for at least two reasons—one, that ulceration is not by any means constantly present in hearts which have been the subject of this disease; the other, that ulceration, or at least necrosis, may occur in other forms of cardiac disease. I have brought here a heart (No. 3) in which there are two perforations of the aortic valves, but they are the result of atheromatous necrosis, and not of septic infection. The patient, who was lately under my care, presented no febrile curve whatsoever, although from the rapid development of his symptoms I am forced to think that the valvular lesion was of recent production. Death seemed to be caused by simple failure of the left ventricle.

There is one point, among many, which occurred to me while watching the case of M. B., upon which I find it difficult to formulate an opinion. In such a case how much of the totality of her symptoms is due to a generalised septicæmia and how much to the local endocarditis? Have we, under the influence of the older school of pathologists, attached too much importance to the heart lesion? In a case of pyæmic endocarditis, secondary, let us say, to a pyelitis or renal abscess, we should have little difficulty in deciding that once the patient had a generalised pyæmia the establishment of a cardiac focus differed from one in the deltoid only in proportion to the importance and mechanical necessities of the two

muscles; but in a case such as M. B.'s, where the septic process may be said not to have localised itself elsewhere than in the heart valves, the circumstances are different. Of such a case it may be suggested that but for the favourable nidus presented to the poison in an endocardium previously, in all probability, diseased, her tissues generally might have resisted the poison successfully. In such a focus opportunity is given for the multiplication and dissemination of a poison out of proportion to the resistive powers of the blood and tissues, and, of course, these powers of resistance are themselves lessened by the mechanical impairment of the very mechanism by which the blood and tissues are refreshed and renewed in their struggle against the invading poison.

I have little to say on the pathological aspect of malignant endocarditis. The whole question of micro-organisms in this connection is too large a subject for me to enter upon, especially as I could say very little indeed which was not second-hand. On that part of the subject I beg to refer you to Professor Purser's paper in the Transactions of the Academy, Vol. IV., 1886. But with regard to the coarse changes in the heart, one may practically group them into vegetative or proliferative and necrotic or ulcerative. The processes leading up to these changes are not antagonistic but sequential—the one succeeds the other. One of the objects of this paper is to suggest that in favourable cases, practically seldom recognised, the necrotic process may not occur, or may occur only to a slight extent, and thus the patient may be saved all the phenomena of septic dissemination.

I venture then, in conclusion, to accentuate these facts:—First, that malignant endocarditis is a name for a variable group of clinical phenomena, and not, so far as we know at present, for a constant pathological condition; second, that "malignant" as here used does not signify that there is a necessarily fatal form of endocarditis which differs in kind from all forms which are not fatal; third, that many cases of endocarditis, those more especially which cannot be traced to the usual diathetic causes, such as rheumatism, are probably the result of septic infection.

DR. WALTER SMITH expressed his belief that so-called "malignant endocarditis" is but a localised expression of general pyæmia. There is no single specific cause for malignant endocarditis, hence malignant endocarditis should be removed from the category of cardiac diseases, and be described in connection with pyæmia. And its special diagnosis, in many cases impossible, is rather a feat of clinical interest than one of great practical significance.

DR. O'CARROLL, in reply, suggested two clinical types of malignant endocarditis—one, that in which the endocardial lesion is but one of many suppurative foci; the other, in which the lesion, so far as one can find, stands alone. In this latter case one may suggest a hypothesis that the pyæmic poison, if small in amount, might be coped with and beaten by the tissues, but that some of the organisms get a lease of a breeding-ground and source of subsequent dissemination in the place of least resistance—namely, a previously sclerosed valve.

CLINICAL CHART OF TEMPERATURE.

Case of **M. B.**