

MUSSEY. (J. H.)

Two cases of malignant endocarditis.

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TWO CASES
OF
MALIGNANT ENDOCARDITIS.

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BOTH the cases included in this report have been named malignant endocarditis, although one of them is without the warrant of a microscopical examination. The clinical course and macroscopical appearances indicate such to have been its true nature, however. I venture to bring it before you thus labelled, to excite criticism, if necessary, and to ask your attention to one of the manifold phases of this interesting disease.

The case was under the observation of Dr. J. Henry Musser, of Lancaster County, and seen by my father, Dr. Benjamin Musser, in consultation. It occurred in their practice, in 1878. I recall the case both from conversations and letters concerning it at the time. Unfortunately, the notes of the case in Dr. Musser's possession have been mislaid. His memory and memo-



randa in his case-book feebly supply the deficiency; fortunately, we have the morbid specimens.

CASE I. *Rheumatism; embolism of large vessels; cardiac murmurs; gangrene; chills and fever; death. Autopsy: vegetations on mitral leaflets; emboli in brachial and femoral arteries.*—H. R., male, æt. twenty-three, laborer, consulted Dr. J. Henry Musser, June 25th, on account of rheumatism. He was visited by his physician the first week in July once, the second week four times, and the third twice. On the first of August grave symptoms set in, and on the third of September he died. It will be observed, therefore, that, in July, H. R. was not very ill; in fact, he continued at light work on his farm, and on the first of August was in the harvest field when the first embolus manifested itself. During that month he had rheumatism, and, for a short time before August 1st, chills and fever.

August 1. Sudden severe pain occurred in the right brachial artery. Could not be removed to his house at once on account of collapse. Dr. Musser saw him, and found the pulse absent at the wrist, the hand cold and cyanosed. Two days subsequently the femoral artery became plugged, much pain being occasioned at the time. The circulation in neither arm nor leg was ever restored, and gangrene ensued. Attention was at once called to the heart, and a distinct systolic murmur was heard at base and apex. During the month an irregular fever, with irregularly recurring chills, was present. Death took place from exhaustion, September 3d, thirty-three days after serious symptoms set in.

At the autopsy the vessels were plugged with emboli and thrombi, as exhibited in the specimens. The heart was enlarged by dilatation, and on the posterior cusp of the mitral valve soft fungating vegetations grew. Some of the chordæ tendineæ were surrounded by vegetations. The auricular surface of one cusp was rough, and minute ulcers were present. Fibrin clung to the vegetations, and recent clots filled the cavities. The aortic valve and the right heart were normal. There was no atheroma.

REMARKS.—Without doubt, in this instance, malignant endocarditis was associated with rheumatism, and

the type was essentially pyæmic. One might presume, however, that the so-called primary rheumatic symptoms were pyæmic in nature, or that the cardiac lesions supervened secondarily to the gangrene. The teachings of the autopsy do not sustain either supposition. The absence of old cardiac and vascular lesions, and the general integrity of the organs, preclude the possibility of pyæmia. The normal condition of the arteries (absence of atheroma), the absence of primary blood dyscrasiæ, or of any cause, save cardiac, for the emboli, defeat the second supposition.

CASE II. *Malignant endocarditis; chills, irregular fever, sweats; cardiac murmur and pain; vomiting and diarrhœa; marked embolic phenomena; jaundice; death. Autopsy: proliferative bacterial endocarditis confined to the mitral valve; emboli of skin, mucous membrane of mouth, of stomach, and of intestines, of spleen, kidneys, peritoneum and endocardium; embolus and thrombus in left iliac vein.*—M. S., female, æt. twenty-seven, white, German, married, admitted to Philadelphia Hospital August 26, 1886. At the same time her husband and children were admitted for a febrile complaint, thought to be of malarial origin. M. S. had been ill about six weeks, of an irregular fever, also said to be malarial. At this time she was nursing an infant which had been born one year before. The labor was natural, and she had a good getting up. Since birth of child M. S. has not menstruated. Three years previous to this illness she had an attack of malarial fever, and at one time had rheumatism of the leg. She never had any other ailment, was strong and hearty, and of temperate habits.

The family history of M. S. is very good. Her parents and several brothers and sisters are living. One sister died of pneumonia; the cause of death of another is unknown. On admission it was noted that the patient was prostrated and complained of chilly sensations, she sweated profusely, and suffered from nausea. The tongue was clean and moist; the bowels loose, five movements in twenty-four hours, thin and greenish in color; the nausea had disap-

peared in twelve hours; tenderness of the epigastrium was observed, but none in the iliac fossæ. The spleen extended, in the axillary line, from the sixth rib to the margin of the ribs. There was no eruption. Both cheeks were flushed. The skin and mucous membranes were pale, the hands quite anæmic. She was somewhat emaciated. The mind was clear. The lungs and heart were normal; the latter rapid in action, and rather feeble. Milk diet, stimulants, and quinia ordered.

Sept. 2. My resident, Dr. Dorland, detected a cardiac murmur two days ago, and to-day the following is noted: General symptoms and appearances about the same. Apex beat of heart in fifth interspace in nipple line; impulse moderately feeble; no thrill; pulmonary second sound accentuated; high-pitched systolic murmur at apex transmitted to axilla; no murmur at aortic orifice. Pulse rapid, small, and feeble. No cough; no dyspnoea. Ulcerative endocarditis suspected.

8th. The fever has continued in spite of quinine. The patient sweats profusely throughout the day, but not at night. Her anæmic appearance is striking. She is quite cheerful, but is evidently losing flesh and strength. The diarrhoea continues. The cardiac murmur has increased in intensity during the past forty-eight hours, and can be heard loudly at the angle of both scapulæ, louder at the right. Marked tenderness on pressure, and some pain complained of in the third left intercostal space. At the base of the right lung impaired resonance, and fine, moist, crepitant râles are observed. A few râles are heard at the right apex also. Short, high-pitched, jerking inspiration and prolonged expiration are detected at the right apex, and high-pitched inspiration at the left.

12th. Two days ago two small purpuric spots were observed on each upper eyelid, and to-day hemorrhagic infarcts are seen all over the trunk and on the upper extremities, pin-head to split-pea in size, bright red, not disappearing on pressure. Hemorrhages in the ocular and palpebral conjunctivæ of both eyes at inner and outer canthus. Rapidly losing flesh and strength. Sweats continue. Abdomen distended in epigastric and umbilical regions; large wavy impulse in epigastrium. Nausea and vomiting recurred the past five days; fluid dark, greenish tinge. The vomiting

occurs in the morning. Diarrhœa continues. Spleen readily felt below the margin of the ribs, tender. Lungs have cleared up. No cerebral symptoms. Heart's action irregular. Pulse small, feeble, rapid, dicrotic. Tenderness behind the sternum. Impulse (systolic) in second, third, and fourth left interspaces. In second interspace to the left of sternum very perceptible thrill. Systolic murmur not so loud as it has been at apex. Over third rib, left auricle, loud, booming systole, with "grinding" murmur. In second interspace to the left of the sternum loud, rough, high-pitched murmur. Pulmonary second sound muffled. Murmur over tricuspid not so distinct as at mitral, and probably transmitted; aortic murmur low in pitch, conducted from base. No œdema. Mind clear.

13th. Physical signs as above. Cardiac dulness extends from the left edge of the sternum one inch to the left in the second interspace. It extends in the third interspace two inches, and in the fourth three inches. Apex beat in nipple line. Intensity of murmur greatest in second interspace. Chill this morning. Urine scanty and high-colored, and contains blood and epithelial tube casts in abundance, and a small amount of albumin. General symptoms the same. Extreme prostration.

14th. Chill at 7 A.M. A little more drowsy than formerly; no delirium. Murmur in second interspace not so distinct. New capillary infarcts in abundance about the neck. Vomiting and diarrhœa continue. Infarcts in lips and on end of tongue. Conjunctivæ slightly icteric. Dyspnœa severe for half an hour at 2 P.M. Partially collapsed state, with gasping and sighing; excessively rapid pulse; stimulants relieved the patient. At 11 P.M. a second attack. During the day very irritable, and latter part of day drowsy. Died at 2 A.M., September 15th.

The following record indicates the daily fluctuations in temperature. The pulse record was not preserved, but from first to last it was rapid, 120 to 130 per minute.

Aug. 25, P.M., 99.3°. *26th,* A.M., 100.4°; P.M., 103.2°. *27th,* A.M., 101.1°; P.M., 103°. *28th,* A.M., 101.1°; P.M., 101.3°. *29th,* A.M., 102.1°; P.M., 102.3°. *30th,* A.M., 103°; P.M., 100.4°. *31st,* A.M., 100.1°; P.M., 101°.

Sept. 1, A.M., 102°; P.M., 101.2°. *2d,* A.M., 102.1°; P.M.,

102.3°. 3d, A.M., 102.4°; P.M., 99°. 4th, A.M., 99°; P.M., 101.4°. 5th, A.M., 101.2°; P.M., 105°. 6th, A.M., 99.2°; P.M., 105°. 7th, A.M., 101.1°; P.M., 97°. 8th, A.M., 97.2°; P.M., 99.3°. 9th, A.M., 101.2°; P.M., 105°. 10th, A.M., 105.4°; P.M., 99.2° (chill at 6.30 A.M., and 9 A.M. temperature taken). 11th, A.M., 101.3°; P.M., 99.3°. 12th, A.M., 101.4°; P.M., 99.3° (chill, 7.30). 13th, A.M., 102.3°; P.M., 102°. 14th, A.M., 100.2°; P.M., 100.4°. 15th, A.M., 99.2°.

During the course of her illness careful search was made for localized purulent inflammation. The pelvic organs were normal, the bones were evidently free from disease, and inflammation of the middle ear was not detected.

Autopsy was made by Prof. Osler, Dr. Mussey, and Dr. Dorlan. *General appearance*: Medium sized body, somewhat emaciated. Skin pale, slightly icteric. Numerous petechiæ cover the face, chest, and arms; very numerous on right arm and wrist. Petechiæ range in size from a pin's head to a split-pea, and vary slightly in color from vivid red to dark blue; one or two only present central yellowish core.

Abdomen, thorax: Numerous petechiæ on parietal peritoneum. Slight serous effusion in both pleural sacs; few adhesions at right base; some petechiæ in parietal pleura.

Pericardium, heart: Excess of pericardial fluid, numerous small petechiæ on visceral layer. Right chamber relaxed, containing fluid blood and small clot. Left ventricle, tolerably firm dark clot. Apex of the left auricle is long, and can be seen turning round to base of pulmonary artery. Before removal of heart, fingers in pulmonary vein and pulmonary artery, both of which are free. Further dissection of heart: right auricle, little blood, staining of endocardium; a few petechiæ. Right ventricle, chamber large; tricuspid segments a little thickened; a distinct nodular thickening at angle of anterior and internal cusps. Pulmonary semilunar valve normal. Left auricle dilated. Seen from above, mitral orifice plugged by loose black clot and large grayish-white vegetations. After removal of auricle, the following conditions of valve appear: from base of posterior cusp of mitral, large irregular gray-yellow vegetations one inch in length extend into the auricle, passing out at right angles to the mitral ring,

and forming a sort of valve across the orifice. Entire auricular surface of this cusp covered with vegetations. Anterior cusp at free edge a large, warty vegetation projecting into ventricle three-fourths of an inch. The anterior part of this cusp is free. One or two of the chordæ tendineæ are encircled with vegetations. No involvement of endocardium, other than that of valves. Aortic cusps normal. So-called unprotected space below aortic ring presents deep depression, is closed, and to its base is attached a nodular thickening at angle of tricuspid cusps. Muscle substance of heart is pale, looks fatty; numerous ecchymoses. Coronary arteries free.

Lungs crepitant; ecchymoses on pleura. No infarcts. Bases œdematous, somewhat congested. Pulmonary artery normal.

Spleen weighs 1 lb. 5 oz., soft, capsule closely adherent at one spot. Pulp soft, reddish-brown; four large infarcts of some age, yellowish; others recent, none suppurating.

Kidneys somewhat enlarged. Capsule free, surface irregular. Numerous recent infarcts. Unusual amount of pigment. Right kidney, same changes; not so many large infarcts; the pigmentary changes about many of these infarcts are peculiar.

Pancreas: Few interlobular hemorrhages.

Stomach: Numerous ecchymoses. Many with small, central grayish spots. Mucosa about cardiac end much reddened.

Duodenum: Bile duct pervious; in mucous membrane small, superficial ecchymoses.

Small intestine: One or two small infarcts in jejunum. Toward valve the ileum was much injected, and the small infarcts were numerous.

Large intestine: Cæcum deeply injected; throughout the colon they are numerous, in size from two to ten millimetres. Some of larger show distinct nodular prominence, which on dissection is grayish-red, firm.

Liver: A little enlarged. No extravasations; tissue soft and light brown. No infarcts. Weight 5 lbs. 4 oz.

Uterus: Medium size. Os shows deep lateral laceration. Mucous membrane deeply pigmented. Muscle substance normal. Ovaries normal. Small cyst in broad ligament. Pelvic veins are normal. No trace of chronic inflammation about broad ligament. No thrombosis of veins.

Bladder : Few ecchymoses.

In the common iliac artery a plug is seen extending into the external iliac as far as the femoral; in the internal iliac it extends for two inches. On slitting up artery the thrombus is closely adherent, and at bifurcation it has softened, and has a purulent (?) appearance. Clot in internal iliac is one and a half inches long at least. Much pigmentation about the internal coats of the artery. No evidence of any local disease. Brain, spinal cord, eyes, and ears not examined.

Microscopical examination of the fresh clot in the artery, and the vegetation on the valve, by Dr. Griffiths, revealed the micrococci common to ulcerative endocarditis, the *Staphylococcus pyogenes aureus*.

Sections of the organs after hardening were made for me by Dr. William Gray. He reports as follows :

Heart : Fatty degeneration of the muscular fibre, with almost entire obliteration of the striæ ; increase of the interfibrillar connective tissue, and decided increase of the nuclear elements.

Spleen : Extensive cirrhosis ; obliteration of the cellular elements by fatty degeneration and an abundance of blood crystals (section from an infarct) are seen.

Kidney : Increase of intertubular connective tissue and tissue of Bowman's capsule. Proliferation of epithelial cells lining capsules and tubes. Cells degenerated and lumen of tubes plugged with casts. Section of infarct shows blood infiltrating into the tubules and between connective tissue fibres.

Intestine : Increase of connective tissue in submucous layer and of adenoid tissue of villi ; extensive lymphoid infiltration into mucous layer, villi, and glands of Lieberkühn. Granular degeneration of cells of villi. Infarct between and into submucous and muscular coat, blood crystals in infarct.

Artery : Entire absence of intima, with thickening of middle coat, and infiltration of blood into media.

Unfortunately, Dr. Gray did not get any of the proliferated mass on the valves. Dr. Gray was unable to find micrococci in the sections he had cut. They were given him without proper labelling, so that he did not know the tissue he was cutting. While this is to be regretted, it is enough to learn from the early and late examinations

of Dr. Griffiths, that bacteria were present. Dr. Griffiths readily found in the fresh treated preparations that micrococci abounded in the granular matter, and about the leucocytes of the vegetation.

The lesions otherwise found, were myocarditis, glomerulo-nephritis, and gastro-enteritis, with the usual histological changes about an infarct.

REMARKS.—So many thoughts arise in the contemplation of a case that represents so complete an evolution of a morbid process that one is tempted to indulge in lengthy remarks. We shall limit ourselves, however, and so be content with calling your attention to one or two prominent features.

Diagnosis. Observe, if you please, in the first place, the perfect picture of malignant endocarditis presented by the case. There was not a moment after the manifestations of the heart lesions were studied, that the diagnosis of endocarditis was doubted. It is true that before the cardiac murmur had been detected we could only say we had to do with a septic process. Who would say otherwise in a case of a young person with irregular fever, profuse sweats, and extreme exhaustion; with vomiting and diarrhœa, and enlargement of the spleen; with an acute inflammation of the kidneys without dropsy; with the physical signs of endocardial inflammation; and with the most pronounced appearance of capillary hemorrhages in the skin and mucous membranes, terminating in suffusion of the skin, conjunctivæ, and mucous membranes with the mild but ominous hue of icterus? It is true we considered at first whether, from the history and association with similar affections, the case was one of malarial origin. The use of quinia without result, the irregularity of the fever, the frequent pulse, the extreme sweats, and the diarrhœa,

led us to abandon this idea. Typhoid fever was rightly thought of, but momentarily. The spots, the tympany, and the characteristic stools were wanting; no approach to the typhoid state was observed in spite of the high fever and rapid pulse; the peculiar features of the tongue were absent, and at no time were the faculties dull or the mind wandering. The physical signs observed in the lungs, the profuse sweats, the quick pulse, and the diarrhoea, naturally induced the question of tuberculosis to arise. The disappearance of the lung affection, the absence of hurried respiration and of the tubercle bacilli in the discharges, were sufficient counter-proofs.

Thus, by exclusion and by a careful study of the disease as it was gradually unfolded to us, we were enabled to make a diagnosis. In this, as in many instances, unfortunately, with waiting and watching, too soon the true nature of the affection was so legibly written that he who ran could read.

Class. So far as can be ascertained, the affection was idiopathic—using the term with the modern reservation. That is, a cause cannot be ascertained for the development of the disease in this case unless the slight rheumatic attack years previously is sufficient to attach the diathesis as cause and effect.

Type. Recall for a moment the marked symptoms present. The fever, the chills and the sweats, the clear intellect, the gastric and intestinal disturbances, clearly group the case with other examples of malignant endocarditis of the pyæmic type.

Special symptoms. Enough has been said of the symptoms in the history of the case. Your attention need not be called again to the vivid temperature range, the recurring chills, the grave renal symptoms, and the

pronounced capillary hemorrhages. The latter were striking—at first but a few, each day more and more appearing; confined not only to the skin, but seen in the lips and the tongue and the conjunctivæ, they presented a glaring picture of the pathological process. The anæmia, which was barely noted, deserves a remark. It was profound, and the appearance due to it at once excited attention. Certainly the hæmoglobin was much reduced, yet the patient was so ill we did not care to disturb her to ascertain its percentage.

One class of phenomena deserves more lengthy remarks—the physical signs of the cardiac lesion. At first, at the mitral orifice a systolic murmur transmitted in the usual direction was heard. It varied in loudness and pitch. Later it diminished in pitch and volume, and at the same time a very loud systolic murmur, high in pitch and grinding in character, was heard on the third left rib and in the second left interspace one and a half inches outside the sternal edge. So loud was the murmur in this situation that, especially as it was attended by a thrill, we thought the pulmonary valves were implicated in the disease. The occurrence, however, of an increased area of pulsation, most distinct in the second and third interspaces, and of an increased area of dulness in the auricular region, led to the conclusion that mitral stenosis was present. A presystolic murmur was not heard, however, and probably was not present. It was not created because of the projection of the vegetation over the orifice, in all probability. For a similar anatomical reason one can infer that the ventricle fills, partially at least, prior to and independent of contraction of the auricle. For with such an effort, at

once, communication would be cut off by the vegetation.

The systolic murmur was heard, in the latter weeks, loudest in the second and third interspaces. Two causes can be invoked for this peculiar localization of the murmur. The projection of the mass from the auricular surface of the valve could readily throw the back flow of blood—the valve not closing from the situation of the second mass—into vibration, and thus murmur and thrill be produced and heard loudest over the auricle. While such an explanation suffices, a second cause for the murmur can be found in the position and dilatation of the auricular appendage. If such a view of the case is held, then the theory of Naunyn and Balfour to explain the mechanism of systolic murmurs in this location is well illustrated. The excessive loudness of the systolic murmur at the angle of the right scapula was very peculiar.

The ease of recognition during life, and the peculiar physical signs, are the features of prominent note in this case.

