A LECTURE ON DIAGNOSIS AND PROGNOSIS IN CASES OF BRIGHT'S DISEASE.*

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CHRONIC Bright's disease, with the anatomical character of a large white smooth kidney, has in many respects a different clinical history from that which we have just now been considering. Like the small red kidney, it is sometimes the result of an insidious chronic degeneration; but, unlike it, the large white kidney is often a sequel of an acute inflammatory attack ("acute desquamative nephritis"), and we are thus enabled in some cases to determine the exact period at which the disease began. We may learn that, some months or even years ago, the patient had acute Bright's disease and dropsy; that the dropsy disappeared, but the urine never ceased to be albuminous. The history of these chronic cases sometimes extends over a period of many years. One case that came under my observation terminated fatally after albuminuria had continued for more than twenty years, one at the end of eleven years, and one after an illness of ten years. These were all cases of enlarged white kidney. Some writers have stated that the cases of chronic Bright's disease which have the slowest and the longest course are those associated with the small red granular kidney; but this statement does not accord with my experience. I have met with no cases of small granular kidney in which the disease has been known to exist for so long a period as in the cases of the large white kidney to which I have just now referred. It must, however, be borne in mind that the course of the disease which results in the small granular kidney is often so insidious, that it may have existed long before it was recognised or suspected.

In some cases, we may trace back the probable commencement of a chronic renal degeneration to a period when the patient began to be troubled to rise once or oftener in the course of the night to pass urine. This increased frequency of micturition may be a result either of a more abundant secretion of urine, or of some abnormal and irritating quality of the secretion acting as an irritant upon the bladder. With a history pointing to the existence of a large white kidney, the condition of the urine may vary considerably. The most favourable appearances are, a natural sherry colour, with normal specific gravity, a moderate amount of albumen, absence of all sediment and tube-casts, or a light cloudy deposit containing small hyaline casts; the amount of urine secreted being about normal. As the structural changes in the kidney increase, the urine loses its natural sherry colour, and at length it may become almost as colourless as water. The measure of urine secreted

Fig. 7.—Oily Casts and Cells. X200.

varies considerably, being sometimes much in excess of the normal amount, more frequently below it. The dropsical symptoms, as a rule, bear an inverse relation to the amount of urine secreted, and a direct

relation to the drain of albumen from the blood. The dropsical tendency, too, is favoured by the dry and inactive state of the skin which is commonly associated with chronic renal disease.

The gland-cells in the advanced stages may undergo a partial oily transformation, or they may be replaced by unorganised fibrine, which fills the tubes. Both these changes are indicated by the character of the tube-casts: the oily transformation by oily casts and cells (see Fig. 7); the fibrinous exudation by large hyaline casts having a diameter equal to that of the tubes in which they are moulded. (See Fig. 3, a.) When urine of pale colour and highly albuminous deposits a copious and rather dense whitish sediment, mainly composed of large hyaline and large granular casts, we may be sure that the glandular tissues are becoming rapidly disorganised, and that uræmic symptoms will speedily occur. I know of no appearances in the urine which are of more unfavourable import than those which I have just now described. The microscopic appearances to which I refer sometimes present themselves in the more advanced stage of a case in which, at an earlier period, oily casts and cells have formed the chief microscopic sediment. This sequence of events occurred in a case which I watched from its commencement to its termination, during a period of ten years. The case was one in which a large white granular fat kidney subsequently underwent a process of atrophy and contraction, the progress of the disease in its different stages being clearly indicated by the microscopic appearances in the urine. (See Figs. 8, 9, and 10.) The preserved specimens from which these drawings were taken I have in my possession, and they still retain their characteristic appearances. The disease began as an attack of acute desquamative nephritis. This passed into a chronic form of disease, with the appearance of oily casts and cells. After a period of nine years, these were associated with, and afterwards replaced by, large granular and large hyaline or waxy casts. The kidneys were much reduced in size, their combined weight being only seven ounces and three-quarters; but their appearance was very different from that of a small red granular kidney. Their cortical substance was pale, and presented numerous yellow oily granulations, showing clearly that in this case contraction of the kidney had followed upon enlargement, with fatty degeneration of the glandular tissues.

I have before stated that the urine in cases of acute Bright's disease is usually more or less tinged by admixture with blood, which probably escapes from the ruptured Malpighian capillaries. During the progress of the various forms of chronic disease, the walls of the minute arteries become hypertrophied, and those of the Malpighian capillaries gradually become thickened. Consequently, the capillaries are less liable to rupture, and the urine is rarely blood-tinged. When the urine is highly albuminous, of low specific gravity, and not only free from blood-tinge, but pale, from a deficiency of its normal colouring matters, there is reason to suspect the existence of advanced chronic renal disease. On the other hand, the appearance of dark coloured, smoky, or blood-tinged urine is, *pro tanto*, evidence of a recent acute affection of the kidney. I must warn you, however, to be on your guard against an occasional source of fallacy. It is this: in the advanced stage of chronic Bright's disease, the blood becomes much deteriorated, partly by the loss of its normal constituents, and partly by the retention of urinary excreta. There is, consequently, a tendency to hæmorrhage from various mucous surfaces—from the nose, the lungs, the stomach and intestines, from the uterus, and sometimes from the bladder. The hæmorrhage from the bladder gives the urine the blood-tinged appearance which it often has in cases of acute Bright's disease, when blood escapes from the substance of the kidney. You may come to a right judgment in these cases by a careful consideration of the past history, together with a close inspection of the urine. You will probably find that there are no blood-casts of the tubes, as there are when the substance of the kidney is the source of the bleeding. You may find some of those forms of tube-cast which point to the existence of chronic rather than recent acute disease: for instance, oily casts (Fig. 7), or large granular and large hyaline casts (Fig. 6). You may also find that the urine, when, after standing for a time, it has deposited the blood, presents the pale colour which is indicative of chronic disease in an advanced stage. When a doubt exists as to the renal disease being of recent origin or of long standing, the evidence of hypertrophy of the left ventricle of the heart, without valvular disease, but with a full and firm radial pulse, the result of excessive resistance to the passage of the blood through the minute systemic arteries, whose muscular walls are also hypertrophied—this evidence points to the existence of chronic renal disease. (Upon this point, see a paper by the author, BRITISH MEDICAL JOURNAL, April 16th, 1870.)

In cases of the large white kidney, the mode of death is usually different from that which is of most frequent occurrence in cases of the small red granular kidney. Uræmic convulsions, coma, and typhoid symptoms, are less frequent; while, on the other hand, an excessive

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dropsical accumulation, causing great distension of the skin, often results in fatal inflammation and sloughing of the integuments. In other cases, a dropsical accumulation within the chest, or engorgement and oedema of the lungs, may cause death by apnoea. In all forms of chronic Bright's disease, gastro-intestinal symptoms, vomiting, and

their walls. This serous transudation, mingling with the urine, renders it albuminous. Sometimes the distended capillaries have their walls ruptured, and blood escapes into the tubes and tinges the urine. It is interesting to note the action and reaction of renal and cardiac disease upon each other. In one case I observed the following se-

Fig. 8.—Period of Fatty Enlargement of the Kidney.

Fig. 9.—Commencing Atrophy and Contraction.

Tube-casts at three successive periods of the same case.

Fig. 10.—Advanced Atrophy and Contraction. *aaaa*. Oily Casts and Cells.

bbbb. Granular Casts. *cccc*. Large Hyaline or Waxy Casts. X 200.

purging, the result of the vicarious elimination of urinary excreta, may induce a fatal exhaustion.

Some recent writers affirm that impairment of vision, the result of degeneration of the retina, with or without hæmorrhage into its tissue, is almost exclusively associated with the small granular kidney. It so happens that most of the cases of retinal degeneration that I have seen have been associated with the large white kidney.

In the advanced stages of valvular disease or degeneration of the muscular walls of the heart, and in cases of emphysema of the lungs, with bronchitis and consequent retrograde engorgement of the systemic venous system, passive congestion of the kidney frequently causes not only dropsy, but also albuminuria and even hæmaturia; the urine containing small hyaline and blood casts. The secondary character of the renal complication is usually apparent from the history of these cases; and the diagnosis may sometimes be confirmed by the fact that, when the circulation has been relieved by rest in bed, by hydragogues and other suitable remedies, while the physical signs of the cardiac disease remain, the albumen disappears from the urine, to return, perhaps, when the circulation again becomes more embarrassed within the chest. I have seen this happen again and again during the progress of the same case.

The mechanism of albuminuria consequent on the passive engorgement of the kidney which results from an impeded return of blood through the veins, may be illustrated by reference to Fig. 11.

When, in consequence of an obstruction at the heart, the systemic veins become overfull, the distension of the renal vein, acting backwards through the intertubular capillaries, causes engorgement of the Malpighian capillaries, and a consequent transudation of serum through

quence of events. A gentleman had acute Bright's disease with anasarca, from which, after many months, he completely recovered.

Fig. 11.—Plan of the Minute Structure of the Kidney. *a*. Artery, sending an afferent branch, *af*, which breaks up into *m*, the Malpighian capillaries. *ef*. Efferent vessel which conveys the blood from the Malpighian capillaries into *v*, the plexus of capillaries between the tubes. These again unite, and form the vein *v*. *z*. The uriniferous tube. *c*. The capsule of the Malpighian body. The course of the circulation is indicated by the arrows.

During the progress of the disease there was developed a systolic bellows-sound over the base of the heart—the result, probably, of uræmic endocarditis. The bellows-sound remained after all symptoms of renal disease had passed away; and in time it became a double basic murmur, with the usual physical signs of incompetent aortic valves. For some years the valvular disease caused but little functional disturbance; but at length, with increasing obstruction of the circulation, dropsy came on, and with it albuminuria from passive congestion of the kidney. Death occurred in about ten years from the commencement of the renal disease. In this case it will be seen that a primary renal disease was the exciting cause of valvular disease of the heart; and that, the renal disease having passed away, the cardiac disease ultimately caused dropsy and a secondary albuminuria, the result of obstructed circulation and passive congestion of the kidney.

ON MUSCULAR ATROPHY (MALADIE DE CRUVEILHIER).*

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CASE II.—The next case which I propose to detail is that of a lady about twenty-five years of age, in whom the disease commenced about three years and a half since. In the year 1867, she was brought to my house by her brother, a young and well-informed physician, who had closely watched the case, and kindly furnished me with accurate notes. Some time before I saw this lady, she had consulted my friend and colleague Professor McDowel.

In the month of May 1866, this lady perceived that her left hand, particularly the thumb, was so weak that she found it difficult to strike the keys of the pianoforte. On investigation, however, it was found that the hand had been for some time previously much weaker than the other. Up to this time, she had been in the possession of fair average health. Her habits were active; she was of a nervous temperament, highly intellectual, and of a cheerful disposition. The only point connected with the history of her family worthy of notice, is that her father was the subject of locomotor ataxy, which existed for six years, and that his death was caused by apoplexy.

On examining the left hand when the lady complained of weakness, the ball of the thumb was found to have lost its natural plumpness, and the thumb had lost the power of adduction, so that she could not touch the tip of the little finger with it. The treatment recommended by Dr. McDowel and myself was, the syrup of phosphate of iron with strychnia and quinine, with faradisation to the affected part. For nine months this treatment was steadily pursued without the disease being in the least influenced by it, the atrophy slowly but perceptibly progressing.

In the December of 1867, six months after the left hand was first observed to be affected, the same phenomena presented themselves in the right hand; and the order was precisely the same, the thumb being the first part assailed. At the end of one year from May 1867, the left hand and the right were almost powerless, both presenting the peculiar and characteristic claw-like appearance, the "main de griffe." The muscles of the forearm were next invaded, the flexors more particularly, those of the left arm more rapidly changing than the right.

In fifteen months from the commencement of the disease, the lower extremities were attacked. At first, weakness and weariness were experienced on slight exertion, soon followed by inability to move. The atrophy of the muscles of the legs was not very marked; but they were soft and flabby. Finally, there was utter and complete inability to support the body. Two years from the first manifestation of the malady, it was evident that muscles which frequently escape the ravages of this most terrible disease, or are only affected at a very advanced period, were engaged. The pronunciation of labials and dentals became difficult; and, finally, the power of speech was nearly lost. Soon deglutition was impeded; soft solids could be got down better than fluids; but the attempt to swallow is always a struggle most painful to witness. The power of mastication is altogether lost. For the last five months no articulate sound has escaped her lips; her effort to speak is a mumbling occasionally intelligible to her sister, but conveying nothing to a stranger. There is a constant and profuse flow of saliva. The atrophy is now almost universal. The tongue lies motionless on the floor of the mouth. Still there are some muscles which have escaped the devastation of disease. The face does not present the appearance of emaciation. When last I saw this poor lady, I was

struck by the want of correspondence between the face and the other parts of the body: she was reading, and her eyes beamed with intelligence. When alone, I was informed that she managed to turn the leaves of her book with her chin. There has been, it should be observed, no diminution of sensibility, rather an exaltation. Nor is there any difficulty of maintaining the temperature of the extremities. At times, she complained of tenderness of the surface, and pain was felt on pressing the spinous processes of the third and fifth cervical, and the eleventh and twelfth dorsal vertebræ. From the earliest period, although the disease pursued its course, invading in succession the various parts indicated with unrelenting severity, there were short intervals when it seemed to stand still, affording delusive hopes of improvement, soon destined to disappointment. A more painful case can scarcely be conceived; and, in truth, no description can convey an adequate idea of the reality. About a fortnight having elapsed since I saw this lady, I visited her brother to-day to ascertain her present condition. He tells me there exists a painful state of hyperæsthesia, the slightest touch causing pain and spasm. The muscles of the neck are rapidly wasting. She has difficulty of supporting the head, and deglutition is more painfully difficult.

CASE III.—Mr. M., aged 25, a civil engineer, the son of strong and healthy parents, still living, with no hereditary predisposition to disease, was temperate and regular in his habits, vigorous and active, and of a high order of intellect. On obtaining an appointment, he proceeded to India, and arrived at Calcutta on the 25th September, 1866—to use his own words—"in perfect health." In a few days he went to Port Canning, a place notoriously unhealthy; so much so, that higher wages than ordinary are given as an inducement to the natives to reside there. The unhealthiness of the locality was proved to Mr. M. by the prevalence of disease among the people under him, although he learned that the season was exceptionally healthy. The place had been partially cleared of jungle, but it was undrained. The water was brackish, and this the natives used; for the use of the Europeans, water was brought from Calcutta. The surface of the ground was four feet below high water-mark of ordinary spring tides.

Notwithstanding the extreme insalubrity of Port Canning, Mr. M. continued to perform his duties, which obliged him to be out until midday, without observing any change from his ordinary good health until November 12th. He then had an attack, the symptoms of which, he says, were headache and sickness of stomach, with fever. The duration of the illness was four days. On going to Calcutta, he quite recovered in two days, and returned to Port Canning.

About the middle of December, he felt pains which he considered rheumatic in both arms. The pains continuing, he sought advice the following month (January 1867), finding, also, that there had been a gradual diminution of strength in the arms. He observed that the weakness commenced in the right hand.

On January 19th, he found it difficult to raise a full glass to his mouth. The weakness increasing, he consulted physicians in Calcutta; and, by their advice, he employed galvanism, a current being passed from his neck or shoulder through each arm for about a quarter of an hour each time; the arms and legs being also well rubbed by hand each day. He also was directed to take syrup of iron and strychnine. Writing soon became a matter of extreme difficulty; and March 11th was the last entry in his diary. Still he could sign his name; but the operation was performed slowly and laboriously—the pen being fixed between his fingers, and, if ink were required, the pen being removed for him. Gradually the arms became powerless.

On March 26th, a second consultation of physicians was held, and he was ordered to leave the tropics forthwith, an order which he disregarded. For some time he had found that he could only manage to feed himself by means of a spoon or fork, stooping his head near the plate; but on the evening of the consultation, he found it impossible by any means to feed himself. During all the time up to the end of March, he walked each day, taking a considerable amount of exercise, but always finding walking on uneven ground, or going up and down stairs, difficult. He was also unable to rise from his chair without assistance. At length, after falling repeatedly and being unable to get up without assistance, he gave up the attempt to walk alone. He had also been in the habit of riding; and this he continued as long as he could, being assisted on to the saddle; and his horse, which had previously the habit of bolting and other bad habits, became so gentle that he could ride him anywhere with the reins lying on his neck, being unable to hold them, at least with any effect.

Still he remained in charge of the municipality up to April 22nd (seven months), having completed a report of the works while he was in charge, and also a detailed statement of projected works for the following year; and so admirably had he done his work, that he received the thanks of the authorities. On May 7th, he left Port Canning; and,

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