A Case of Malignant Endocarditis

1921

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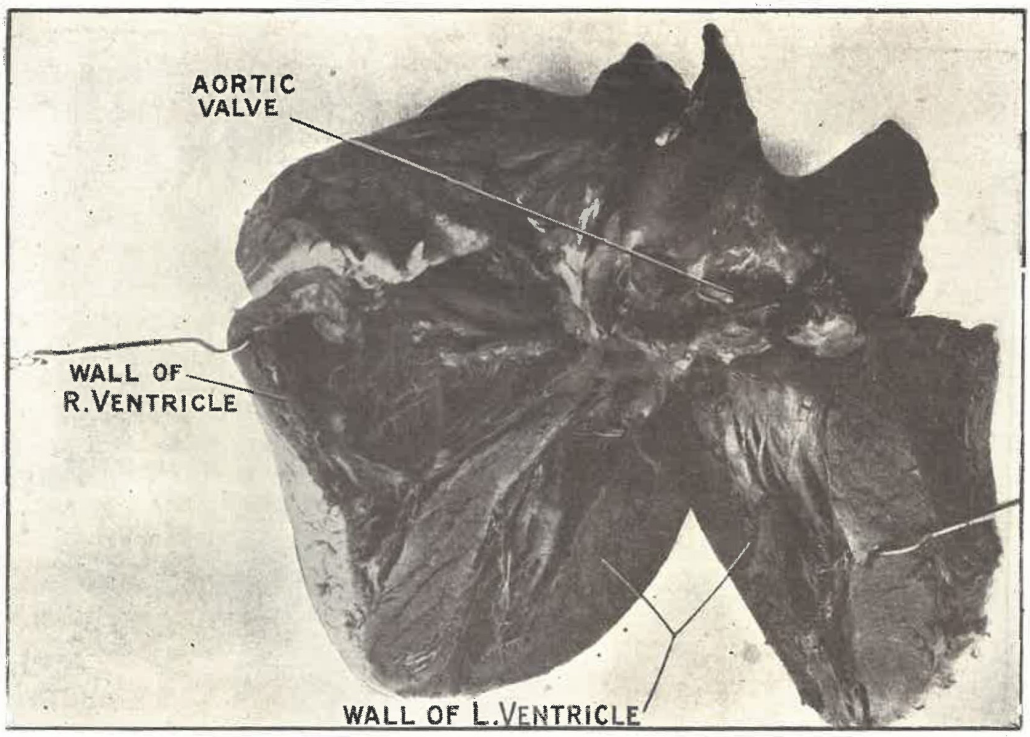
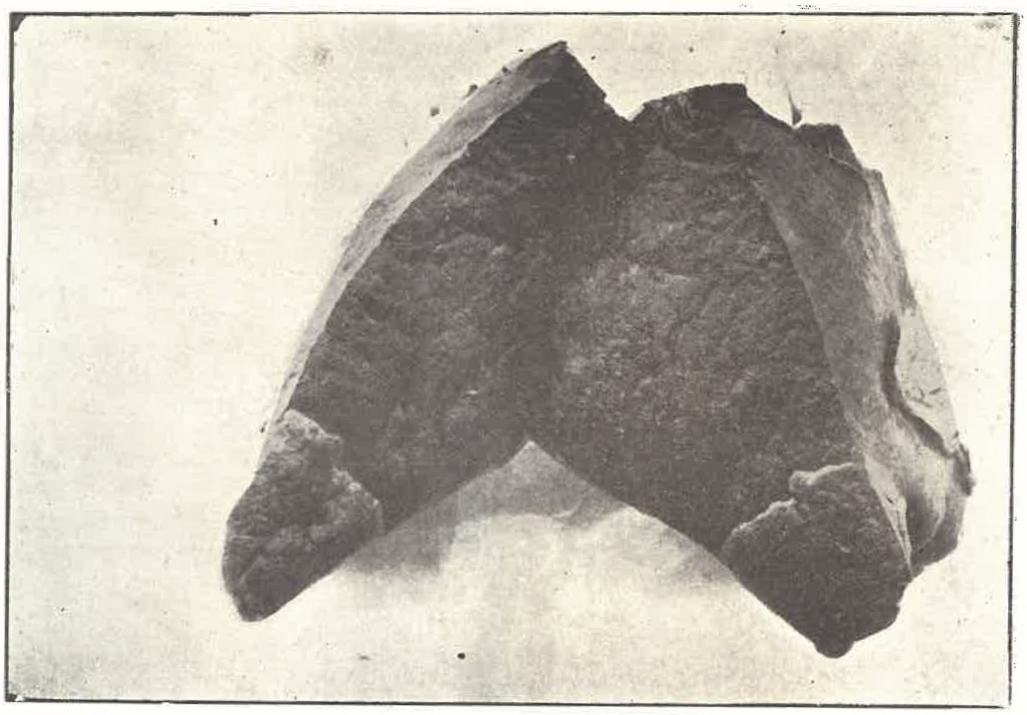
On account of the somewhat unusual features which it presents, this case is considered worthy of being recorded. The history of the case is as follows:—

Sergeant A.B., aged 52 years, three years' services overseas, admitted 1st January, 1921. Had no illness pre-war. Enteritis (mild) in Egypt, April, 1916, but went to France with his battalion in that month and carried on with full duty during the battle of the Somme (September, 1916), and was quite well up to December, 1916. About this time, the weather being very cold and damp, he began having attacks of sweating at night. These attacks were always of the same nature, a chill followed by a heavy sweat, and always occurred in the early morning. He would feel better after the sweating, would fall asleep again, and wake feeling well enough to go on duty, so that he did not report sick. At this time he was having "chills" every ten to fourteen days for the space of two months, when the weather became warmer and the attacks ceased. He remained on full duty and free from "chills" until the following winter. During the Battle of Passchendaele (October, 1917) the condition returned with much greater severity, accompanied this time by severe pains in both legs (not localised). He was evacuated to England. He stated that he was diagnosed "rheumatism and heart trouble." He was sent to Torquay and returned to New Zealand in January, 1918. He had no attacks from the date of leaving England until June, 1918 (midwinter), when he began having chills as before. These were present without intermission from that time, June, 1918, up to the date of admission to hospital, January, 1921 (2 ½ years). At first every ten to fourteen days they increased in frequency until during six months before admission they were as frequent as three every week. They were of the same nature throughout. During the day he would feel restless, complaining of headache, of feeling hot and thirsty. In the early morning he would wake up very cold, would shiver, and then become very hot, with heavy sweating. After the attack he would fall asleep and wake feeling relieved and able to go to his work, which was of a moderately heavy and exacting nature. He worked up to within a fortnight of his admission to hospital. During the last year his weight fell from 16 to 14 stone, but at the same time he noticed increasing distension of his abdomen. There had been no cough. His appetite was good; there was no vomiting or other gastric disturbance; there had been obstinate constipation. There had been no urinary disturbance. Not long before admission he noted a tenderness below the left rib margin, and found that this prevented him from lying on that side. He had been trouble with pain and tenderness in both calves and with occasional swelling of both ankles. Careful questioning failed to elicit any further symptoms.

Condition on Examination.—Patient showed average development with no sign of emaciation. The abdomen was greatly distended. There was relative wasting of all muscles of the left leg (patient was unaware of this). Both feet were clawed, the left more than the right. The fæces was mildly suggestive of a septic condition. There were no petechial hæmorrhages of the skin. The fingers did not show any clubbing. The temperature was 99.8deg., pulse rate 88. Sensation, epicritic and protopathic, was normal over trunk and limbs. There was no spasticity. Reflexes; definite extensor response left big toe, variable extensor right big toe; no ankle clonus, no knee clonus. Knee jerks normal and equal on both sides; abdominal reflexes indefinite. Pupils reacted sluggishly; there was no nystagmus. Examination of the lungs



HEART: A CASE OF MALIGNANT ENDOCARDITIS.



SPLEEN: A CASE OF MALIGNANT ENDOCARDITIS.

NZMJ 21 May 2021, Vol 134 No 1535
ISSN 1175-8716
www.nzma.org.nz/journal

revealed no abnormality. Examination of the heart: no cardiac enlargement could be made out, rate and rhythm were normal, the first sound was obscured in all areas, a marked systolic murmur was heard in the second and third interspaces at the left border of the sternum; the second sound in this area was indefinite and no decision as to its precise nature was made. The brachial and radial arteries were felt to be somewhat thickened. Readings of blood pressure were not taken. The abdomen was distended, making examination difficult. There was tenderness and increased resistance in the left hypochondrium, but it was not possible to determine enlargement of the spleen. The liver was enlarged to one and a-half inches below right costal margin; the enlargement appeared to be regular. There was no free fluid in the abdominal cavity. Rectal examination revealed no abnormality. Examination of the urine showed a trace of albumin, no other abnormality. Subsequent daily specimens showed uniformly small quantities of albumin and a few pus cells; urine was acid. Blood counts showed both red and white cells reduced in number, red cells 4 ½ millions per cubic m.m., hæmoglobin 85 per cent., white cells 4600 per cubic m.m., polymorphs 66 per cent., small lymphocytes 22 per cent., large lymphocutes 7 per cent. Diagnosis was deferred.

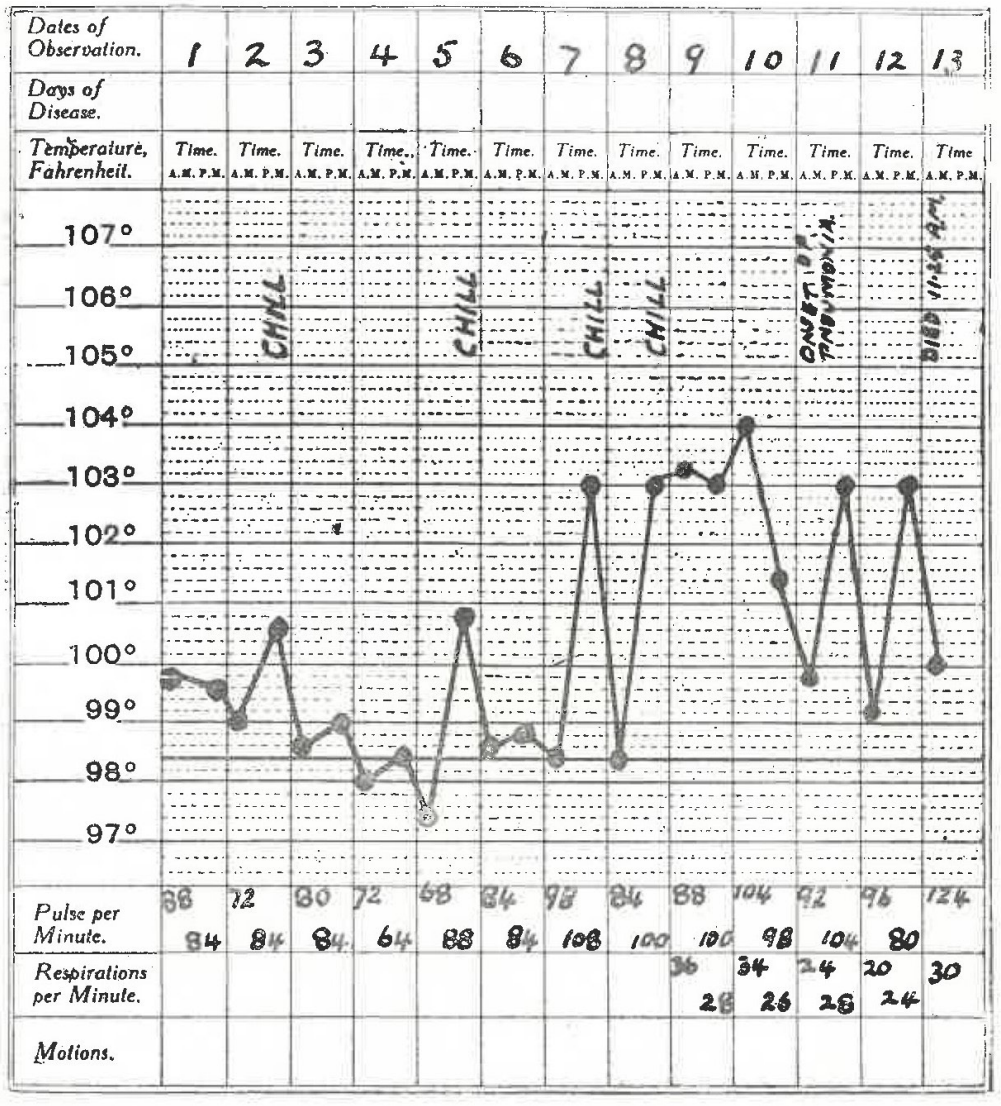


Chart of a Case of Malignant Endocarditis.

NZMJ 21 May 2021, Vol 134 No 1535
ISSN 1175-8716
www.nzma.org.nz/journal

The subsequent history of the case was as follows:— January 1st—Admitted. January 3rd—In the early morning patient had an attack exactly of the type described by him; the temperature rose to 100.6deg. at 4 a.m.; temperature 98.6deg. at 9 a.m. January 6th—A similar attack; temperature 100.8deg. at 4 a.m., temperature 98.6deg. at 9 a.m. In each case the patient's general appearance was improved on the morning follow the attack. January 8th—A more severe attack; temperature 103deg. At 4 a.m., temperature 98.4deg. at 9 a.m. Repeated examinations of blood films showed absence of malaria parasites. The blood serum did not agglutinate bacilli of the typhoid and paratyphoid groups. Complement fixation test for syphilis was "indefinite owing to the amount of anti-complement present" (Mr. Hurley). January 9th—At 4 a.m., as before, a severe rigor; temperature 103.6deg., pulse 98; no fall of temperature after this rigor. Careful examination of the lungs showed no sign of pulmonary involvement; the resonance at the bases was not impaired, the line of resonances moved normally to deep inspiration and deep expiration. Blood examinations showed leucopænia still present. No malaria parasites detected in films taken during rigor. January 10th—Morning temperature 104.8deg. at 9 a.m., pulse 104, respiration 34. Heavy doses of quinine, up to 15gr. four-hourly, had given no relief to the condition, and malaria was finally excluded. Enemata and other appropriate measures had been only moderately successful in reducing abdominal distension, but enabled a large and somewhat tender spleen to be made out. A diagnosis of malignant endocarditis was made. (This was confirmed in consultation with Lieut.-Col. D. E. Fenwick.) The patient was now becoming more ill, but no new physical signs could be made out. Up to this date patient had complained of no symptoms whatever except as regards the distention of his abdomen and the tenderness below the left rib margin. January 11th—Temperature 99.8deg., pulse 92, respiration 24 at 9 a.m. Breathing was accompanied by fine expiratory crackles; on auscultation these were heard widespread over both lungs. During the forenoon there occurred an acute attack of respiratory embarrassment with cyanosis. This soon passed off; no pain was complained of during the attack. Examination two or three hours later revealed consolidation of right middle and lower lobes, with bronchial breathing, bronchophony, etc. Temperature 103deg., pulse 104, respiration 28. Patient showed no symptoms of pneumonia, was quite comfortable, in no pain, breathing was regular and deep, no cough, no sputum; there was very little cyanosis. Patient was examined by the Radiologist (Major D. F. Myers), who reported on the screen appearances as follows: "Right lower and middle lobes are less transradiant than normal, suggesting pneumonic process. No definite cardiac enlargement can be detected. The diaphragmatic cupolæ appear normal in contour." January 12th—Patient in status quo, comfortable, with the same signs of lung consolidation and with the same absence of symptoms. January 13th—Patient was very restless during the night, collapsed in the early morning, and died at 11.25 a.m. from respiratory failure.

The follow report on blood cultures taken early during he stay in hospital was received from Captain D. C. Low, too late to assist in either diagnosis or treatment:—"In first culture gram positive cocci and gram negative bacilli were isolated. In subcultures a growth of gram positive cocci only could be obtained. These resembled staphylococcus aureum."

Owing the circumstances, only a very limited autopsy could be undertaken, but sufficient, however, to obtain the following findings:—Appearance of right lower and middle lobes of lungs that of lobar pneumonia in the stage of red hepatisation. Right upper lobe and left lung not abnormal to naked eye examination. Heart in position of systole, muscle of left ventricular wall much more developed than that of the right ventricle, aortic valves thickened and showing numerous vegetations, other valves normal to naked eye appearances. No apparent disease of the aorta. Liver greatly enlarged, no irregularity of surface, no sign of infarct; liver tissue is paler and more friable than usual. Spleen much enlarged, well-marked infarct at lower pole. Small intestines distended with gas. Left kidney does not show any infarct, but some pus present in the pelvis of the kidney. This was as far as the post mortem was carried, but it served to confirm the diagnosis of malignant

endocarditis, the aortic valve alone being infected. In the illustrations the irregular outline of the aortic cusps (which are distended with cotton wool) is well shown; also the relative thickening of the left ventricular wall. The infarct in the spleen in also shown.

This case presents may departures from the text-book description of malignant endocarditis. In this case the duration of the disease was at least four years. Osler (1918 edition) refers to the duration of chronic infective endocarditis as a matter of months—"six, eight, ten, even thirteen"—and Price ("Diseases of the Heart," 1918) similarly states, "according to some, even eighteen months of longer."

This case conforms more to the septic or pyæmic type of the disease than to the type recently described as subacute bacterial endocarditis with its typical clubbing of the fingers and skin petechiæ, both absent in this instance.

Most observers describe a leucocytosis in infective endocarditis ("10 to 20 thousand per cubic m.m."—Price), but in this case there was a leucopænia present during the whole of the time that the patient was under observation. This increased the difficulty of excluding malaria. The leucopænia may be due to the fact that there is a fair presumption that the infecting organism was a gram negative bacillus; does this explain also the comparative chronicity? Or it may be that by the time the patient came under observation an initial leucocytosis had been converted, by the exhaustion of the defensive mechanisms of the patient, into a leucopænia. It is a remarkable fact that for the space of two and a-half years the patient, although he was having constantly recurring chills, was able to continue his usual work. He did not report until within four weeks of his death. Osler mentions that "the patient may keep at work for months with a daily rise of temperature, or perhaps an occasional sweat," but this patient was having up to three sweats a week. When it occurred, the terminal lung condition was thought to be due to pulmonary embolism, but it now appears to have been a lobar pneumonia, the absence of obvious reaction on the part of the patient being due to a breaking down of those processes whereby the normal organism resists the invasion of disease. The evidence of old injury to the pyramidal tract (left side) is interesting and may date back to the attack in October, 1917, and have been caused by an embolus in the cortex or capsule of the right side of the cerebrum.

I have to thank Colonel A. D. Carbery, C.B.E., Officer Commanding of this Hospital, and General McGavin, D.G.M.S., for permission to publish notes of this case.

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