Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, ASSISTANT EDITOR

CASE 10181

An American shoe cutter of forty-six entered February 2 complaining of prostration and pain in the chest in the region of the lower four ribs in the right back.

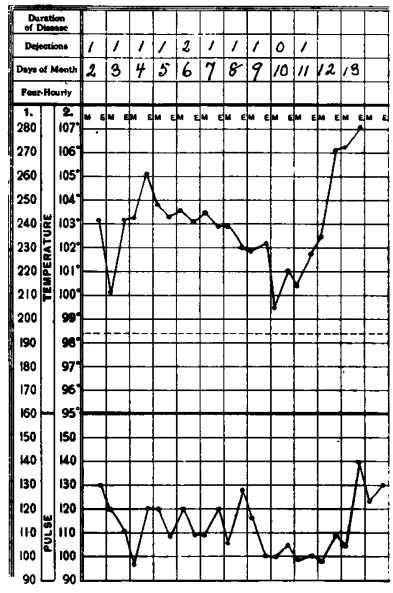
F. H. Unimportant as far as known.

P. H. He had always had good health. He thought he had had all the minor diseases of childhood. A year ago an abscess in the rectum was lanced by a physician without ether. Two days before the onset of the present illness a dentist drilled a hole in a painful tooth to let out pus. The patient thought he was rather nervous. Eighteen years ago he weighed 135 pounds, his best weight. For ten years he had weighed 125.

P. I. Six days ago he had malaise and "a feeling of congestion in his bronchi" which he attributed to a cold, although there was no cough or discharge. Two days later his hands were lame and sore. That evening he vomited and had a chill. The next morning he had pain under his right shoulder blade and began to cough and raise small amounts of thick white sputum. A physician gave him some brick colored powder which the patient thought made his urine red the next morning. It had been high colored ever since. January 31 and February 1 the cough became worse and he had rusty sputum. The pain under the right shoulder blade persisted until the morning of February 1, when it stopped. Since then he had had pain in the right costovertebral region only when he moved about in bed. He had taken nothing but an occasional glass of milk for four days. His bowels had been kept open by "licorice powder" given by his physician. For forty-eight hours he had urinated about once an hour. He had had some difficulty in breathing. The morning of admission his prostration was increased.

P. E. A thin, ptotic man lying flat on his back, breathing rather rapidly. Chest expansion much greater on the left. *Lungs.* Entire front chest and left back clear. Dullness, loud bronchial breathing, increased vocal and tactile fremitus, much increased whispered voice, and rare consonating râles throughout the right back. Apex impulse of the *heart* not found. No enlargement to percussion. P_2 greater than A_2. No murmurs. *Abdomen.* Slight spasm in the right upper quadrant. Liver dullness from the sixth rib; edge felt below the costal margin. *Rectal examination.* Small external hemorrhoids. *Genitals, extremities, pupils* and *reflexes* normal.

T. and P. as shown in the *chart.* R. 33-60. *Urine.* Normal amount, sp. gr. 1.020-1.026, the slightest possible trace of albumin at two of four examinations, rare hyalin casts, once with cells attached. *Blood.* Hgb. 80%, leucocytes 28,000-10,000, polynuclears 89%-96%, platelets markedly increased at one examination, slightly at another; moderate achromia at the first examination. *Two Wassermanns* strongly positive. *Blood culture* at entrance negative. *Sputum.* February 2. Mucoid material with several small areas of red brownish purulent material. Smith stain showed polynuclears, occasional phagocytes, rare red blood corpuscles, occasional Gram-positive spindle-shaped diplococci and rare streptococci in a short chain; no intracellular organisms. By mouse test type II pneumococcus, atypical. Agglutination and precipitation only in type II serum undiluted. One of three later specimens was blood streaked and showed streptococcus mucosus capsulatus.



Orders. February 2. Individual precautions. Soft solid diet. To be fed. Force fluids. Russian oil ℥ ss t. i. d. Veronal gr. x, repeat once if necessary if restless. If not effectual morphia gr. 1/6 s. c. and every three hours p. r. n. for comfort unless respirations below 18. Aspirin gr. x. Veronal gr. x. February 4. Morphia gr. 1/6 s. c. (Three doses.) Digitalis gr. iss daily. February 5-11 inclusive morphia in

1/12 grain doses once a day to every four hours p. r. n. February 5, 7 and 11. Veronal gr. x. February 7. Russian oil ℥ ss b. i. d. Castor oil ℥ ii daily, in capsules if necessary. February 13. Digifolin one ampule given twice. Pituitrin one ampule.

The night of February 3 the patient was slightly delirious and tried to get out of bed. The following night the pulse and general condition were good, although the temperature was higher. February 6 there was a pleural friction rub in the right axilla and at the right base. He complained of pain. February 7 the right lower lobe was resolving. The friction rub persisted. The right chest anteriorly to the level of the third rib showed dullness, bronchial breathing, increased vocal and tactile fremitus, a few coarse inspiratory râles. The night of February 9 he was slightly delirious. The morning of the 12th the condition was very poor. He did not respond to questions. There was no change in the lung signs. He was incontinent of urine and feces. That night the condition was much worse. There was slight bilateral Kernig and double sustained ankle clonus. The knee-jerks were lively and equal. A blood culture showed pneumococci. The blood pressure fell from 120/70 February 12 to 80/50 February 13. That day the patient died.

Discussion

by dr. richard c. cabot

notes on the history

1. The old idea that ischiorectal abscess means tuberculosis has broken down with better understanding, and to-day I should say that only a minority can be shown to have any relation at all to tuberculosis. Most of them come from a septic pile, a septic phlebitis of the rectum, and do not differ from other abscesses except that they are kept going by the necessary passage of feces over them.

2. There are two suggestions of a septic focus here, one in the rectum and one in the teeth; we carry this fact over into our thought about the present illness.

3. I have never known pain in the back to be a symptom of pneumonia. I do not know why it should not be, but so far as I have seen, when we have pain in that region it does not make us think of pneumonia nearly as much as of certain other things. This pain apparently had no relation to cough.

4. The suggestion we get from the present illness is of a general infection which shows itself here and there and by various symptoms, but which has more to make us think of the region of the right kidney than of any other particular lesion. The frequent urination, chill, vomiting, sore hands, cough, I take to be all evidences of a general infection.

notes on the physical examination

The lungs give exactly the signs of pneumonia, though I have been saying from the pain that I do not think that suggests pneumonia. We are still not sure that it is pneumonia, because lesions below the diaphragm with a high diaphragm sometimes get mistaken for pneumonia.

This is a perfectly possible chart for pneumonia, though the last three days of it look like some complication if it is pneumonia. Of course it would go equally well with some other infection.

He had the sort of treatment they would give if they thought he had pneumonia—not the sort of treatment they would give if they thought he had a sepsis.

differential diagnosis

I do not see what we can say except pneumonia with a complicating and terminal meningitis. We have to remember that it is impossible to be sure clinically of a diagnosis of meningitis as contrasted with a diagnosis of meningismus, in which we have Kernig, stiff neck, sometimes an actual increase of cells in the spinal fluid, but without anything which the pathologist is willing to call meningitis, anything more than congestion of the brain post-mortem. I should say that in the long run perhaps we shall be able to recognize this as a stage of the disease, but I do not know that anybody has yet definitely said that.

The only chance of our being far wrong is from some lesion below the diaphragm, e. g., perinephric abscess, which does not seem at all probable. In the first place, people do not die as quickly as that with any such disease, so far as I know. Also I have never heard of meningitis or of meningeal symptoms complicating such a lesion.

We got out of his sputum and later out of his blood a pneumococcus, and I think that is sufficient to rule out other diseases and to say this is a pneumococcus infection, first of the blood stream, then of the lung, then of the brain, whether with definite meningitis or only with congestion we cannot say. If he had lived a little longer undoubtedly they would have tapped his cord and got information there.

When one says pneumonia it generally turns out post-mortem to be more extensive than one thinks. That is to be remembered here.

Especially when we have two different parts of the body apparently attacked by an infection like this, one thinks of the heart as a distributing center, that is, of an acute endocarditis, which as our post-mortems show is not at all uncommon in fatal pneumonias. But here

we can only mention it. There are no signs on which we can base anything more than a guess.

DR. JAMES B. AYER: What would be your feeling about the possibility of infection's starting with the drilling of the tooth two days before the onset of the septicemia?

DR. CABOT: My guess would be that it has nothing to do with it. If we consider the enormous number of those drillings which are done we should naturally expect this to happen much more often if an infection could start in this way. I feel that the relation of the teeth to general conditions is greatly overestimated. We have got into the way of saying "teeth and tonsils" as a single phrase; because the tonsils certainly have a relation to general infections we go on the assumption that the teeth have. It does not seem to me that the teeth have held their place as a source of general infection.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Lobar pneumonia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Pneumococcus septicemia.
Lobar pneumonia.
Terminal meningitis.
Acute endocarditis?

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Septicemia, pneumococcus.

2. *Secondary or terminal lesions*

Focal pneumonia and abscess of right lung.
Acute leptomeningitis.
Edema of lungs.
Acute pleuritis, right lung.
Acute endocarditis of mitral valve.
Soft hyperplastic spleen.
Infarct of spleen.

3. *Historical landmarks*

Chronic pleuritis.
Obsolete area of tuberculosis, apex of left lung.
Luetic aortitis.
Arteriosclerotic degeneration of the kidneys.

DR. RICHARDSON: Was a Wassermann done?

DR. CABOT: Yes, two Wassermanns were positive. I should have discussed that. Nothing in the physical examination or in the history, it seemed to me, gave us any reason for saying syphilis merely because of those Wassermanns. We cannot connect it with his death in any way.

DR. RICHARDSON: There was an acute leptomeningitis present, and rather peculiarly distributed. It was spread over the frontal lobes and the parietal, ceasing abruptly in the region of the anterior margins of the occipital lobes, and with but little good evidence of any meningitis at the base.

The skin and mucous membranes were pale. There was no fluid in the peritoneal cavity. The trachea and bronchi contained a large amount of coppery red frothy fluid. The mucosa was slightly reddened.

A short distance below the apex of the right lung there was a focus of gray-red to gray pneumonia with exudate on the pleura. In the lower part of the lobe just beneath the pleura there was another area of consolidation in which there was a small abscess. There were no areas of consolidation in the middle lobe, but in the upper part of the lower lobe there was a focus of red pneumonia. The tissues elsewhere were dark reddish, yielding a large amount of bloody frothy fluid.

On the left side in the apical region there was a small area of obsolete tuberculosis. The tissue of the upper lobe showed considerable edema. The lower lobe tissue was dark red and generally spongy. There were no definite areas of consolidation in this lobe.

The heart weighed 250 grams. The myocardium was a little lax. The cavities showed slight dilatation on the right. The mitral valve was 9.5 cm. in circumference, and showed scattered along it several small brownish soft vegetations. There was no definite evidence of chronic endocarditis, and the microscope showed the vegetations to be recent. That is, acute endocarditis of the mitral valve.

In the ascending thoracic portion of the aorta there was a strip of definite luetic aortitis. The process faded out when it reached the arch, and the remaining portion of the aorta showed only a slight amount of fibrous sclerosis.

The spleen was slightly enlarged and showed a large infarct.

The kidneys combined weighed 325 grams and showed a few foci of atrophy and a little arteriosclerosis.

Fom the heart blood we recovered the pneumococcus.

DR. JAMES B. AYER: This is the only clinical case I have seen in which the meningitis is apparently so early that we do not already have generalized distribution of the exudate. Of course we get pockets of exudate in chronic cases, but that is different. This is an acute case in which apparently organisms could have traveled in the subarachnoid space, but have not yet done so, or at least if the oganisms are widespread the exudate is not. That is very interesting in connection with the paths of septicemias in hematogenous meningitis.

It so happens that in 1919 I was given the problem to work out in animals the path of infection in hematogenous meningitis. In studying sections I came to the conclusion that while it was not certain, it was highly probable, that the infection was over the convexity in the animal, for the reason that we found the organisms and exudate there earliest, within perhaps two hours after the introduction of the organisms into the blood stream. In these cases the ventricles were not affected, so far as one could judge by pathology, as early as the subarachnoid involvement.

This case, then, seems to check up from the clinical point of view the hypothesis that the infection of the meninges in septicemias is over the convexity. That brings up the question of how the organisms get through. That of course was not demonstrated here or in the animals. One hypothesis is that the route is the reverse of what we think we know about the method of absorption from the subarachnoid space, namely, that the fluid leaves by the arachnoidal villi, a thin membrane which connects the venous sinuses with the subarachnoid space. Here we have to assume that there is a back pressure in some way—perhaps venous pressure is enough to do it—and that the organisms in some way go through with this back pressure. The other route through which organisms are supposed to come from the blood stream is the choroid plexus. The animals did not show pathology so quickly in the choroid plexus as in the subarachnoid space. This patient had a turbid fluid, but we do not know details about it. If the organisms were there they should have gone to the cisterna magna and given a basal meningitis first, and they did not, as the base was practically free. Therefore while the organisms may have gone though the choroid plexus in this case, the exudate, which is probably a safe guide, is primarily over the convexities, and this is what seemed to be the point of origin of hematogenous meningitis in the animals.

DR. CABOT: When you say "infection coming through the choroid plexus"—how does it get there?

DR. AYER: The choroid plexus is a very vascular affair—some call it a gland. Some substances come through—iodin comes through—but most things do not. It is perfectly possible that engorgement of the vessels of the choroid plexus would allow organisms to come through, and of course frequently we find the organism there and the question is whether it came through or has backed up from the subarachnoid space.

DR. CABOT: You mean that there are two hematogenous routes?

DR. AYER: I mean that there are the two chief hypotheses to explain the infection of the meninges from the blood stream. Of course it has a good deal of therapeutic importance whether infection appears first in the ventricles or in the subarachnoid space.

DR. CABOT: In ordinary meningitis isn't the hypothesis of infection through the nose losing credit?

DR. AYER: It is in my opinion.

DR. CABOT: Have you speculated at all why the convexities should be the site in which the infection first appears?

DR. AYER: Apparently there are more arachnoid villi there, and these villi are of course very thin membranes between the blood and the spinal fluid; if organisms pass through these villi the convexities near the longitudinal sinus should show the first traces of infection.

DR. CABOT: Have you any views as to the propriety or impropriety of the term "meningismus"?

DR. AYER: Yes, I have decided views. I do not think we should make a diagnosis of meningitis until we have the organism. The fluid may look alike otherwise. Two years ago at the Eye and Ear Infirmary we had a case in which the fluid was frank pus, but we could not find the organism; this patient got well. I do not believe he ever had bacterial meningitis. It was simply an exaggerated form of aseptic meningitis.

One more point experimentally. It is very interesting how a number of procedures will cause the organisms to come through from the blood stream into the subarachnoid space. Just drawing off the fluid rapidly will turn a septicemia into a meningitis, where if that procedure was not done we should not get meningitis. Simply jugular compression will do the same thing. I am wondering if there is any clinical analogy. I refer especially to trauma as a possible cause of meningitis.

DR. CABOT: Does this work of yours make you feel that we are a little too quick in tapping the spinal cord sometimes?

DR. AYER: No, I think not. We have hunted hard and tried to find clinical cases, but we did not find any that were a sure proof. The point is this. In order to produce meningitis we have to have a septicemia of the right degree of severity,—enough organisms and organisms of sufficient virulence for the meninges. Also we have to have a sudden change in the venous pressure. In the animals, if we withdraw one or two c. c. the pressure drops quickly from 130 mm. to zero. We seldom have the same factors in man that we can have in animals. But I do think we ought not to take an excessive amount of fluid and should use a needle of small bore.

There is one condition in which I should not advise lumbar puncture, i. e. in a patient with known septicemia due to pneumococcus or streptococcus. I should not wish to satisfy my curiosity as to "meningitis" or "meningismus" in such a case.