Lectures.

TUBERCULOSIS OF THE SEROUS MEMBRANES; DEATH; AUTOPSY.

A CLINICAL LECTURE DELIVERED AT THE HOSPITAL
OF THE UNIVERSITY OF PENNSYLVANIA.

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GENTLEMEN, — I shall briefly read the history of this young woman, as it is of importance in the diagnosis.

C. K., seventeen years of age, was born in this country. She has lived in a malarial district of Delaware, and has been occupied as a domestic, working hard, and sometimes, especially in summer, much exposed to damp and bad weather. This was particularly so in the summer of 1882. Her father and mother present a history of rheumatism. She had all the ordinary diseases of childhood excepting scarlet fever. She was perfectly strong and hearty until eight months ago, when, after slight exposure, she was taken sick with pains through the body, headache, loss of appetite, feverishness, some sore throat, with a hacking cough, dull pain behind the sternum, and so much shortness of breath that it was impossible for her to lie down for several days. This reads very much like an attack of influenza. The attack came on suddenly, with general soreness, pains through the body, headache, loss of appetite, fever, sore throat, cough, pain behind the sternum, and so much dyspnea that she could not lie down for several days. This was probably not pneumonia, for we find that in a few days the general symptoms and dyspnea had disappeared, and that she had returned to her normal condition, with the exception of a little cough, which remained for three months. This was dry and hacking, accompanied with scanty mucous expectoration. This, as I have said, sounds very much like the history of an attack of influenza, followed by a catarrhal condition. Of course it is not impossible that it might have been an attack of rheumatism with some heart complication.

Last May she had an attack of acute articular rheumatism, beginning in the feet and involving almost all the joints in the body, attended with very little fever or sweating, but with pain which was sometimes so severe as to bring on a nervous chill. This rheumatism lasted for two months and a half, and since then she has never felt well. Her appetite has not been good, and after eating she has had distress and a great deal of flatulence. The bowels have been regular. She has steadily lost flesh and strength. About October 1st, nine weeks ago, she noticed that the belly was beginning to swell, and this has continued to increase. At the same time she began to have occasional pain in the hypochondriac region. This was never very continuous or severe, and recently has been even less. Since the first of October she has had to urinate more frequently, and the quantity of urine seems to have been increased.

When admitted she was pale. The pupils were dilated, the right somewhat more than the left, but this may have been a natural peculiarity. The pupils reacted well to light. The tongue was coated, the appetite moderate, and the dyspepsia less marked than it had been, the bowels regular. There were occasional

pains through the right chest on taking a long breath. There was a hacking cough, which was worse at night, and was accompanied with a little scanty mucous expectoration. All the time she complained of a sense of shortness of breath. Physical examination of the right side showed no marked signs, but on the left side, in front, from the third rib to the base of the chest, there was flatness on percussion; behind, the whole left side was flat. Over this region there was little vocal fremitus and resonance, and the breath sounds were distant and feeble. The breath sounds were heard a little more distinctly in front, and above the level of flatness a few friction sounds were heard. The area of flatness seemed to change somewhat with change of position. The heart was pushed a little to the left, and on auscultation there was heard a high-pitched, very rough, mitral, systolic murmur. The pulse was dicrotic. The belly was distended, but the veins were not enlarged. As far as could be determined neither the liver or spleen was altered. As I have told you, she passed water too frequently, but it contained no albumen. During November she menstruated and then suffered from some dysmenorrhea. During all this time she has had some fever, which has ranged between a morning temperature of 99° F. and an evening temperature of over 100° F. or 101° F. On one occasion it was as high as 103.5° F. It presents a considerable diurnal variation of a hectic type. There have been no night sweats. The belly has been diminishing a little in size. She has had a good deal of pain in the right side. About December 4th, four days ago, she began to complain of pain in the head, and she also had some deafness. The fever continued as before.

December 6th the following note is made: "Had no headache this morning, but severe during the night. She had slight wandering delirium at night; no vomiting; bowels regular."

December 7th. More delirious during the night, singing, crying, and laughing, and complaining of severe headache. This passed off in the morning. The dullness of hearing increased. During the day the mind was perfectly clear."

When admitted she was put on Basham's mixture with alkaline diuretics. When the fever continued full doses of quinia were given for a number of days, eighteen grains per day being given with solution of acetate of ammonia. This proving unavailing we returned to Basham's mixture and cod-liver oil. As you see, the treatment has consisted of careful attention to diet and in the use of diuretic iron mixture and alkaline diuretics to carry off the pleural and peritoneal effusions, and more recently in the use of bromide of potassium to control the cerebral symptoms which have been mentioned.

This case has given a great deal of anxiety, largely on account of the difficulty in diagnosis, as you see the morbid elements which are present are numerous. You also see why it was that I referred to the possibility of the first attack being one of rheumatism. Her family presents a strong tendency to rheumatism, and in May, five months after the first attack, she had a severe attack of ordinary acute articular rheumatism, which has left behind it mitral regurgitation, as shown by a strong, low-pitched, rough mitral, systolic murmur. In addition, we have coming on without any oedema, internal dropsies, right pleural effusion and ascites, and this long-continued febrile action, and more recently these cerebral symptoms.

In trying to arrive at a correct understanding of the nature of this case the following thoughts have occurred to me: Is this a case of chronic malaria, complicated with sub-acute rheumatism and great anæmia leading to these internal effusions? Is it a case of protracted rheumatic fever with ulcerative endocarditis, with the endocarditis progressing, keeping up the irregular fever with irritation of the serous membranes causing effusion, as will sometimes occur in these long-continued cases of septic rheumatic trouble? Not rarely in such cases will you find nervous symptoms appearing in their course. Is it a case of rheumatic trouble with serous tuberculosis, causing tubercular pleurisy and peritonitis and recently tubercular meningitis? It is very difficult to answer these questions.

You will observe from the history that all renal complications must be left out of consideration. The urine has always been normal. There is no question as to the lesion of the heart. The only question is whether or not it existed before the last attack.

The effusion on the right side interfered greatly with determination of the condition of the lung. Now that this is somewhat removed we may be able to obtain better results. On auscultation, I detect on the left side, in front, below the outside of the cardiac region, sharp, crackling, friction sounds. Over the lower part of the right chest in front, where it is still comparatively dull and slightly tender, the respiratory murmur is weak, but there are no râles or friction sounds. Posteriorly the respiratory murmur is feeble, distant, and blowing. After coughing, I hear a few deep-seated, coarse, snoring râles. The right pleura is thickened by exudation, compressing the lung. This exudation is plastic. There is a condition of chronic pleurisy of the right side, and there is a small patch of dry pleurisy on the left side.

Let us now turn to the abdomen. The distention has been somewhat reduced, but fluctuation is still very distinct. The abdomen is very tender on pressure; I think that there can be little doubt that this effusion is dependent on peritonitis. This is not a passive effusion. There is no affection of the kidneys nor of the liver. The liver is not enlarged, and there is no jaundice. There has been pain which has been referred to the region of the liver and to the right shoulder as though the capsule were involved, but that would not be improbable on the supposition that there was peritonitis. The heart lesion cannot explain the ascites. There is no œdema of the feet or hands, and in cardiac dropsy internal effusion, and especially ascites, does not occur unless the œdema is very great. The degree of the heart trouble is not very serious, but its quality may be. To this I shall refer in a minute. I think that we must regard the peritoneal and pleural effusions as irritative and the result of inflammation of the membranes.

I feel less certain in regard to the nature of the cerebral symptoms, and before expressing any opinion I shall wait until I have obtained the result of the ophthalmoscopic examination, which I consider necessary. At present her condition is a typhoid one, and it would not be unnatural that, after such an illness, cerebral symptoms of a low, adynamic, typhoid type should develop. I should warn you against concluding, even in the presence of the most threatening symptoms, that organic disease of the membranes had developed. We have in the ophthalmoscope a most important aid in the diagnosis of such cases as this, and

one which should always be employed. In meningitis, especially of the tubercular variety, retinal changes are apt to develop, which are sometimes so characteristic as to enable the diagnosis to be positively made at a glance. If the symptoms are due to the typhoid condition the results of the examination will be negative.

What other condition than tubercular peritonitis and pleurisy could we have running a slow course and engendering a low typhoid condition with cerebral symptoms the result of the adynamic condition or of meningeal irritation? Another explanation might be found in a septic state of the system from ulcerative endocarditis, a rare condition, but one which is met from time to time, and presents such peculiar symptoms that it requires careful study before it is recognized. Ulcerative endocarditis runs the course of chronic pyæmia. The detritus from the degenerating endocardium, which presents little ulcers which sometimes perforate the valve and sometimes form little masses on the surface of the valve, is thrown into the blood, and passing through the system gives rise to numerous minute septic emboli. Septic foci are established in the spleen, kidneys, and lungs, and with this there is pyæmic fever of a hectic type with marked diurnal variations in temperature, sometimes with distinct sweating, but not, as a rule, with the drenching sweats which attend the rapid development of surgical pyæmia. This runs a slow and chronic course. It is attended with various secondary lesions. Sub-acute inflammation of the serous membranes is not rarely met with. Here we have a girl who has had an attack of a vague character and a second attack of distinct rheumatism, who now has marked cardiac disease. There is no history of tubercular disease in her family. But this is not essential. The association of tuberculosis with cardiac disease is not frequently met with, but still it may occur.

In either case the prognosis is most grave. If there were lesions in the lung there would be no doubt as to the diagnosis. As it is, I must say that my own judgment is in favor of the view that there is tuberculosis of the serous membranes. The ophthalmoscopic examination will be of great importance. I shall have it made and give you the results next week.

The diagnosis rests between these two rare and grave conditions, and on the whole I think that the balance of evidence is in favor of the view that there was an old mitral lesion which was increased by the last attack, and which is now associated with tuberculosis of the serous membranes.

The treatment has been simply a supporting and apyretic one. We cannot use specific remedies against either of these conditions. I do not think that we can do better than continue the careful regulation of diet and the tonic and deobstruent treatment which has been pursued, and the use of bromide when called for to allay the excitement.

(The ophthalmoscopic examination showed indications of intra-cranial trouble. Descending neuritis with œdema of the retina was found, but no retinal tubercles were detected.)

December 22d Dr. Pepper again referred to this case as follows:—

The young girl, C. K., whom you saw two weeks ago, gradually failed and died on December 16th. The autopsy was made by Dr. Formad, who gives us the following report: "A tall, well-shaped girl, not markedly

emaciated, but very anæmic in appearance. The abdomen greatly distended with gas and ascitic liquid. Old adhesions obliterated entirely the right pleural cavity. There was no liquid effusion. The pericardium was normal, but contained about five ounces of clear fluid. There was slight thickening of the mitral valve and shortening of the cordæ tendinæ, allowing regurgitation. The right lung was collapsed, the pleura much thickened by plastic exudation and the seat of tuberculous formation. A moderate amount of gray miliary tubercles were found scattered through the substance of the right lung. The left lung also contained a number of gray tubercles, but not as many as the right lung, and without plastic pleurisy.

"The peritoneal cavity contained two quarts of straw-colored liquid. All parts of the peritonæum presented gray tubercles. There were old adhesions in many parts, particularly in the lumbar region around the mesentery and pancreas, matting together the viscera. This new-formed mass was crowded with tubercles. The lymphatic glands were moderately enlarged but not cheesy.

"The liver was normal, its capsule studded with tubercles, but no tubercles were discovered in the parenchyma. The spleen was covered with tubercles, but none inside. The kidneys showed signs of an early stage of chronic parenchymatous nephritis, but this certainly had not advanced far enough to cause alteration of the urine, for this was carefully examined a number of times with negative results.

"The brain was normal, only a few gray tubercles being found in the Sylvian fissure. There was about sixteen ounces of serous effusion."

Original Articles.

CONCERNING THE ÆTIOLOGY AND PATHOLOGY OF HYDRAMNIOS.¹

BY CHARLES M. GREEN, M. D.

SINCE writing the paper² which I read to this Society in February last, wherein I sought to formulate the present knowledge of the origin and pathology of hydramnios, I have met with another contribution to our knowledge of the subject by Dr. Nieberding of Würzburg, who has recently observed and carefully recorded³ two cases.

In the winter of 1883 Nieberding observed hydramnios in a case of twins from one ovum which he reported in a monograph entitled *Beitrag zur Kenntniss der Genese des Hydramnion*. In this case not only was one twin more developed than the other; but there was a considerable effusion in its cavities and great œdema of its whole body: hydramnion existed in only one of the amniotic sacs, and that one contained the larger fœtus. The placenta was single, and that part of it which nourished the larger fœtus was of greater size and was in a high degree infiltrated with serum. The pregnancy terminated at the end of the sixth month, and while the smaller fœtus showed no anomaly except a certain hyperæmia, examination of the larger fœtus disclosed a considerable hypertrophy of the heart and kidneys, and great infiltration of all

the organs with red and white blood-corpuscles. The hypertrophy of the heart was chiefly of the left side.

If now the question is asked, says Nieberding, to what causes are to be attributed the pathological increase of the liquor amnii of one sac and the dropsy of the fœtus contained in this sac, it must be answered that it is possible that an increased transudation from the maternal blood-vessels might have been the causal factor. Since, however, originally the size and presumably the permeability of both embryos were approximately the same, there is no reason why one sac should be thus filled and not the other. The idea that the vasa propria of one part of the placenta contribute conspicuously to increase the amount of the liquor amnii over and above the same vessels of the other part Nieberding says must be abandoned, since it was established by microscopical examination in this case that the chorionic capillaries were equally developed on both parts of the placenta and that those of the hydramnic part, instead of remaining patent longer than usual, were in great part obliterated. Nieberding therefore believes that the cause of the hydramnic condition of one sac in conjunction with dropsy of the contained fœtus must be sought in the circulatory conditions of this fœtus; and this belief is warranted by the pathological appearances in the present case, which were as follows: hypertrophy of the heart, especially of the left side; great enlargement of the kidneys with dilatation of the pelvis, filling of the glomeruli with red and white blood-corpuscles, and the presence of moderate blood-extravasations; considerable dilatation of the ureters and of the bladder, although the urethra was entirely permeable. These pathological changes must have their causes, and these causes were traced by Nieberding to a premature obliteration of the ductus arteriosus. The early closure of this duct must surely entail great changes in the fetal circulation: more blood must perforce enter the lungs; at the same time the blood-tension in the right ventricle must be greatly increased. On account of this tension not only would there result a backing-up in the whole venous circulation; but at the same time a greater quantity of blood would be forced through the foramen ovale into the left side of the heart, which on account of the greater demands thus made upon it to force on the increased amount of blood would hypertrophy and thus increase the tension in the arterial system. Herein, according to Nieberding, lies the explanation of all the anomalies of the affected ovisac and its contents: while the fetal œdema and dropsy were caused by the venous backing-up, which through the umbilical veins occasioned also the serous infiltration of the placenta, the increased arterial pressure resulted in an increased filtration by the kidneys, an increased diuresis: this increased amount of urine must have been evacuated from time to time, else there would have been found an enormous distention of the bladder; and this polyuria, according to Nieberding's firm conviction, must have had the most conspicuous share in the dropsical distention of one ovisac, while, on the contrary, the weakened heart of the other twin caused a lower arterial tension, a smaller secretion of urine and a minimum amount of fluid in its containing amnion.

Quite recently Nieberding has had a second instance of hydramnios in a case of single fœtation. A previously healthy secundipara, thirty-two years of age, whose first pregnancy and labor had pursued a normal

¹ Read before the Obstetrical Society of Boston, May 10, 1884.

² See vol. cxi., No. 6, page 121, of this JOURNAL.

³ Allgemeine Wiener Medizinische Zeitung, March 11, 1884.