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#### FOUR CASES OF MALARIA ASSOCIATED WITH ACUTE ABDOMINAL PAIN.\*

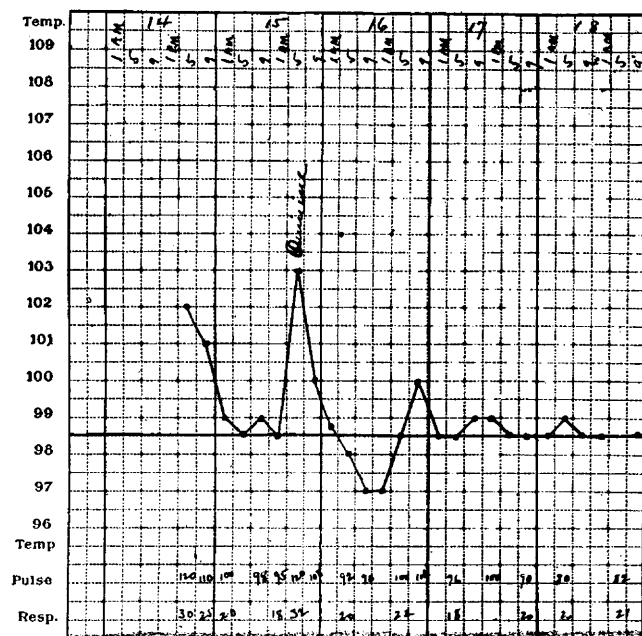
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For the privilege of reporting the following cases from the wards of the Massachusetts General Hospital, I am indebted to Dr. E. S. Cutler, of the visiting staff.

CASE 1.—A married woman of 25 years, living in Arlington, Mass., entered the hospital July 14, 1896. Her father died of pulmonary tuberculosis. Catamenia was always normal. She has one healthy child. Until recently her health had been excellent, with no history of malarial fever nor any of pelvic disease.

For three weeks she had not felt as well as usual, and for ten days had suffered from malaise, headache, occasional



Case 1.—Malaria, double tertian.

Blood: Hemoglobin, 70 per cent.; reds, 4,100,000; whites, 5800.

vomiting, chilly feelings and backache. During the preceding five days she had been confined to bed with daily fever. Two days before admission, upon attempting to walk, she was taken with a chill and vomiting, accompanied by severe pain in the lower abdomen. Since then all food had caused a sense of fullness and soreness in the epigastrium. On the afternoon before admission she had fever, but no distinct chill. She complained also of sleeplessness, loss of appetite and chronic constipation.

The patient was sent to the hospital for surgical interference with the diagnosis of acute salpingitis.

The physical examination showed a body well developed and nourished, face pale, tongue moist and clean, pulse rapid and of low tension. On the upper lip were several herpes vesicles. The lungs were normal. Over the base and apex of the heart a soft systolic murmur was heard and was not transmitted to the axilla. The spleen was palpable and its area of dullness enlarged. There was no especial tenderness of the abdomen to pressure. By vaginal examination the uterus was found retroverted and on the left side there was a slight resistance and tenderness. The urine was normal and failed to give the diazo-reaction. In the evening the temperature reached 102 F., pulse 20, respiration, 30.

No actual chill occurred on the first day, but the blood drawn at the height of the fever showed a few plasmodia malarie. The organisms were all intracellular, and contained fine pigment granules in active motion. The hemoglobin was 70 per cent., red corpuscles 4,100,000, white corpuscles 5800.

At 3 p.m. the next day she had a chill, followed by vomiting, sweating and a temperature of 103 F. During the paroxysm and for several hours afterward there was severe abdominal pain, not sharply localized. Again the plasmodia were seen in the blood and twenty grains of quinin were given in four divided doses every half hour. The fever fell and the pain gradually died away. No recurrence of headache, chill, fever, nor abdominal pain took place, and on July 29 she was discharged well.

The daily intermittent fever and the type of organism demonstrated this to be a case of double tertian malarial infection. The pain came on with vomiting during a paroxysm and persisted for several hours afterward. As to localization, the pain was referred at first to the lower abdomen, then to the epigastrium, later to the whole abdomen.

CASE 2.—A widow, 31 years of age, admitted July 16, 1896. Her father and brother died of phthisis. Seven years ago while living in Hyde Park, Mass., she had malaria. For a year she had been subject to indigestion, and eight months before vomited about a teaspoonful of bright blood.

The present illness began three weeks previously with headache and frequent vomiting, which for the last ten days had been daily symptoms. For six days she had been in bed because of weakness, frontal headache, vomiting, fever and pain in the epigastrium. Four days before entrance these symptoms became more pronounced, especially the epigastric pain, which lasted all day and required morphia. That afternoon a chill came on, followed by sweating, fever and vomiting. The following day she had no chill, but the epigastric pain and headache continued. Two days previous to admission she was taken with a chill, followed by fever, sweating and abdominal pain. The next day there was no chill and no discomfort aside from the persistent pain in the upper abdomen. On the morning of the day of entrance she was delirious, had headache, vomiting and cutting pains in the epigastrium, and at 10 a.m. a chill, succeeded by fever and sweating.

On account of the acute abdominal symptoms she was brought to the hospital by her physician as a surgical emergency, the provisional diagnosis being either gallstone colic or gastric ulcer with perforation.

Upon examination she was found to be in good flesh, dark skinned with a yellowish tinge to the sclerotics. The knees were drawn up as if from pain. Nothing abnormal was found in the heart and lungs. The edge of the spleen was felt. Over the epigastrium there was

\*Read at the Chicago Society of Internal Medicine, May 31, 1900.

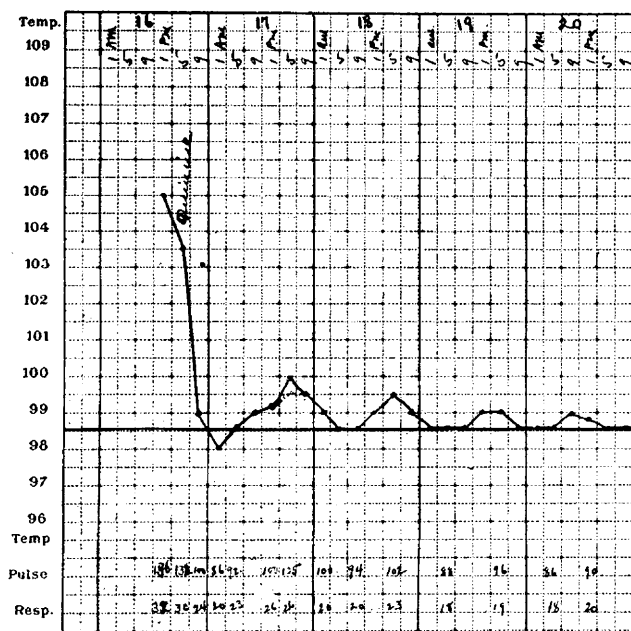
great tenderness to pressure, which was not localized to one spot; otherwise the abdomen was negative. At 1 p.m. the temperature was 105 F., pulse 132, respiration 30.

The blood analysis gave hemoglobin 64 per cent., red corpuscles 3,900,000, and leucocytes 8900 in a cubic mm. This absence of a leucocytosis was such a strong evidence against any acute inflammatory process that a specimen of fresh blood was examined for plasmodia malariae. A few organisms were found, but only after an hour's search in three different drops. These, however, were typical tertian forms with pigment in active motion.

At once twelve grains of quinin were given in solution, and five-grain doses repeated twice at intervals

she had been subject to frontal and occipital headache. Two years ago, after her first confinement, the uterus was prolapsed but was restored to position. From the time of her last confinement, ten months ago, she has been troubled with more or less pain in the right iliac and pelvic regions.

June 17, about one month before admission she had for the first time a chill and fever. Similar attacks came on June 18 and 20, when she began to take daily small doses of quinin. On the 22d another slight chill occurred. After an interval of twelve days, in which quinin was regularly administered in small quantities, she again had a chill with fever following it. No abdominal symptoms were present in any of these attacks. Four days previous to entrance she was compelled to go



Case 2.—Malaria, tertian.

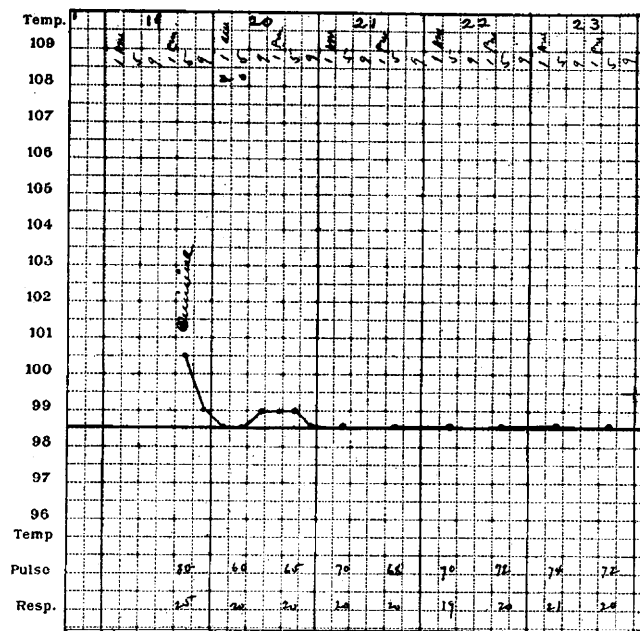
Blood: Hemoglobin, 64 per cent.; reds, 3,900,000; whites, 8900.

of an hour. Within two hours of the first dose she became cinchonized, and in three hours was practically free from the pain. The temperature thereafter was normal, except for a rise to 100 F. on the second day. The epigastrium remained somewhat sore and tender for several days, but no real pain returned. After nine days of treatment she was discharged in good condition, although still weak and anemic.

In this instance there is no apparent relationship between the vomiting and the pain, since the pain continued with greater severity during the intervals between vomiting attacks. It is to be noted that the seat of pain was invariably the epigastric region.

CASE 3.—This was a woman from Brighton, who was admitted July 19, 1896. Two brothers died of "quick consumption." Catamenia as a rule was normal. She had one son living and one dead. When she was last with child menstruation continued the first three months of pregnancy. Her most recent period was of three days' duration, instead of six, as was usual, and at that time several blood-clots of offensive odor were noticed.

Six years ago she was run over by a carriage and her right hip stepped on by a horse. Nearly ten weeks of treatment were necessary before she could walk about again and the right side of the pelvis has always remained a "weak spot." Since an attack of sunstroke five years ago, when unconsciousness lasted seven hours,



Case 3.—Malaria, tertian.

Blood: Hemoglobin, 62 per cent.; reds, 3,610,000; whites, 5000.

to bed on account of cramp-like pains in the lower abdomen and nausea without vomiting. There was no chill nor fever. The next day the pain was more severe, localized in the right iliac region and relieved only by morphia. Intense frontal headache was also present. Two days before entrance she was weak and exhausted from the abdominal pain and fainted away several times. In the afternoon she had a severe chill, frontal headache, vomiting and fever. The day following she was free from all symptoms, save a soreness in the right iliac region. During the day of admission she complained of frontal headache and frequent twinges of abdominal pain and toward evening became chilly and nauseated.

The physical examination showed a delicate, pale, thin woman with a sallow skin. Her expression was one of pain, but not pinched. The thoracic organs appeared normal. The area of splenic dulness was enlarged and the edge of the spleen was felt with some difficulty. The abdomen was tender and resistant over the right iliac region. Vaginal examination revealed a large lacerated cervix and a mass in the left ovarian region, not tender: also a distinct resistance in the right ovarian region with considerable tenderness. The temperature was 100.5 F., pulse 80, respiration 25.

The diagnosis had been made of extra-uterine pregnancy, and like the preceding case, she was brought to the hospital for immediate laparotomy.

That evening a small number of fully developed tertian malarial organisms were found in the blood in active motion. The count of white corpuscles was 5000, red 3,610,000, hemoglobin 62 per cent. Twenty grains of quinin were given in four doses at half-hour intervals.

After this no chill nor fever occurred, while the pain gradually diminished. A week later, however, another severe attack of pain in the abdomen came on, which was greatest in the splenic region and of less degree in the right iliac region. Chilliness accompanied the pain, but no true rigor nor fever. No leucocytosis was present and no parasites were discovered in the blood. A pelvic examination revealed nothing that explained her symptoms. Subsequently under daily treatment with iron and quinin her convalescence was rapid.

January 1, 1897, she wrote that the soreness remained in the right pelvis, but no further chills nor severe attacks of pain had taken place.

In this case there was a chronic underlying condition in the pelvis that probably caused much of the pain, but it was the supervening malarial infection that gave it such an alarming aspect. It would seem that the malarial poison converted a chronic pain and soreness into acute pain, a theory that is borne out by the subsidence of the pain under quinin. Here again the low leucocyte count was of much value in ruling out extra-uterine pregnancy or a fresh pelvic peritonitis. The pain was most intense in the right inguinal region and slight over the spleen. It was not influenced by vomiting.

CASE 4.—A woman of 30 years, living in Somerville, entered the hospital August 6, 1896. Her family history was good. For several years she was troubled somewhat with dyspepsia; otherwise her health had been excellent.

Three weeks before admission she was suddenly taken with slight diarrhea and cramp-like pains in the lower abdomen, chiefly on the left side. Soon vomiting came on and continued at frequent intervals for twenty-four hours, when it gradually ceased. The bowels again became regular in a few days, but a dull aching pain persisted in the lower abdomen. On August 3 she had chill, associated with headache and vomiting. On August 5 the same symptoms recurred, but she suffered mostly from the severe abdominal pain, this time localized in the right iliac region. The pain slowly eased up after the paroxysm until another chill came on the following evening with the same acute pain.

This patient was admitted to the hospital as a case of appendicitis.

The physical examination at the time of entrance was negative, except for a moderate degree of tenderness in the lower abdomen, not especially marked over McBurney's point, and for an enlarged area of splenic dullness. The temperature was 103 F., pulse 100, respiration 30. Blood examination: hemoglobin 73 per cent., red corpuscles 3,820,000, white corpuscles 3600. The urine gave the diazo-reaction.

After the chill a few pigmented tertian forms of the plasmodium malarie in active motion were found in the blood. With the falling temperature five grains of quinin were given and repeated three times at intervals of thirty minutes. Subsequently her temperature remained normal and every symptom disappeared completely. Neither the vomiting nor the diarrhea explains the pain, for it preceded the former and recurred when the bowels were acting normally. Localization was over the lower abdomen, particularly on the right side.

It is to be observed that in all these cases:

1. There were intermittent fever, nausea and vomiting and enlargement of the spleen.

2. There was acute abdominal pain of such intensity that exploratory laparotomy was considered by men of experience.

3. The acute pain subsided along with the fever and with the other symptoms after the administration of quinin.

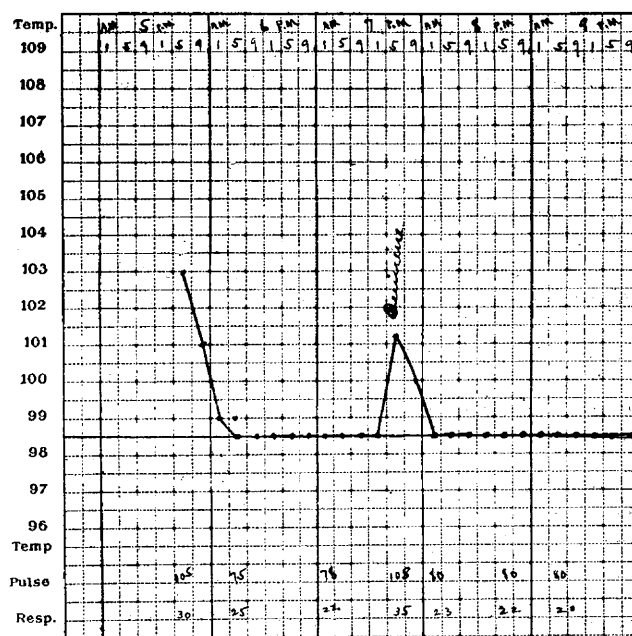
4. Typical intracellular forms of the tertian parasite of malaria with motile pigment granules were present in the peripheral blood, but were never very numerous.

5. Leucocytosis was invariably absent.

6. A considerable degree of anemia existed of the secondary type.

The cause of the abdominal pain was not at first apparent. The explanation does not lie in the vomiting and the incident straining of the abdominal muscles, for the pain often preceded and outlasted the vomiting, and was also present independently. The pain must, therefore, be attributed to: 1, some co-existing disease; 2, a neuralgia from the malarial poison, or 3, both combined.

In two cases local lesions were found that might have caused some pain, but not of such acute character. More-



Case 4.—Malaria, tertian.

Blood: Hemoglobin, 73 per cent.; reds, 3,820,000; whites, 3600.

over, a lighting up of an old pelvic cellulitis or salpingitis sufficient to cause an acute attack of pain, fever and gastro-intestinal disturbance would always produce a leucocytosis, while these cases had counts of 5800 and 5000, respectively, which are even lower than normally.

The chronic disease may be considered in the rôle of a predisposing condition, rendering the part peculiarly susceptible to the malaria poison. The most rational supposition is that the pain is a neuralgia caused by the malaria toxin in the blood.

#### COMPLICATIONS IN THE NERVOUS SYSTEM IN MALARIA.

Since Laveran's discovery of the specific organism, the tendency has been steadily to decrease and limit the number of complications of malaria, because many affections formerly called malarial do not afford the only final and conclusive proof of the disease, namely, the presence of the parasites in the blood. We have, however, abundant indisputable evidence that the malarial poison directly and indirectly affects the nervous system.

The brain, spinal cord, or peripheral nerves may be involved. Authentic cases of delirium, hemiparesis, monoparesis, aphasic disturbances, symptoms resembling those of multiple sclerosis and ataxia, have been reported, but our subject confines us to complications of the peripheral nerves.

*Neuritis and Neuralgia.*—Peripheral symptoms may be motor or sensory, the former being rare and appearing in connection with neuritis. Many cases of malarial neuritis are on record, but those in which the organism has been found in the blood are strikingly few. Among the most reliable observers is Glogner,<sup>1</sup> who describes six cases of multiple neuritis, four of which developed during the infection, while the parasites were present in the blood. Similar observations have been made by other authorities, notably Chiarani and Bardanelli<sup>2</sup> in Rome.

Gowers<sup>3</sup> describes multiple neuritis in malarial subjects, attacking chiefly the lower extremities, where the muscles show true degenerative paralysis.

The neuralgias are frequently met with in practice. The usual forms are supraorbital and intercostal, but they may be located in the brachial plexus, musculo-spiral, crural, sciatic, or sensory nerves supplying the abdominal viscera. Pain in the abdominal nerves is very unusual, but Laveran<sup>4</sup> first pointed out that gastralgia sometimes occurs, referable presumably to the vagus nerve.

Strümpell<sup>5</sup> believes that both cardialgia and enteralgia may be produced by malaria.

According to Thayer<sup>6</sup> very severe attacks of abdominal pain may be associated with a pernicious paroxysm. There is usually profuse vomiting, and not infrequently hematemesis; intestinal symptoms may be quite absent.

*Pathology.*—Most writers are agreed that neuralgia is more often a sequence of malaria than a complication of the fever. Laveran states that in his observation, the pains almost always exist previous to the chills, and that when they occur in conjunction with malaria, are a result of the associated anemia. He considers that the paralyzes are carried by thromboses or emboli in the vessels, made up of plasmodia.<sup>7</sup> Osler finds neuralgias more often from the malarial cachexia than during the fever.<sup>8</sup> The neuralgia, in Gowers' opinion, is not a distinct result of the malarial poison, but due to an anemic and depressed state of the nervous system induced by malaria.

Marchiafava and Bignami question the theory that neuralgia is set up by a localization of the parasites. They assert that sufferers from malaria are apt to have in addition to the fever frequent neuralgias of the supraorbital and other branches of the trigeminal, of the sciatic, crural, intercostal and other nerves, probably as a result of intoxication.

Mannaberg<sup>9</sup> states that no part of the nervous system is exempt from malarial neuralgia and that the pain may be coincident with the paroxysm or follow it.

The neuralgias of malaria may be classified in two groups, the *toxic* and the *post-infective*. The toxic neuralgias come on during the febrile attack, are associated with the presence of plasmodia in the blood and are due to an intoxication with the malarial poison. The post-infective cases are not associated with fever and the presence of parasites, and are the result of anemia and cachexia.

*Diagnosis.*—The recognition of malarial neuralgia appearing during the febrile period, is comparatively simple. The difficulty is in identifying the post-infective cases. According to Gowers the chief feature of the ma-

larial neuralgia is the periodicity, in intervals between attacks ranging from one to four days. Occasionally there are slight symptoms of an ague fit, a trifling hot and cold stage.<sup>10</sup> Leube considers the most weighty symptoms in the diagnosis are enlargement of the spleen, and chills and fever along with the neuralgia.<sup>11</sup> Neuralgias, whether of the head or stomach, that are quotidian or tertian and yield to quinin, Manson<sup>12</sup> would classify as probably malarial.

As pointed out by Marchiafava and Bignami, however, periodicity of pain and relief by quinin are not sufficient to establish the diagnosis of malaria; the organisms must be seen in the blood. Other neuralgias than malarial may be periodic and may yield to large doses of quinin.

We would lay especial stress on the importance, not only of examining a fresh drop of blood for malaria, but of counting the white corpuscles in all obscure acute abdominal diseases, for not infrequently it furnishes the clue to the diagnosis. Salpingitis and appendicitis, when at all acute, beginning peritonitis from any cause, and rupture of an extra-uterine pregnancy are usually accompanied by a decided leucocytosis, while malaria and typhoid fever and simple neuralgic pain are characterized by a normal or subnormal number of leucocytes.

The treatment of malarial neuralgia is first the destruction of any parasites in the system by large doses of quinin. When associated with febrile paroxysms the pain usually disappears in a short time unless the fever is of the estivo-autumnal type. The post-infective neuralgias are best combated by giving in addition to quinin some preparation of arsenic or iron to correct the anemia that is nearly always present.

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### SOME INCIDENTS IN THE EVOLUTION OF THE MODERN PHYSICIAN.\*

WILLIAM ALFRED ELLISTON, M. D.

IPSWICH, ENGLAND.

The president commenced his address with notice of the associations of Ipswich and the deaths of Drs. Stewart and Paget, then passed to the subject of development of British medicine, which was the theme of his address. The early history of medicine in England is that of the Dark Ages; and down to the commencement of the sixteenth century, England had taken no part in the scientific advance of the century, and for almost one hundred years later no discovery of any importance was due to English research. The commencement of British medicine he dates from the return of Thomas Linacre to Oxford from the Italian universities, whence he came imbued with what was termed "new learning."

The great event in the sixteenth century, so far as medicine was concerned, was the success of Linacre in persuading the king to grant a charter to a small body of medical graduates, who were thenceforth called the Royal College of Physicians. He succeeded, it is said, principally through the great and powerful help of Cardinal Wolsey. The charter of the Royal College of Physicians was granted Sept. 23, 1518. It gave them the sole power to license to practice physic in London, or within seven miles radius, with other privileges, which were

\* Abstract of Presidential Address, delivered at the Sixty-eighth Annual Meeting of the British Medical Association, held at Ipswich, August, 1900. [From advance galley-proofs kindly furnished by the Editor of the British Medical Journal.]