LECTURES

ON THE

PATHOLOGY, DIAGNOSIS, AND TREATMENT OF BRIGHT'S DISEASE.

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LECTURE IV.—CHRONIC BRIGHT'S DISEASE WITH A LARGE WHITE KIDNEY.—(Concluded.)

Symptoms.—*Dropsy.*—*Pulmonary Complications.*—*Inflammation of the Serous Membranes.*—*Endocarditis.*—*Dyspepsia.*—*Vomiting.*—*Diarrhoea.*—*Hypertrophy of the Heart.*—*Cerebral Symptoms.*—*Hæmorrhage, etc.*—*Defect of Vision.*—*Hæmorrhage from Mucous Membrane.*—*Diagnosis.*—*Prognosis.*—SIMPLE FAT KIDNEY, OR GENERAL FATTY INFILTRATION OF THE KIDNEY.—*History.*—*Microscopic Characters of the Kidney.*—*Pathology and Clinical History.*—*Points of Difference between it and the Granular Fat Kidney.*

SYMPTOMS.—Amongst the symptoms of chronic Bright's disease with large white kidneys, *dropsy* is one of the most frequent and prominent. The cases in which dropsy is absent throughout the whole progress of the disease, form a very small minority. Some years since I found, from an analysis of twenty-six fatal cases, that there had been dropsy in twenty-four; the proportion being, in round numbers, 92 per cent. And in the majority of cases the dropsy was great, general, and of long duration. (See my paper on the Forms and Stages of Bright's Disease, *Med.-Chir. Trans.*, vol. xlii.) When chronic disease has supervened upon an acute attack, the dropsy which attended the onset of the malady may continue throughout, or it may pass away and return after a variable interval of months or years; or, if the urine become copious, as it sometimes does in the second and third stages, the dropsy may never return. Acute renal dropsy usually becomes general within a short period; but the dropsy which accompanies this chronic form of disease shows itself first in the face and feet, and gradually extends to other parts, including, in the worst cases, the serous membranes of the chest and abdomen. The pale, pasty, and puffed appearance of the face is very characteristic of this form of renal disease. As a rule, the more scanty is the secretion of urine, and the larger the proportion of albumen which it contains, the greater is the tendency to dropsy.

When dropsical swelling of the legs becomes excessive, so as to cause great tension of the integuments, the circulation through the skin and the subcutaneous tissues is seriously impeded; and this may result in erysipelatous inflammation, sloughing, and gangrene. In one case, I saw gangrene and sloughing of the skin over the back of both legs, excited by the pressure of the heavy dropsical limbs upon the bed.

Congestion and œdema of the lungs are often associated with, and may be said to form a part of, the dropsical symptoms. The lungs are not rarely the seat of inflammatory mischief. *Bronchitis and pneumonia* are amongst the more frequent and serious complications. *Submucous œdema of the larynx* occasionally occurs, and renders the voice husky; more rarely it causes stridulous breathing and dyspnoea. *Inflammation of the serous membranes* is one of the less frequent complications. According to my experience, the pleura is more frequently inflamed than the pericardium; the peritoneum less frequently than either. *Endocarditis* is sometimes set up, and may result in a chronic valvular disease. *Derangements of the stomach and bowels* are of common occurrence in all stages of the disease, but more especially in the advanced stages, and when, from any cause, the secretion of urine becomes scanty. There are loss of appetite; dyspepsia, with flatulent distension after food; nausea; water-brash; and vomiting, especially in the morning, when it is often excited by the attempt to clean the teeth. When the urine is scanty, the vomited matters often have a fetid ammoniacal odour. In the advanced stages, the vomiting may be almost incessant and quite irrepressible. *Diarrhoea*, too, is not an unfrequent symptom. It may be excited by ill-digested food, or by the vicarious excretion of urinary products; not unfrequently, perhaps, by both these influences combined. In the advanced stages, the skin is usually unperspiring, dry, and harsh. *Hypertrophy of the heart* is less frequently associated with this form of disease than with the chronic

desquamative disease. The more advanced the stage of disease, the more frequent is the occurrence of hypertrophy. Dr. Grainger Stewart found this condition in only 12 per cent. of the cases fatal in the first stage, in 38 per cent. of those fatal in the second stage, and in 100 per cent. of those fatal in the third stage—that is, the stage of contraction (*A Practical Treatise on Bright's Diseases*, 2nd edition, p. 90). The probable explanation of the comparative infrequency of hypertrophy of the heart in the earlier stages is, that the watery condition of blood which results from a defective secretion of urine excites less resistance in the minute systemic arteries than the more concentrated blood-poison which results from the retention of urinary solids, while the water freely filters off through the small red kidney. For the same reason, too, *cerebral symptoms of uræmic origin* and *cerebral hæmorrhage* are less frequently associated with the large white kidney than with the contracted granular kidney. It appears that, while dropsy results from hydræmia, arterial resistance, hypertrophy of the heart, toxæmic nervous symptoms, and cerebral hæmorrhage, are more direct results of uræmia.

The two forms of *defect of vision* which I described as of frequent occurrence in connexion with the small red kidney, are less frequently associated with the disease which we are now discussing. The difference is one of degree. I have seen several cases of uræmic amaurosis and albuminuric retinitis with an unquestionable history, though not with *post mortem* evidence, of an enlarged, white, and fatty kidney.

Although cerebral hæmorrhage is a less frequent result of this form of disease than of the chronic desquamative disease, *hæmorrhage from mucous membranes*, and especially from that of the nose, is, in the advanced stages, a frequent and often a formidable symptom. These hæmorrhages are probably in part explained by the blood-deterioration, more especially the deficiency of albumen and hæmoglobin, and in part by the malnutrition and consequent brittleness of the walls of the vessels in the advanced stages of the disease.

Diagnosis.—On the subject of diagnosis, I need not add much to what I have already said. A careful consideration of the general history and the symptoms, together with the characters of the urine, will rarely leave you in doubt as to the form and stage of the disease. Renal disease dating from an attack of acute general dropsy, followed by persistent albuminuria, can rarely be other than the particular form of disease which we are now discussing. The stage of the disease is to be determined chiefly by the character of the urine. In the first stage, the urine is usually normal in quantity, in colour, transparency, and specific gravity. There is either no deposit, or a cloudy sediment containing some small hyaline casts. As the disease advances, the urine gradually loses its sherry tint, and becomes lighter coloured. The amount secreted and the specific gravity usually bear an inverse relation to each other. In the second stage, the small hyaline and oily casts are found; and in the third stage the oily casts are mingled with or replaced by the large granular and large hyaline casts. (Figs. 25, 26, and 27.)

Prognosis.—Your judgment as to the probable result of the malady will obviously be greatly influenced by the opinion which you may form as to the stage of the disease and the rate at which it is making progress. In my lecture on acute Bright's disease, I told you that I have seen cases of complete recovery after oily casts and cells had appeared in the urine continuously for many weeks, and after albuminuria had existed for one, two, and even three years.

The most favourable condition of urine is that in which it retains its normal colour, deposits no sediment on standing, and contains but little albumen. There can be no question that recovery may take place after the disease has passed into the second stage—namely, that of fatty degeneration. You are not, therefore, to despair of a patient whose urine contains oily casts even in large numbers. As a general rule, the longer the continuance of albuminuria in spite of careful treatment, and the greater the amount of albumen, the more unfavourable is the prognosis. In estimating the amount of albumen, never omit to compare the urine after food and exercise with that passed after rest and fasting. When the urine is very pale, and of low specific gravity, yet highly albuminous, when it deposits a copious and dense sediment composed in great part of large granular and large hyaline casts, it will be evident that the disease is in the third stage, and that the kidney is contracting. The number of the large sized casts may be taken as an index of the rate at which the degenerative and atrophic changes are progressing. The duration of the disease, in cases which ultimately prove fatal, varies extremely. I have notes of the case of a child aged 7, who had acute renal disease with dropsy after scarlet fever; and, the malady having terminated fatally within five weeks from its onset, the kidneys were already in the second stage, the pale and enlarged cortex being scattered over with the characteristic yellow spots of fatty degeneration. In this case, the disease ran

an unusually rapid course. I have seen a considerable number of cases in which the symptoms have continued for from five to ten years. My experience does not accord with the statements of some recent writers, who affirm that this disease is of shorter duration than that which results in the red granular kidney. I have before referred to one case which extended over a period of ten years; but the most prolonged case that I have seen or heard of was that of a medical practitioner whose history is partly given in my book on the *Kidney* (p. 374). He had dropsy after scarlet fever in 1836, when he was about seventeen years of age. He recovered from the dropsy, and thought no more of his malady until five years afterwards, when his urine was accidentally discovered to be albuminous by a fellow medical student. It had probably been so since the attack of dropsy; and it certainly remained albuminous from that time until his death, which resulted from dropsy in May 1866. He was then in his forty-seventh year; and, if we assume, as we safely may, that the urine had not ceased to be albuminous between the attack of dropsy and the accidental discovery of the albumen five years later, this gentleman had albuminuria for thirty years before his death; yet the greater part of that time he was a hard-working general practitioner, and, to all outward appearance, in good health. During the last year or two of his life, his urine was saccharine as well as albuminous. When I first saw him in 1851, his urine was of normal colour and specific gravity, but albuminous; it deposited no sediment, and contained no tube-casts. I believe that for many years it retained the same characters, but I have no note of any subsequent microscopical examination. This case teaches two practical lessons. The first is, not to take for granted that a patient who has recovered from acute renal dropsy is well until his urine has lost all trace of albumen; and the second is, not to assume that persistent albuminuria of necessity involves early death or the speedy occurrence of formidable symptoms.

THE SIMPLE FAT KIDNEY OR GENERAL FATTY INFILTRATION OF THE KIDNEY.—Before passing on to the subject of lardaceous degeneration of the kidney, I wish to direct your attention for a moment to a condition of the kidney which may be designated “the simple fat kidney” or “general fatty infiltration of the kidney”.

There is a form of fat kidney very different from that which I have described as the granular fat kidney. It consists in an uniform infiltration of the epithelium of the convoluted tubes with oil. This state of kidney is analogous to the fatty infiltration of the cells of the liver which occurs often in cases of phthisis and other wasting diseases. This form of fat kidney I first discovered and described in the year 1846 (*Med.-Chir. Trans.*, vol. xxix). The fatty infiltration of the renal epithelium may be found in various grades. When the fat is very abundant, the kidney is increased in size and weight. The colour of the cortex is either uniformly pale, or more frequently mottled by a blending of pale anæmic with red vascular patches. Occasionally, hæmorrhagic spots are scattered through the cortical substance. The medullary cones retain their normal colour and vascularity. The consistency of the kidney is usually softer than natural, and frequently the gland has an edematous feel and appearance. On a microscopic examination, the convoluted tubes are found to be universally distended with oil which has accumulated in their epithelial cells. There is an uniform oily infiltration of the renal gland-cells. (See Fig. 29.) This

Fig. 29.—A portion of a Convoluted Tube distended with Oil, from a “simple fat Kidney.” Three detached Epithelial Cells: two are filled and distended with oil, the third contains oil in less quantity, and the Cell-nucleus is visible.— $\times 200$.

condition of the kidney is found not unfrequently associated with a similar condition of liver in persons who have an excess of adipose tissue beneath the skin, in the abdomen and about the heart. It is commonly found after death from diabetes and from some other chronic diseases which are attended with great emaciation, such as cancer, phthisis, and dysentery. It is probable that the immediate cause of this fatty infiltration of the gland-cells is an excess of fatty matter in the blood. In the case of very fat persons, who are usually

large consumers of fat-making adipose and amyloid food, the materials whence the fat is derived are introduced directly into the blood through the stomach. On the other hand, in cases of wasting disease, it is probable that the fat absorbed from the adipose tissues enters the circulation and infiltrates the gland-cells, more commonly those of the liver, less frequently those of the kidney.

Fat kidneys are common in the domestic dog and cat, probably because these animals lead indolent lives, and consume large quantities of food rich in hydrocarbon. These animals are the counterparts of the human animal when, from eating and drinking to excess, he grows fat and gets fat liver, heart, and kidney. The unhappy Strasbourg geese afford an illustration of fatty infiltration of the liver resulting from a wasting disease. In order to obtain fat livers for patties, the animals are well fed and fattened; then they are confined in heated cages without food and water. They become feverish, and rapidly waste, while their livers grow large. It seems probable, as Baron Larrey long ago suggested, that the oil absorbed from the adipose tissue enters the circulation and infiltrates the cells of the liver, and probably in a less degree those of the kidney also.

It is a remarkable fact, that the liver and kidneys have been found in a state of extreme fatty infiltration in cases of poisoning by phosphorus, death occurring within a week after the poison was taken. Two cases of this kind are recorded in the fiftieth volume of the *Medico-Chirurgical Transactions*—one by Dr. Habershon, the other by the late Dr. Hillier. Fatty infiltration of the liver and kidney appears, as a rule, to have but little influence on the functions of these glands. We sometimes, however, find jaundice and ascites associated with fatty liver, and with no other structural change to explain the symptoms; and I have notes of several cases in which albuminuria and the usual symptoms of chronic Bright's disease have occurred in connexion with simple fatty infiltration of the convoluted tubes of the kidney. One such case I published in my book on *Diseases of the Kidney* (case of Ann White, p. 414). In that case, the disease followed a second attack of scarlet fever with anasarca. There was dropsy, and the urine contained much albumen and oily cells. She died with uræmic convulsions and coma. The convoluted tubes were greatly and almost uniformly gorged with oil; and Dr. Beale, analysing the cortex of the kidney, found that more than one-fourth of the solid matter was fat. It is reasonable to suppose that so large an accumulation of oil within the gland-cells must impair their secreting power, and also impede the circulation through the intertubular capillaries, which are compressed by the distended and swollen tubes.

I will now briefly recapitulate the chief points of distinction between the “granular fat kidney” and what I have here called the “simple fat kidney”, which is perhaps a better term than “the mottled fat kidney”, which I formerly employed, but which, I am told, has often been misunderstood. In the granular fat kidney, there are disseminated spots of fatty degeneration in the cortex; and these are secondary results of previous structural changes in the gland. In the simple fat kidney, on the contrary, there is a general fatty infiltration of the gland-cells in the convoluted tubes of the cortex; and this is a primary change. The granular fat kidney is always associated with albuminuria, and often with other signs of serious disturbance of function. The fatty infiltration, although it is sometimes associated with albuminuria and other symptoms of renal disease, is, in the majority of cases, unattended by obvious signs of functional derangement. This condition of kidney, therefore, while it has great interest for the pathologist, has much less clinical importance than the “granular fat kidney”; but I cannot assent to the statement that, because overfed animals leading unnaturally indolent lives have an excess of oil in their kidneys, this condition is normal or innocuous. By parity of reasoning, it might be maintained that an excessive growth of fat about the heart is a harmless addition of hydrocarbon to the weight of the body, because that state of heart often coexists for a time with apparently good health and great bodily activity.

PUNCTURE OF THE BLADDER ABOVE THE PUBES.—Dr. J. I. Little records in the *New York Medical Journal* for November 1872 a case of retention of urine from enlarged prostate, in which he punctured the bladder fourteen times with a capillary aspirator. Subsequently, a catheter could be introduced by the urethra. No disturbance, local or constitutional, followed the operation. Dr. Little recommends that, in performing the operation, the patient be placed on his back, with the hips slightly elevated by pillows if the bladder be not much distended; and that the punctures be made on or near the median line, about an inch or an inch and a half above the pubes, and each time in a separate place. The bladder, he says, may be washed out by filling the cylinder with water, and reversing the action of the instrument, without removing the trocar.