ON THE
FORMS AND STAGES
OF
BRIGHT'S DISEASE OF THE KIDNEYS,
WITH ESPECIAL REFERENCE TO DIAGNOSIS AND PROGNOSIS,
(FOURTH COMMUNICATION.)

BY
GEORGE JOHNSON, M.D., F.R.C.P.,
PHYSICIAN TO KING'S COLLEGE HOSPITAL.

Received March 18th.—Read March 22d, 1859.

It will probably be conceded by most persons who have given any consideration to the subject, that the question of the relationship which exists between the various conditions of the kidney which are commonly included under the term Bright's disease, is one not only of speculative interest, but of practical importance.

That the appearances of the kidney in different cases of this, so-called Bright's disease, are very diverse, is sufficiently obvious. In one case the kidney is found large, vascular, and congested; in another large, pale, anæmic, and wax-like; in a third also large, pale, and anæmic, but

having numerous fine, yellow granulations like specks of sand scattered through its cortical substance. Again in other cases, the kidneys are more or less reduced in size, and their surface is uneven and granular.

Now there are some pathologists who maintain that, different as are the appearances presented by the kidney in these cases, they are nevertheless the result of a single morbid process. Bright's disease, according to these authors, is ever one and the same disease; and the various morbid appearances in the kidney, to which it gives rise, result simply from the greater or less degree of intensity of the disease, and the more or less advanced stage at which the renal degeneration has arrived before the fatal termination.

That this theory of the *oneness* of Bright's disease is untenable, and inconsistent with the clinical history and the morbid anatomy of the kidney in its various conditions has been, I think, abundantly proved by myself and others who have written on the subject.

Amongst the opponents of the theory of the *oneness* of Bright's disease I may particularly mention Dr. Todd¹ and Mr. Simon;² Dr. Wilks, too, has ably advocated the doctrine of a diversity of the forms of Bright's disease;³ and Dr. Richard Quain, in a paper published in the '*Lancet*'⁴ nearly fourteen years ago, argued for the recognition of three principal forms of the disease, namely, 1, the simple enlarged mottled kidney; 2, the truly granular or atrophied kidney; and 3, the large, flabby, fatty-looking kidney. Dr. Quain's remarks on this subject, published at a time when microscopic examinations of morbid kidneys were in their infancy, deserve more notice and credit than they have received.

The *chronic* forms of Bright's kidney, of which only it is my intention to speak in the present communication, may

¹ 'Clinical Lectures on certain Diseases of the Urinary Organs.'

² 'Med.-Chir. Transactions,' vol. xxx.

³ 'Guy's Hospital Reports,' 1852-3, p. 232.

⁴ 'Lancet,' November, 1854, p. 594.

conveniently be arranged in two main divisions; 1st, the large white kidney, whether simply pale, anæmic, and wax-like, or containing more or less oil; and 2d, the small, contracted, granular kidney.

The chief points of distinction between the large white Bright's kidney and the contracted kidney are these:

1st. The urine secreted by the large kidney is less abundant, of higher specific gravity, more constantly and copiously albuminous, and usually contains clear fibrinous wax-like casts, either with or without oil, but none of the granular casts which are thrown off from the tubes of the contracting kidney.

2d. The minute anatomy of the two kidneys is very different.

In that form of disease which leads to the contracted kidney, the gland-cells which line the uriniferous tubes undergo—even at the very commencement of the malady, and sometimes long before the urine contains a trace of albumen—a process of disintegration; as a result of which they become detached from the basement membrane, and appear as a sediment in the urine in the form of the granular tube-casts just now mentioned. The uriniferous tubes thus become entirely denuded, or they are found to be lined by a layer of small, transparent cells, entirely different from the normal glandular epithelium. It is probable that a tube thus denuded, has lost, in a great degree, its power of secreting the solids of the urine, while it may yet be able to separate the watery constituents from the blood. And hence, perhaps, the abundant flow of urine, pale in colour, and low in density, which characterises this form of disease.

In the case of the large white kidney, on the other hand, the gland-cells do not become disintegrated, detached and washed out, so as to leave the tubes denuded. They remain, for the most part, adherent to the basement membrane, and undergo changes, varying from a slight granular opacity to a complete oily degeneration, or they may at length become replaced by an albuminous or a fibrinous material, which

more or less fills and obstructs the tubes. This condition of the renal tubes appears to be less favorable for the transudation of water than the denuded condition before described.

Another very important distinction between the two forms of disease, is to be found in the relative frequency of *dropsy* as a symptom. There are very few exceptions to the rule, that patients dying with the large Bright's kidney have had dropsy, in a greater or less degree, at some period of their history. Whereas, the majority of those who die with a contracted kidney, have not suffered from dropsy in any form or degree. I have lately been engaged in analysing the main features of about 300 cases of Bright's disease which have come under my observation; and amongst other points for inquiry, I have endeavoured to ascertain the relative frequency of dropsy in cases of the large and the small Bright's kidney respectively. For this purpose it is necessary to exclude entirely from consideration all that numerous class of cases in which valvular disease of the heart or other acknowledged and sufficient cause of dropsy coexists with the renal disease.

I find, then, that out of twenty-six fatal cases of enlarged Bright's kidney, there was dropsy in twenty-four; the proportion being, in round numbers, 92 per cent. And in many of the cases the dropsy was great, general, and of long duration.

Whereas, of thirty-three fatal cases of contracted kidney, there had been dropsy in only fourteen, the proportion being 42 per cent.; and in most of these cases the dropsy was very slight and partial.

Surely these facts are fatal to the hypothesis, which assumes that a contracted kidney is, in every case, but an advanced stage of the same form of disease as that which gives rise to the large white kidney. If all the contracted Bright's kidneys have passed through a previous stage of enlargement, it is difficult to understand how it can happen that the majority of those patients who have reached this final stage of renal degeneration, should escape the dropsy,

which, in a greater or less degree, troubles nearly all those who die in what is assumed to be an earlier stage of the same disease.

It is, I think, sufficiently obvious that there is an intimate relation, on the one hand, between the comparatively scanty secretion of highly albuminous urine by the large Bright's kidney, and the frequent occurrence of dropsy in these cases; and, on the other hand, between the copious secretion of watery urine by the small kidney, and the comparative infrequency of dropsy as a symptom. The risk of dropsy being, *cæteris paribus*, directly as the drain of albumen from the blood, and inversely as the secretion of water by the kidney. In other words, the more scanty the secretion of urine, and the greater the amount of albumen which it contains, the greater is the probability that the patient will become dropsical.

Now, I maintain, as I have done for years past, that the small Bright's kidney, through which there usually flows a large daily measure of watery urine, is the result of a different series of morbid changes from that which gives rise to the large white kidney. The rule is that a large Bright's kidney remains large to the end, and does not become a small one; and, on the other hand, a contracted Bright's kidney does not pass through a previous stage of enlargement.

Having thus stated what I believe to be the rule with regard to this subject, I come to the chief object of this communication, which is to point out some exceptions to this rule, exceptions which, when carefully considered and rightly interpreted, tend, as I think, to prove and confirm the rule.

There are undoubtedly cases of Bright's disease in which the kidneys having become enlarged, subsequently undergo a process of contraction, in a greater or less degree. These cases, however, are so exceptional in many of their most important features, that they afford a remarkable confirmation of the doctrine that in ordinary cases the contracted Bright's kidney is not an advanced stage of a previously

enlarged kidney, but rather that it is the result of a distinct series of morbid changes.

The cases of Bright's disease in which contraction of the kidney has followed upon enlargement may, for convenience of description, be divided into three classes.

First, there are cases in which the size and weight of the kidney are found after death to be considerably increased, yet in the cortical portion of the gland there are unquestionable indications of commencing atrophy and contraction. The cortex of the kidney in these cases is anæmic, pale, and wax-like, the surface being more or less uneven and nodulated, while the thickness of the cortical substance appears diminished in a greater or less degree.

I have notes of six cases in which the kidney was found thus enlarged yet partially atrophied. The surface and section of the kidney in one of these cases (that of a man named Revels) is represented in a plate from an original drawing by Dr. Westmacott. I have no note of the weight of the kidney in that case, but in the other five cases it was as follows: In one the two kidneys weighed thirteen ounces. In one, a boy aged fifteen, their combined weight was ten ounces, and in another man they weighed together twelve and a quarter ounces. In one the weight of a single kidney was seven ounces, and in another eight ounces. Now, as in each of these cases, there was more or less atrophy of the cortical portion of the kidney, we have so many unquestionable examples of contraction following upon enlargement of the gland.

In a second class of cases this contraction of a white and waxy kidney had proceeded further; so that the size and weight were reduced below the average of the healthy organ.

I find three cases of this kind in my note-books. In one case a single kidney weighed four ounces, in another the weight of the two kidneys was eight and three quarter ounces, in the third case the weight of the kidney was not noted, but the gland was of small size, and its surface coarsely granular.

The third class of cases are those in which the kidney, having become enlarged and undergone *fatty degeneration*, has subsequently contracted, the fat granulations being still visible in the atrophied gland.

I have notes of five cases of this kind, the weight of the two kidneys varying from seven ounces and three quarters to six ounces and a half.

The second and third of these classes of cases call for some additional remarks, and I will speak first of the atrophied granular fat kidney.

That a fat kidney is generally of large size will scarcely be denied by any one, and that the fatty disease has no great tendency to produce atrophy and contraction of the gland is conclusively shown by the fact that the fat kidney is usually found to be of large size after the disease is known to have existed for many months, and even for several years.

During the last fourteen years I have met with only five cases in which a *small* fat kidney was found on post-mortem examination. And during the same period I have seen only three cases besides, in which I inferred from the character of the urine, and especially from its microscopic appearances, that a fat kidney was becoming atrophied.

The diagnosis of these cases during life is sufficiently certain and easy. In two of the five cases above mentioned the commencement and the progress of the atrophic change in the were clearly ascertained by repeated examinations of the urine; and the casts and cells preserved as microscopic specimens at different stages of each of these cases retain their characteristic appearances at the present time; so that the most important signs marking the progress of a disease, which ended fatally more than two years ago, are still open to the inspection of the microscopic observer.

The following is an outline of the history of one of these cases :

Stephen G—¹ was first admitted into King's College Hospital in October, 1846; his age being then twenty-three. He was suffering from acute general dropsy with albuminous and bloody urine. The dropsy disappeared during his stay in the hospital, but the urine continued to be albuminous, and it deposited tube-casts and cells containing oil. In short, there was evidence that the kidney was undergoing fatty degeneration.

I saw him occasionally for several months after he left the hospital. His general health continued good, and he had no return of the dropsy, but the morbid characters of the urine remained unchanged. I then lost sight of him for several years, in fact until October, 1855—when he again came under my care at the hospital. He then stated that for the previous six years he had been serving as steward on board an Antwerp steamer, and had enjoyed excellent health until January of that year (1855), when he was suddenly seized with hemiplegia, from which he had but partially recovered; the leg being still very weak, and the arm nearly powerless. He thought that his paralytic attack might have been induced by his having for some time before taken daily a large quantity of Scotch whisky. Remembering his previous history, I was now much interested in ascertaining the condition of his urine. We found that it was still highly albuminous, and that it still deposited oily casts and cells.

Here, then, was a case of fat kidney of nine years' duration. But during the next few months, while the patient still remained under my observation, the deposit in the urine underwent a marked change. The oily casts and cells gradually diminished in number, and were replaced by the large granular and large waxy casts. I now felt sure that the secreting cells of the kidney were being rapidly destroyed, and that a process of atrophy had commenced; and I frequently spoke of the case as one in

¹ The early history of this case is fully given in the author's work on the 'Diseases of the Kidney,' p. 411.

which the somewhat rare transition from a large fat to a contracting kidney was in progress.

FIG. 1.

FIG. 2.

FIG. 3.

Sediment in the urine at three successive periods of the case of Stephen Grey, $\times 200$.

- Fig. 1. Period of fatty enlargement.
 „ 2. Commencing atrophy and contraction.
 „ 3. Advanced atrophy and contraction.
a a a a. Oily casts.
b b b b. Granular casts.
c c c c. Large waxy casts.

The patient died in the hospital from uræmia, without any return of dropsy, in October, 1857, exactly ten years from the time of his first admission with acute renal disease.

The kidneys, the appearance of which is represented in Plate III, from Dr. Westmacott's drawing, were found much reduced in size, their combined weight being only seven ounces and three quarters; and the yellow fat granulations, which were still plainly visible, showed that contraction of the kidney had followed upon fatty degeneration of the glandular tissues.

The appearance presented by a contracted fat kidney, with its characteristic small yellow granulations, is so remarkably different from that of a small kidney which has been the subject of the chronic desquamative disease, that it is scarcely possible to mistake the one for the other, or, as I think, to doubt that they have resulted from a different series of morbid changes.

It is, however, not so easy to identify the second series of cases before referred to; namely, those in which a wax-like, but not a fat kidney, having once been enlarged, contracts until its size is less than that of the healthy organ. Yet it is not difficult to distinguish even these from the kidneys whose atrophy, having resulted from the crumbling and desquamation of their gland-cells, has not been preceded by a stage of enlargement.

The chief points of distinction between these two classes of cases are the following. A kidney which has been *enlarged* by chronic Bright's disease is always more or less anæmic, pale, and wax-like in its cortical portion; and this appearance of anæmia does not pass away if the kidney should afterwards contract. On the other hand, the small kidney which results from the chronic desquamative process is often found to be comparatively red and vascular, even in its extreme stage of atrophy and contraction.

The different appearances presented by the kidneys in these two classes of cases will be found illustrated in Plates IV and V.

The contracted waxy kidney is usually much firmer and harder than the other, and its surface is more uneven and nodulated, or coarsely granular. Again, on microscopical examination, the appearance of denuded tubes, which results from the destruction and removal of the gland-cells, is much less frequent in cases of contracted waxy kidney than in cases of the chronic desquamative disease. Lastly, it is probable that a waxy kidney which has once been much enlarged never contracts to the small size often attained by the essentially atrophic form of Bright's disease. Only three out of the nine contracted waxy kidneys which I have met with were reduced below the normal size, and the smallest of these weighed four ounces; whereas, kidneys which have been wasted by the chronic desquamative disease are not uncommonly reduced to half that weight; and in the last volume of the 'Transactions of the Pathological Society of London' (vol. ix, p. 308), Dr. Wilks has briefly reported the case of a woman whose kidneys were so extremely degenerated, that "the two together weighed less than one ounce and a half."

I think, then, that a careful observer would scarcely fail to distinguish between these two forms of disease, from an examination of the kidneys alone, and without any previous knowledge of the patient's history. But the distinction will be easier if the progress of the disease has been observed from the beginning. The early history of these cases of contracting waxy kidney is that of a chronic enlarged Bright's kidney. In some instances the disease originates in an attack of acute dropsy with albuminuria; in other cases its origin is latent, but dropsy in some form or degree usually occurs sooner or later during the progress of the disease. The urine, moderate in density and quantity, is copiously albuminous, and it is either free from sediment, or perhaps it deposits a cloud, which is found to contain the small, clear, wax-like casts. At a later period the sediment in the urine is dense and abundant, being in great part composed of the large granular and waxy casts, the diameter of which is equal to that of the

uriniferous tubes. These appearances in the urine mark the stage of atrophy and contraction of the kidney. The destruction of the renal epithelium by disintegration, and the choking of the tubes by unorganized fibrin, appear to be constantly associated with atrophy of the glandular tissues; and the rate of progress of the degeneration may be accurately estimated by the amount of the before-mentioned dense sediment in the urine.

It is a well-known fact that patients who have constantly a large amount of albumen in the urine, may go on for many months, and even for years, without any serious impairment of their general health. In cases of this kind the entire absence of sediment, or the presence of a light cloudy deposit containing a few small waxy casts, without oil, is the most favorable condition of urine; while an increase in the amount and density of the urinary sediment, and an abundant appearance of the large-sized casts, are signs of unfavorable import, indicating, as they do, a rapid destruction and displacement of the renal gland-cells, which in all probability will be soon followed by the alarming symptoms of suppressed secretion and uræmic poisoning.

DESCRIPTION OF PLATES III, IV, AND V.

The illustrations are intended to represent the characteristic appearances of three different forms of contracted Bright's kidney.

Plate III.—The surface and section of the kidney in the case of Stephen G— (p. 160). The gland has been enlarged, and has undergone fatty degeneration; it has subsequently contracted to less than the natural size, the small, yellow, fat granulations being still visible in the anæmic and atrophied cortical substance.

Plate IV.—A large, waxy kidney in process of contraction. The entire gland is larger than natural, but the section shows the cortical substance much reduced in thickness, while the surface is uneven, and presents an irregular blending of wax-like anæmia with congestion. (pp. 161-2.)

Plate V.—An extremely contracted kidney, the result of the "chronic desquamative disease." The section shows that the cortical substance has nearly disappeared, yet the capsular surface of the kidney retains an appearance of redness and vascularity (pp. 161-2).