Clinical Lectures

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LECTURE XI.

ON THE DIAGNOSIS OF DROPSIES.

Diagnosis of External from Internal Abdominal Dropsy. Diagnosis between Primary and Secondary or Indirect Dropsies.

GENTLEMEN,—I wish to call your attention to-day to some cases illustrating the Diagnosis of Dropsies.

The seat of certain dropsies is often diagnostic of their cause. Dropsy as a general symptom, taken in the broad, may result from an almost endless variety of diseased conditions; and it is only by determining the characters and particular features of the dropsy in each case, that it becomes diagnostic of the disease that generates it. Of all the circumstances of dropsy that impart to it a diagnostic character, its localisation is, perhaps, the most important and indicative. This circumstance, taken alone, is almost sufficient, in a great number of cases, to determine the diagnosis. Thus, if I were asked what dropsy of the face pointed to, I should say disease of the kidneys; if I were asked what dropsy of the abdominal cavity pointed to, I should say disease of the liver; and if I were asked what dropsy of the legs pointed to, I should say disease of the heart or lungs. And, in the majority of instances, I should be right, especially if these dropsies occurred without any other distribution.

The particular point for to-day—that to which I would especially direct your attention—is the diagnosis between external and internal abdominal dropsy; between the accumulation of fluid in the peritoneal cavity, and the accumulation of fluid in the subcutaneous areolar tissue of the abdomen: in other words, between ascites and oedema of the abdominal wall.

From what I have said just now, you will see the importance of this:—Ascites points to the liver; subcutaneous dropsy does not. The determination of this question, therefore, determines approximately whether we are to regard the liver as concerned in the diseased process, or not. But, though you may admit the importance of the distinction, you may be disposed to regard the discussion of the method of making it as a superfluous inquiry. This, however, is not the case. Many a time I have had patients sent to me as labouring under ascites who have not had a drop of fluid in their peritoneal cavity, but the increased girth of whose abdomens has been entirely due to oedema of the abdominal wall.

On what rules, then, may we fall back for the resolution of a case in which there is undoubtedly an increase of the girth of the abdomen, in which that increased girth is undoubtedly due to fluid, and in which the causes of dropsy undoubtedly exist? How

shall we determine, in such a case, whether the effusion is external or internal to the abdominal cavity?

1. If the effusion is external—if the abdominal enlargement is due to oedema of the parietes—the following conditions will be found to be present.

a. On attempting to pinch up the skin of the abdomen, we shall find that we pinch up a thick firm “roll” of integument, firm and doughy, an inch or more in thickness; and we generally find that the lower down on the abdominal surface that we attempt thus to pinch up the skin the thicker is the roll of integument which we raise; because the lower is the abdominal surface the more developed is the oedema.

b. We find the umbilicus deep-set, and deep-set in proportion to the oedema. This is always the case; and the reason of it is this:—At the umbilicus the skin and the deep fascia are fastened to one another, and cannot be separated; elsewhere, from the intervention of a loose and extensible areolar tissue, the one can be freely raised from the other. Now, it is into this areolar tissue that the dropsical effusion takes place; and by this effusion, and in proportion to it, the deep and superficial fasciæ are separated from one another, and the skin raised. In proportion, therefore, to the effusion which raises the skin from the deep fascia will be the depth of the pit at the point where it cannot be raised. This deep-set umbilicus is very characteristic, and I would especially recommend your attention to it.

c. Again, the parietes have a peculiar white opacity about them—an unnatural and uniform whiteness; and this, I think, is in part due to another appearance—an absence of any visible veins. The superficial veins lie in the subcutaneous areolar tissue; and this is so thickened and distended by the oedema, and the skin thereby so much raised, that the veins are no longer immediately beneath the surface.

d. Another characteristic of oedematous abdominal parietes is a peculiar quaggy vibration in them when they are tapped—a sort of jelly-like tremor. This may even be seen, but it is better felt by the hand. It is very important to recognise it, because it may be confounded, and often is confounded, with the true “fluctuation” of ascites. It is best felt in this way:—Place one hand lightly on the surface, and with the fingers of the other hand “flick” the skin close by; the peculiar thrill or tremor will be at once felt. And it will be found that the further the hand is removed from the point “flicked” the fainter are the vibrations, until, at some little distance, they are quite lost. I do not think they can ever be felt quite across the abdomen, from one side to the other. As far as they can be felt, the sensation is as if they were conducted along the surface, and never as if they were conducted through the abdominal cavity. I shall recur to these points presently, in contrasting this spurious fluctuation of oedema with the true fluctuation of ascites.

e. Again, the phenomenon so distinctive of oedema—pitting—is always present. Sustained pressure leaves the impression of the fingers; if we attempt to pinch up the infiltrated integument, by so doing we squeeze the fluid from it; and, on releasing it, depressions are left corresponding with the parts pinched.

f. Another appearance, and one which should

always raise a suspicion of oedema wherever you may see it, because it is indeed but another form of pitting, is visible marks of the bedclothes, or folds of the patient's dress, on the skin.

The three first of these signs—the thickness of integument when pinched up, a deep-set umbilicus, and an invisibleness of veins—you get equally in very fat subjects; and for this simple and manifest reason, that fat, like oedema, raises the skin, and inserts a material in the subcutaneous areolar tissue. But you will have no difficulty in distinguishing the one from the other: the proportionate distribution of fat elsewhere, the patient's condition in other respects, and, above all, the presence or absence of pitting, will leave no doubt upon your minds.

2. If the effusion is internal—free in the peritoneal cavity—the following signs will be present.

a. In the first place, if the fluid is in any appreciable quantity, sufficient conspicuously to enlarge the abdomen, there will be what is called “fluctuation”. This is something very different from that quaggy tremor which I have described as characteristic of oedema of the abdominal wall. I think a better name for it would be “vibration”. It depends upon the transmission across the abdominal cavity, from one side to the other, of a vibration imparted by a light stroke. It is best elicited in this way:—Apply your hand to the lateral or inguinal region of one side, and then flick or touch lightly the surface on the opposite side; you will find at each touch or stroke a little single wave transmitted through the fluid, and impinging on the fingers on the opposite side. The sensation which it imparts can never be mistaken when once felt; and you should all of you take an early opportunity of making yourselves practically acquainted with it: it will make a clearer impression on your minds than any description of it I can give you. It differs from the spurious vibration of oedema in these three points. In the first place, distance makes no difference to it; it is felt just as plainly completely across the abdomen as half-way—indeed, I think, better; whereas the strength of the vibrations in the spurious form is always proportionate to the shortness of the distance between the part felt and the part struck. In the second place, it consists, not of a quaggy tremor, but of a single wave; the impulse on one side being transmitted unchanged to the other. In the third place, it is evidently transmitted, not along the surface, but through and by the contained fluid.

When I said just now that distance makes no difference to it I should have qualified this expression in one particular, for distance does make a difference as to the *time* at which the vibration is felt; for, if the distance is great, as from one flank to the other, the vibration impinges on the fingers at one side, at an appreciable interval after it has been imparted at the other, the interval being proportionate to the distance. This lapse of time between the stroke and its resulting wave is one of the most striking and characteristic parts of the phenomenon.

From what I have said you will see that a vibration felt near the part struck is of no value as implying the existence of a true so-called fluctuation. It is only when transmitted quite across the abdominal cavity, that it implies the accumulation of fluid in the peritoneum.

b. In the second place, if we have fluid in the abdominal cavity we shall have dulness of percus-

sion in the most dependent parts, and to an extent corresponding with the amount of the fluid. If the patient is supine, the dullest parts will be the flanks; if he is erect, the hypogastric, or hypogastric and umbilical regions.

c. Again, the umbilicus, instead of being deep-set, as in oedema, is unnaturally flattened out—indeed, in some instances protruded. The distending fluid dilates the umbilical orifice, and then drives through it a sort of hernia, the fluid within which, acting like a wedge, dilates the orifice more and more, till a considerable dropsical hernia exists, raising the thin integument over it to the size of a walnut, or even half a billiard-ball, through which there is often an appearance of an opalescent transparency, like that of a hydrocele. This appearance, however, is exceptional; the common appearance of the umbilicus in ascites is merely an unnatural flatness or very slight prominence.*

d. Lastly, instead of an absence of veins in the abdominal wall, there is often, in ascites, a great conspicuousness of them, sometimes even amounting to a varicosity. This depends on two causes: the veins are *visible* because, by the distension, the integument is stretched and thinned out, and therefore rendered more diaphanous; and they are *enlarged*, because the incumbent weight of the accumulated fluid exercises such a pressure upon the inferior cava as to impede the return of blood through it, and compel it (the blood) to find its way back to the heart by the superficial collateral venous circulation formed by the anastomosing epigastric and internal mammary veins.

Now let me call your attention to some cases in the hospital illustrating the practical application of these rules. We have two patients with abdominal enlargement due to dropsy—Mary Jones and George Davis; and they illustrate very well the striking contrast between external and internal abdominal dropsy.

In Mary Jones's case, which is one of chronic bronchitis, the enlargement of the abdomen is entirely due to an oedematous condition of the abdominal walls. Here we have the thickening of the integument, making it almost impossible to pinch it up, and then only in a roll an inch or an inch and a half thick; the deep-seated umbilicus; the quaggy tremor; and the pitting. The oedema of the abdominal wall is but a part of the general oedema; the legs, as you have seen, are enormously oedematous.

In the case of George Davis, which is one of mitral disease, we find, on the other hand, that while the abdomen is greatly enlarged, the abdominal wall is extremely thin; when we pinch up the attenuated skin, it has not much more than the thickness of paper. We find, too, the flattened umbilicus, almost effaced by distension; the conspicuous veins meandering over the surface; and, above all, true fluctuation.

There is one more point in relation to the diagnosis of dropsy, on which I would wish to say a few words to you.

I have two cases in the hospital at the present time, both of which are characterised by the following four circumstances:—

* Since this lecture was delivered, I have seen a case in which this dropsical umbilical hernia was well developed as a result of ovarian dropsy; it gave me the impression, on first seeing it, that the case was one of ascites.

1. Mitral regurgitant disease ;
2. A greatly enlarged liver ;
3. Ascites ;
4. Oedema of the lower extremities.

One of these cases is that of George Davis, to which I have just referred; the other is that of John Flynn, a boy aged 12 years, lying in the Bow Ward, Bed No. 14.

Now, with regard to these cases, the two following questions suggest themselves:—

1. Is the enlargement of the liver due to primary hepatic disease, and the ascites, therefore, true hepatic ascites? or is it due mainly to hepatic congestion dependent on the impediment to the circulation through the heart, and the ascites, therefore, not true hepatic ascites, but indirectly cardiac?

2. Is the oedema of the legs the direct and immediate result of the heart-mischief? or does it result from impediment to the return of blood through the inferior cava, produced by the pressure which the incumbent weight of the ascites exercises upon that vessel? or is it due to the pressure of the enlarged liver upon the inferior cava in the supine posture?

According to the answers we may make to these questions we shall arrive at one or other of the following conclusions:—

That the liver is the seat of organic disease of such a nature as to obstruct the circulation through it.

That the ascites is the direct result of this liver-disease.

That the liver is not diseased at all, but is enlarged because congested.

That the obstruction, therefore, which gives rise to the ascites, though immediately at the liver, is primarily at the heart.

That the oedema of the legs is due to the systemic venous stasis of the heart-disease.

That it is due to the pressure of the enlarged liver upon the inferior cava in the supine posture.

That it is due to the incumbent weight of the ascites pressing on the cava. This last will give rise to two alternatives; for if we consider the ascites due to liver-disease, then the oedema of the legs is a secondary hepatic symptom; if the ascites is due to the heart-disease, then the oedema is a secondary cardiac symptom.

You see, then, to how many alternatives our answers to these two questions may give rise.

Now, with regard to the first question—the nature of the liver-enlargement—I came to the conclusion that, in the case of Davis, it was secondary to the heart-disease—due simply to congestion; and that the liver was not the seat of any real disease at all: for the following reasons:—First, the patient was of an age, 20, at which organic enlargement of the liver is not common; secondly, there was nothing in his antecedents or habits—no intemperance—to make liver-disease likely; thirdly, the liver was not the seat of any pain or tenderness; fourthly, although greatly increased in size, it was not altered in shape; fifthly, the very existence of the ascites rather pointed to the cardiac origin of the liver-enlargement, for such a cause of enlargement would necessarily also be a cause of ascites. In the case of Flynn, I thought at first that the liver was the seat of independent enlargement unconnected with the heart; the enlargement was so great and the ascites so moderate. And even now I do not feel certain. No doubt the volume of an organ so highly vascular as

the liver may vary within very wide limits according to the amount of its turgescence. We know, too, that the liver is more advantageously placed for having its circulation influenced by the state of the heart than any other organ of the body. And yet, as a clinical fact, we find that it is comparatively rare for the liver to undergo any great amount of enlargement as a result of mere passive congestion from heart-disease. Is the organ so greatly enlarged in these cases on account of the youth of the patients? Does the liver in the young yield more readily to the enlarging influence of mechanical congestion than in those of more advanced age?

With regard to the part that ascites or enlarged liver might be supposed to play in the production of the oedema of the lower extremities, that question is, I think, set at rest in the case of Davis, in whom, for some days past, the ascites and enlargement of the liver have both been greatly diminishing, while the oedema of the legs has been continuously increasing. Had the latter been due to either of the former, it also must have diminished as well. It is clear, then, in the case of Davis, that the oedema of the lower extremities is to be assigned entirely and directly to the heart.

The future of these cases, gentlemen, must resolve the other alternatives that I have indicated to you; and I have pointed them out in order that you may see how complex and dependent a symptom dropsy is, and how many and what varied conditions it may imply.

FACTORY-SMOKE AND HOUSE-SMOKE. From the researches of Dr. Crace Calvert, F.R.S., it is ascertained that whilst dwelling-house smoke is comparatively harmless, factory-smoke is highly deleterious. He explains that the smoke from private dwellings carries with it only carbonic acid, carbonic oxide, and sulphuric acid gases, and a small quantity of the most volatile hydrocarbons, the less volatile products forming "soot," which remains in the flue. The gases, the escape of which continues but a few minutes after firing, have but little or no action on vegetation or man. In burning coal under steam-boilers the results are very different. The fuel is not, as in private dwellings, perfectly consumed, but there is a continuous distillation of deleterious tarry products, which by the draught of the tall factory chimney are carried, with all the other noxious products, into the cold atmosphere at the top of the stack, where they quickly condense and fall, to poison all within their reach. (*Mining Journal*.)

SUDDEN BLANCHING OF THE HAIR. The *Boston Med. Journal* cites from a recent number of *Virchow's Archiv* a case of sudden blanching of the hair. The case occurred in Professor Mosler's clinic. The patient was 34 years old, and was suffering from delirium tremens. On the fifth day of his stay in the hospital, the visiting physicians and the patients noticed that the hair upon his face and head had become grey. On looking at himself in a mirror, he exclaimed, "Ach, Gott, mir sind die Haare grau geworden." A microscopical examination showed the presence of a great many minute bubbles at the white points, both in the cortical and central portions of the hair. The pigment was perfectly preserved throughout the whole shaft of the hair, and had undergone no change whatever. As the hair gradually is changed to grey, the pigment disappears, but in this instance, the rapid whitening during a single night was produced by the development of gas within the substance of the hair.