CLINICAL LECTURE ON ULCERATIVE ENDOCARDITIS, OR ARTERIAL PYÆMIA.

Delivered at Guy's Hospital.

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THE cases on which I wish to make a few remarks, are examples of a form of disease by no means uncommon, and now holding a well established position in the nosology; yet it is one which is still frequently overlooked, and is altogether unrecognised by some in the profession. I refer to the embolism of Kirkes, or that form of malady which I subsequently styled arterial pyæmia, and which again is now, according to the usage of foreign writers, termed ulcerative endocarditis. None of these appellations can be regarded as good, seeing that they give a too determinate meaning to the nature of a complaint which is by no means perfectly understood. It is a disease full of interest and importance, for not only is its pathology obscure, but the symptoms are often so ill-defined, that it is constantly mistaken for other complaints, and especially for the various forms of fever. During the last two months there have, I believe, been three or four cases of this disease sent to the hospital on the supposition that they were typhoid fever, and certainly, the distinguishing marks between the two maladies are most difficult to recognise. The patient is generally in a highly pyretic state, or in that condition which, in ordinary language, is styled typhoid, without presenting any characteristic symptoms, the only sign present to raise a suspicion of the true nature of the case being a cardiac *bruit*. It is this alone, in many instances, which excites a suspicion as to the nature of the case, especially when the indications of every other form of disease are wanting. The case is really one of blood-poisoning, or a form of septicæmia; the source of the infection being, as it is supposed, in the heart, or in any place near the centre of the circulation, where ulceration and vegetations exist; but whether the septic material be decomposing fibrin, or the *débris* arising from the breach on the endocardium, or whether there be any distinct infectious substance circulating through the system, is still a question.

For some years past, it has now been known that persons have died with symptoms of typhoid fever or pyæmia, and the necropsy has revealed the presence of fibrinous masses, or infarcts in the spleen and kidneys; also that, associated with these deposits, there have been vegetations on the heart; these being usually of recent formation, and situated on chronically diseased valves. But long anterior to the recognition of symptoms attendant upon these pathological conditions, the fact of the association of embolic masses or infarctions with vegetations on the valves was well known. Bright portrayed these fibrinous deposits in the kidney more than fifty years ago; and soon afterwards Rokitansky described what he called "capillary phlebitis," of organs in association with heart-disease, especially directing attention to the deposits in the spleen and kidneys, as arising from endocarditis. Kirkes, however, may be regarded as the first in this country who gave a clear account of the effects of fibrinous matter circulating in the system when washed from the valves of the heart; and held that it was productive of symptoms of blood-poisoning. He first of all gave a very lucid account of those cases where portions of fibrine of some dimensions are carried into the larger vessels, and productive of local effects, as for example, gangrene of the leg if the femoral artery be plugged, or softening of the brain if a cerebral artery be blocked; and then proceeded, but more briefly, to describe the present class, where small particles of fibrinous matter are carried into the arterioles and capillaries of organs, and so produce constitutional disturbance and fever. From this time, I have clearly recognised the existence of this disease, although I have not always diagnosed it; my earlier cases (some published) were chronic ones, of some weeks' duration, in which the paroxysms of fever were so marked and regular that these were regarded as the phenomena of ague. Of late, the examples of this disease have been more acute, resembling typhoid, rather than intermittent fever; the temperature remaining very high throughout the course of the complaint. The mistake in confounding them with fever has, no doubt, been mainly due to the late great prevalence of typhoid, which has led us to see this disease in all forms of febrile dis-

order. You may remember the rule which we lay down as to the method of diagnosis in febrile disorders. We first look for a local inflammation as the cause of the pyrexia; failing this discovery, we consider the possibility of a general or blood-disease, and look out for an exanthem. If this do not appear, we are apt to regard the case as one of typhoid; and unfortunately, we are justified in styling a case typhoid when no characteristic symptoms are present. At the same time, however, we ought to remember the possibility of blood-poisoning from a local source of self-infection, and not forget the condition known as pyæmia on the venous side, or that of ulcerative endocarditis on the arterial side of the system. Tubercle, too, should not be forgotten. In these last-named blood-diseases there are fever, rigors, and pains in the limbs, so that they closely resemble one another; although the veins in one case have taken up the septic material from the surface, and the arteries, in the other case, from the centre of the circulation. The symptoms are either very acute, like those of typhoid, or more chronic and paroxysmal, like those of ague. In the arterial case, softening embolic masses may be found in the organs, more especially in the spleen and kidneys, and vegetations discovered on the valves of the heart. Sometimes the symptoms of blood-poisoning are most profound, and warrant the expression of Virchow, "malignant endocarditis".

The term ulcerative endocarditis implies that an acute change has taken place in the endothelium, whereby the products of inflammation, including the disintegrating membrane with portions of fibrine, are carried into the circulation, and infect the whole system. It might be thought that this affection would be one of the sequences of rheumatic fever when endocarditis has occurred, but such does not seem to be the case, for it does not appear to be a common experience that septicæmia is one of the sequelæ of rheumatism. In the large majority of cases of the disease of which I am now speaking, the affection of the valves has been old, evidenced by thickening of the tissues, narrowing, or adhesions. It is upon these chronically affected valves that the recent action has taken place, and vegetations have formed. Although this has been my usual experience, I have met with instances where no chronic disease of the valves has existed, but, at the same time, there has been no evidence of a recent endocarditis of a rheumatic character. In these cases, I have questioned if the vegetations found on the valves have originated there, and been the source of the infection, thinking, probably, that the deposits on the heart were due to a previous change in the blood, and that both they and the infarcts were due to a profound alteration in the blood itself. Even should it be shown that the blood is primarily affected from the centre of the circulation, it has yet to be proved that there is, of necessity, an inflammation and ulceration of the endothelium, since, in one of the most marked cases of arterial pyæmia which I have ever witnessed, the source of infection was a small aneurysm near the mitral valve in which fibrin was disintegrating. Several years ago, in discussing this subject, I mentioned cases of scarlatina and death from burn, where fibrinous masses were found in the spleen, kidneys, and other parts, but where there was no appearance of endocarditis. It must be remembered that, if endocarditis give rise to blood-poisoning, so, on the other hand, an endocarditis may result from blood-poisoning, as witnessed in pyæmia, scarlatina, etc.

The difficulty indeed, is so considerable, in regarding an endocarditis as a source of infection, that some observers maintain the necessity of the presence of bacteria or micrococci in the vegetations, and have accordingly styled this affection "infectious endocarditis". Indeed, some German physicians have given a description of small granular bodies found in the valvular formations; and during the late congress Dr. Osler, professor at Montreal, communicated an essay in maintenance of this view. He objected to the term ulcerative endocarditis, because he had seen the disease without ulceration, and preferred the term infectious. He did not object to the term diphtheritic, because he thought it probable that analogous changes took place on the valves as are seen in other parts of the body in diphtheria; and if there were adventitious bodies present, the expression "mycosis endocardii" might be allowable. Dr. Osler exhibited drawings from a case where, in the material scraped from the tendinous cords, there were seen a number of spherical bodies which he called "micrococcus balls". If the observation of this professor, and those of others, be correct, a further question arises as to the way in which these parasitic bodies find an entrance into the body.

The disease is one of great interest clinically, for not only may the symptoms be of that general kind which may lead us to confound it with fever, but occasionally, owing to one organ being especially selected for the manifestation of the infection, the inflammation attending it may be regarded as primary, and the real nature of the complaint be overlooked. It is probable that this, in former years, was constantly occurring, as two of my late cases suggest. In the one case, owing probably to the plugging of some of the cerebral vessels, a meningitis was set up; and this, if an incomplete examination had been made, might have been

regarded as the essential disease. In the other case, the patient had empyema, and which, in spite of being treated according to the best methods, appeared to be the cause of her death. The necropsy showed, however, an ulcerative endocarditis of which, probably, the pleurisy was an effect.

I should also mention that, when the symptoms are those only of blood-poisoning, they are not always of the acute kind which we have lately seen, but may continue over a course of several weeks, and then put on the appearance of an intermittent fever. The symptoms are also probably regarded sometimes as rheumatic, especially if the patient have already had rheumatic fever; for example, a patient suffering from known heart-disease is attacked by febrile symptoms, pains in his joints, etc., and from this he recovers. The case is regarded as one of subacute rheumatism, but it is truly one of blood-poisoning, and the proof of this lies in the condition of the organs found after death. In the spleen and kidneys there may be seen infarcts of different ages—some recent, others drying up, and on the surface of the organs indentations and cicatrices indicative of the presence of much older deposits.

The following is the case of a young man supposed to have fever, and where ulcerative endocarditis and meningitis were found.

Thomas T., aged 26. (Reported by Mr. F. Dixon.) Porter in wine and spirit vaults, and a free liver. Eight years ago he had acute rheumatism. On November 8th, he had pain in the back and vomiting; and, becoming very ill, he gave up work on the 10th. The fever continued, together with the sickness, until his admission on the 13th. He was a strong-looking well nourished man, but excessively ill, so that he could not give a very clear account of himself. His tongue was furred, temperature 103.6°, pulse 132, and he said he had had diarrhoea; no maculae. The liver could be felt below the ribs, and the spleen was thought to be somewhat enlarged. A well marked mitral *bruit* existed. In absence of all other cause for the fever, the case was regarded by the house-physician as one of typhoid. The patient became very delirious towards night, and the temperature rose to 104°. On the 14th he was still very ill, and the question was discussed as to the nature of the case. He was very restless and delirious, temperature varying from 103.8° to 104°; some cough and expectoration. The presence of the mitral *bruit* raised the question about endocarditis; but he had no rigors, nor any symptoms positively pointing to that form of disease. At the same time, the characteristic signs of typhoid were wanting. On the 13th he was worse: pulse tremulous and irregular; stools showed nothing peculiar; temperature 104°, but had been a few hours before as low as 101°. He had been very sick. On the 14th, he was evidently sinking, talking incessantly, picking the bed clothes, and before he died in the evening his temperature had risen to 105.2°. The *post mortem* examination showed chronic thickening of the curtains of the mitral valve, and on these were patches of vegetation. The spleen was large, and contained infarcts or fibrinous deposits. The liver was large, the kidneys were unaffected, and the intestines healthy. The brain was inflamed, with lymph along the sides and in the fissure of Sylvius. He was treated with brandy and opium.

The following case was also regarded as one of fever.

John P., aged 23. (Reported by Mr. E. Wakeland.) Draper's assistant. He had rheumatic fever when a boy. Five days before admission he was seized with a severe headache, accompanied by vomiting; he afterwards had pains in his joints, and then took to his bed. He had no diarrhoea. The sickness ceased, but continuing otherwise very ill he was sent to the hospital as a case of typhoid fever on September 26th. He was a well nourished man, and his illness was quite recent. He looked anxious, skin hot, tongue furred, eyes rather hollow, spleen full three inches below ribs, liver not enlarged, abdomen full, no rash, bowels opened once a day. A loud systolic *bruit* was heard over the mitral area. He was very restless; had pain in the head, limbs, and joints. Pulse 140, dicrotic. Temperature 103.7°. September 27th: All symptoms were somewhat aggravated; no maculae or positive signs of typhoid. September 28th: Delirious and constantly calling out; other symptoms unchanged. Temperature 104°. Cold was applied to the axilla by means of Mr. Strachan's apparatus, and the temperature fell. September 29th: In the morning he seemed quieter, more rational; temperature 102°, and took nourishment. In the evening the temperature rose to 104.8°, and the ice-bags were again applied during the night. September 30th: He had slept well. Temperature 99. Tongue dry and brown. In the middle of the day, the temperature having again risen three degrees, cold was again applied to the axilla. The temperature rapidly fell and the cold was removed; he gradually cooled down after this until the feet became quite cold, and the patient seemed collapsed. He gradually sank and died in the evening, the temperature having again risen to 103.2°. The *post mortem* examination showed the mitral valve much thickened and covered with vegetations; there were also some on the aortic. The kidneys contained several fibrinous masses or infarcts; the spleen was

large, soft, and contained similar deposits. The brain healthy, excepting a clot (apparently *ante mortem*) in the middle of the cerebral artery.

The next case was one of empyema, and this no doubt was a result of infection.

Mary F., aged 26. (Reported by Mr. W. Harris.) Married, with family; seven months pregnant at time of admission on September 2nd. Four days before this she was seized with pain in the chest, and other symptoms of an inflammatory attack. Soon after admission she was delivered of a child. The left side of the chest was dull on percussion below, with loss of vocal fremitus, bronchial breathing above, and other signs indicative of pleurisy with effusion. There was also some bronchitis of the right lung. She was very ill, with much distress of breathing for some days, when she appeared better; but the dulness and other physical signs of pleuritic effusion still remained. She had wine and quinine given, with liniments of iodine and mercury. Subsequently the fluid increased in the chest, and she was tapped. Nine pints of pus were removed, and a drainage-tube inserted. The patient was now for a time better; but after a few days it was evident that she was weaker, and her powers were gradually failing. The chest discharged well, and the dressings were well attended to; and therefore it was by no means obvious, whilst she was improving as regards her chest-complaint, why she should be daily losing ground. At last she became cold and collapsed, and slowly died a month after admission. The *post mortem* examination showed the lungs healthy, but there was acute ulcerative endocarditis of the aortic valves. The kidneys contained small infarcts; and the spleen contained several, which had softened into fluid.

Dr. Fagge has recently had under his care the case of a woman where the cerebral vessels were probably plugged, inducing a partial paralysis, and for which she was admitted. But the patient had, besides, febrile symptoms, making her temperature chart resemble one of ague; and after death she was found to have been the subject of ulcerative endocarditis.

Rebecca T., aged 30. (Reported by Mr. Gleher.) Admitted under Dr. Fagge, September 10th. Housemaid at hotel for fourteen years, mostly occupied in carrying heavy articles up and down stairs. She did not know that she had rheumatism, but was subject to pains in the joints, and for four years had shortness of breath. A week ago, she was suddenly seized with giddiness, and at same time with weakness of the left side of the body. On admission, she had partial paralysis of motion and sensation of the left arm and leg. An examination showed a mitral *bruit*, and also a diastolic *bruit* over the sternum. Liver-dulness was increased. The spleen was large, and was felt considerably below the ribs. She continued extremely ill during the six weeks until her death, with febrile symptoms, continual change of temperature, and towards the last excessive prostration. There was a continuous rise and fall from 97 to 104°. For a few days before death there was weakness, profuse perspiration, and great prostration. *Post mortem* examination showed discoloration of convolutions, but no marked embolus. The spleen had several abscesses, or deposits of a creamy matter. The kidneys contained several infarcts, which were hard. The mitral valve was like a button-hole, and had vegetations on it. The aortic valves were thickened.

CONJUNCTIVAL TRANSPLANTATION.—M. Marc Dufour, in the *Revue Médicale de la Suisse Romande*, reports four cases in which he transplanted the conjunctival mucous membrane of the rabbit to the human eye. He operated in two cases of ocular symblepharon. The adhesion having been dissected away, a fragment of the mucous membrane of the rabbit's eye was applied to the denuded surface, and fixed in place by sutures. In an early case, that of an old woman, the operation failed; the fragment became necrosed, the woman being old. The fragments had been fixed with silk sutures. A little later, the operation was performed on a child, aged 11, and the transplanted fragment was fixed on the eye and the eyelid at the same time. The success was complete, and, two months afterwards, the fragment was quite living. In the case of a man, after the removal of an epithelioma of the eyelid, M. Dufour transplanted mucous membrane taken from a small tumour on the lower lip of the patient himself. This operation was completely successful; nevertheless, it had to be followed a year afterwards by a fresh operation, the disease having recurred. This time, also, a fragment of the mucous membrane of the lip was taken. M. Dufour recommends that the transplantation should not be made too soon after the incision; that the fragment to be transplanted should be previously fixed with sutures to prevent the rolling up of the edges. He recommends that the revived place and the fragment should be washed with salicylic acid.