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Atrophy of the suprarenal capsules with enlarged spleen and dark-coloured urine.

By Robert Saundby, M.D.

On November 24th, 1879, I was asked to see W. H. E—, aged 16. He complained of cough and pain in the left side; he had been ill two days. Pulse 120; respirations 18; temperature 102°. Percussion showed about two fingers' breadth of diminished resonance at the left base posteriorly; respiratory sounds everywhere slightly harsh; inspiration prolonged. The heart's apex was in the fourth interspace, three inches and a quarter to the left of the middle line just inside the nipple; there was a systolic murmur at the base propagated into the neck and a loud *bruit de diable* in the jugulars. In the left hypochrondrium there was a large tumour which reached across to beyond the middle line of the abdomen; its inner border was rounded, tense, and presented a well-defined notch; there was much tenderness in this region. No enlargement of the liver was detected. The bowels had not been moved for three days. There was great difficulty in micturition, which could be effected only in the "knee-and-elbow" position, apparently on account of the enormous splenic tumour. The urine was porter-coloured and deposited brown flocculent matter which was composed of urates, oxalates, blood-casts and material resembling broken-down blood-corpuscles. It was acid, sp. gr. 1017, and contained a considerable quantity of albumen, but no sugar or bile. Dr. MacMunn, of Wolverhampton, examined it for me with the spectroscope, and reported that the colour was due to methæmoglobin and urobilin. The blood examined under the microscope showed only a very slight excess of leucocytes. His skin was a peculiar dusky yellow, but the conjunctivæ were quite white. His mother informed me that he had "always passed dark urine from his birth, that the enlargement of the spleen had been known to exist for some years, that his father died at the age of thirty-seven with the same symptoms, viz, enlargement of the spleen (which weighed 7½ lbs. on *post-mortem* examination), dusky skin and dark urine, and that the younger of two other living children, both girls, passed the same peculiar coloured urine, though she was otherwise well and strong."

W. H. E— got over that attack in a few days, and is still alive, presenting the same physical peculiarities, with the exception that the spleen is rather smaller when he is not suffering from one of his attacks, not passing beyond the middle line. In his ordinary condition the vertical splenic dulness in the axillary line is ten centimetres, and in the long axis of the tumour twenty-six centimetres.

I saw the younger sister, B. E—, for the first time during my visits to her brother. She appeared a bright active girl, with a decidedly dark complexion, but not more than corresponded to her hair and eyes. Early in 1882, I was asked to see this young lady for a trifling indisposition, of the details of which I have no record, and I then made the following note:—She presents no abnormal physical signs, especially no evidence of splenic enlargement; her complexion is slightly ochre-tinged, like her brother's; her urine is always dark. She is now sixteen and menstruated for a short time regularly, but this has stopped for the last six months. She appears in other respects a well-grown, well-developed, and intelligent girl. Her urine was dark amber, turbid, 1015, acid, no albumen; turbidity disappeared on heating; it gave a faint blue colour with guaiacum and ozonic ether.

A few months later in the same year I saw her again; she was then complaining of weakness, loss of appetite, superficial ulceration of the gums, and constipation followed by diarrhœa. Her urine was darker than usual, and at this time the spleen could be readily detected by palpation.

I subsequently arranged to have a specimen of the urine from each case sent to Dr. Mac Munn, who favoured me with the following reports:

"No. 1. B. E—. Urine deep yellow or brownish yellow colour with mucous cloud at bottom of bottle. Reaction acid, sp. gr. 1012, no albumen; no blue colour with guaiacum and ozonic ether; with nitric acid gives only red and violet. The colour is due to urobilin, indican, and it also yielded onucleolin (so called). It contained no blood-pigment and no bile-pigment.

"No. 2. W. H. E—. Urine the colour of dark tea, depositing a sediment of red brick urates; sp. gr. 1024, acid; on boiling a cloudiness which was only partially dispersed by acetic acid. On boiling with cupric sulphate and caustic potash a partial reduction took place, the solution becoming olive brown (glucoside—indican), with nitric

acid it gave red and violet. With guaiacum and ozonic ether a green colour, but no blue. Its colour was due to indigo-red (as such) indican (which could be split up into indigo-blue), urobilin, urerythrin, and, strange to say, a trace of a pigment which had a great likeness to cruentin, a pigment got from hæmoglobin and hæmatin by the action of sulphuric acid."

The urine from which these reports were taken was passed during intervals of ordinary health, not during attacks.

After this time (1882) B. E— was never strong. She became more decidedly anæmic, there was a systolic murmur over the base of the heart; menstruation was irregular; she had very little appetite; her digestion was feeble, and she appeared weak and languid.

During 1884 she consulted me several times. In August of that year I have the following notes:—"26th.—She came looking very ill. Has had some annoyance. Sick, no appetite; ordered to bed. 27th.—Systolic murmur over base of heart. Spleen enlarged (a sketch in margin shows that it came nearly to the umbilicus in the middle line and passed still lower in the left lumbar region). Was unwell late yesterday. Had seen nothing for two months previously."

On September 19th I noted that the spleen had receded so as only to be felt just below the ribs. She had several similar attacks, and her health continued to be very unsatisfactory. She was a good deal away the latter part of the year seeking benefit from rest and change. There was no increase noticeable in the colour of her skin, which remained much the same. I examined her blood more than once; there was never any increase of white corpuscles or marked diminution of the red, but these were pale.

On January 17th, 1885, she got over-fatigued and felt faint, and on the following day (Sunday) she was worse and fainted with loss of consciousness. On Monday she was sick, and complained of headache and faintness. On Tuesday I saw her; her temperature was 102°, pulse 120 or more, full and fairly firm. Tongue pale and clean. She complained of headache, pain at the epigastrium, and nausea. Bowels open. Spleen reached down to the umbilicus. Urine dark. She slept from 9 p.m. to midnight, and afterwards became very restless. I saw her on Wednesday at 9.30 a.m. Pulse very rapid and feeble. She could answer questions and was free from pain. In spite of all that could be done she never rallied

and became unconscious about 5 p.m., dying comatose at 6.30 the same evening.

The autopsy was made the next day (January 22nd), Mr. Eales being present with me. She was 18 years of age, and the body presented the appearance of a well-developed, well-nourished girl. There were no marks or patches of pigment, but the skin of the face and on the backs of the hands was darker than on the rest of the body. Rigor mortis was well marked; there was very little hypostatic congestion, and no œdema. The head was not examined.

Thorax.—The heart showed some excess of fat in the epicardium; there was a pale clot in the right side; the left ventricle was contracted and slightly hypertrophied; the valves were normal; the pericardial cavity was dry. The pleural cavities were dry; both lungs were crepitant and healthy; there was no tubercle.

Abdomen.—The spleen occupied the whole of the left hypochondrium; it weighed 66 oz.; it was dark purple in colour, hard, and tense. On section it looked like damson cheese. It gave no amyloid reaction. Under the microscope a large quantity of granular brown pigment was diffused through the pulp, but there was no other structural change. The liver was quite healthy in appearance. The stomach was dilated and its mucous membrane was covered with bile and mucus; it contained half a pint of altered bile. The kidneys were rather large, their cortices looked dark, and the medullary portions pale. Under the microscope pigment could be seen lying in the tubules. The suprarenal capsules were very small, the right weighing 18 grains and the left 10 grains. The semilunar ganglia appeared normal, the right weighing 5 grains and the left 6 grains. Under the microscope the capsules showed no trace of inflammation or fatty or fibroid degeneration; they *seemed to be simply atrophied.* The semilunar ganglia were healthy, no change being apparent in their nerve-cells, connective tissue, or blood-vessels.

To these facts I may add that both parents were English, and never lived out of England. The children were born at Ipswich, and lived afterwards at Gloucester. The mother is dark, and B. E— resembled her very much. She is subject to rheumatism, but otherwise healthy. The son is not at all like his mother. Careful inquiry has failed to show that any of them had ever been exposed to malarial influences.

During B. E—'s life I never made a diagnosis. The attack in which I first saw W. H. E — resembling in many features paroxysmal hæmoglobinuria, I reported it under that title (see 'Medical Times and Gazette,' May 1st, 1880), and I was at first disposed to regard B. E— as having a similar tendency; but except on one occasion I never found any blood-pigment in her urine. I regarded the cases as quite unique, unlike anything else described in medical literature. The result of the *post-mortem* examination somewhat modified that view by suggesting that they are examples of Addison's disease from atrophy of the suprarenal capsules.

Atrophy of the suprarenal capsules is quite new to me. The only specimen I have had an opportunity of examining microscopically, besides the present case, was that shown by Dr. Hadden at this Society, and a very striking difference is noticeable between the microscopical appearances in his case and in mine. In Dr. Hadden's case the atrophy is evidently secondary to a chronic inflammatory process with increase of connective tissue, but in mine there is no inflammatory change, the structure of the capsules being quite normal, so far as there is any.

This is important, for it has been strongly held, especially by Dr. Addison's successors in the great pathological school of Guy's Hospital, that Addison's disease is essentially related to a special form of chronic scrofulous or tuberculous inflammation of the capsules, and that other diseases of these organs, *e. g.* cancer, do not give rise to the significant group of symptoms. Dr. Hadden's case, and perhaps other atrophied specimens which have been observed, might be explained in accordance with this view by assuming them to be in a later stage when fibroid transformation and atrophy had succeeded to an inflammation originally typical in character, a sequence quite familiar to pathologists. But I doubt the accuracy of the narrow doctrine which denies the occurrence of the special group of symptoms apart from one form of anatomical change. I do not believe the cases recorded to the contrary can be disposed of so easily as the upholders of the doctrine of unity would have us believe, and we know that Addison himself did not share this exclusive opinion. There is good reason to believe that any destructive lesion of the capsules is capable of giving rise to pigmentation.

The recent observations of Dr. Mac Munn have tended to show that the capsules are glands concerned in the elimination of effete

blood-pigment, and his researches if followed up and confirmed will explain, perhaps the most striking of the phenomena of Addison's disease. But how shall we explain the fatal course of the disease? Why should mere accumulation of pigment give rise to anæmia, to gastric crises, and to death? I cannot accept the proposition upheld by Greenhow that the semilunar ganglia are the real seats of disease, as I have frequently seen them in a very advanced stage of the lesion he describes without any of the symptoms of Addison's disease being present, while in the case of B. E— they were quite normal. Viewed in connection with the state of the urine, especially the great increase of indican which has been previously observed by Rosenstirn, I am disposed to regard the general symptoms as the result of some poison in the blood, possibly a product of effete pigment. My case is so unique in some of its features that some may think it should not be grouped with Addison's disease. But it seems to me to be more scientific and more modest to place it in a group with which it has certain fundamental affinities, than to class it as something entirely independent.

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