CLINICAL DEPARTMENT

REPORT OF A CASE OF NONPARASITIC CHYLURIA, CHRONIC NEPHRITIS, IN A CHILD

WITH NECROPSY

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Nonparasitic chyluria is a rare affection in adults and exceedingly rare in children. While most of these cases are due to a lymphangiectasis and a consequent rupture of the lymphatics in the pelvis of the kidney or in the bladder, the etiology of this disease is nevertheless somewhat uncertain.

Jennie G. was admitted to the Beth Israel Hospital on Jan. 1, 1916, when eight years old. Her parents and their four other children were alive and well.

The child had been nursed until 1 year of age. She had suffered from a chronic cough from the eleventh month until the end of the second year, when she had a severe attack of measles. During the fourth year it had been noticed that the child's abdomen became enormously enlarged and that she steadily lost weight. The parents consulted a number of physicians, including the writer. At that time, having found ascites in the puny, undersized child, with a history of cough of long standing, normal urine and no abdominal tumor, I suspected tuberculous peritonitis, although the temperature was normal and the von Pirquet test was negative. The parents would not permit tapping of the abdomen. On prolonged use of tonics with creosote, together with good nourishment, the child gained weight, and in the course of eighteen months the ascites almost disappeared.

In the month of May, 1914, the child had been brought to me, and the mother had told me that for the previous two months the patient had again been losing weight, had had frequent micturition and the urine had been milky in appearance. She was very anemic, weighed only 28 pounds and some dullness could be detected at the lower part of the abdomen. The urine looked turbid, and fibrinous coagula were observed when it had stood for some time. By shaking the urine with ether, the dissolved fat rose to the surface and the urine became quite clear. Microscopically, the fat globules were in a fine state of subdivision. Sudan III stained the fat particles red. No filaria and no blood had been detected. By advising complete rest, involving the absence from school, and the administration of salol, urotropin and belladonna, there was slow improvement, some weight was gained and on July 31, 1915, the urine was examined by Dr. Max Kahn who reported that it contained a huge amount of mucin, but no lactose, fat or casein.

On Nov. 27, 1915, the frequent micturition and milky urine reappeared. Dr. D. Constantinides examined the urine and found albumin 0.365 per cent., fat 0.225 per cent., sugar negative; also some pus cells and epithelium. On allowing the urine to stand, a creamlike mass of fat rose to the surface.

Condition on Admission.—The child was anemic and very emaciated, weighed only 34 pounds, was not playful, complained of frequent frontal headache, was nauseated and vomited several times a day, was very thirsty, with general

weakness, constipation and loss of appetite. The heart, lungs and abdominal organs were normal; there was some dullness at the lower part of the abdomen and some nodules could be palpated at the right inguinal region. The glands at both groins were also palpable. There was no edema and no cyanosis. The patella reflexes were somewhat exaggerated. The temperature was 99.4, pulse 120, respiration 28.

A blood examination showed erythrocytes 3,800,000, total leukocytes 14,400, polymorphonuclear cells 70 per cent., lymphocytes 30 per cent. There was no eosinophilia. The hemoglobin was 60 per cent. No filaria were found in the blood.

Von Pirquet tests were done twice with negative results; Wassermann test was also negative. The stools contained no ova or red cells. The guaiac test for sugar was positive.

REPORT OF URINE EXAMINATION BY DR. D. CONSTANTINIDES

Amount in 24 hours.....	500 to 600 c.c.
Appearance	Somewhat milky
Specific gravity	1.012
Albumin	0.6 per cent.
Sugar	Negative
Urea	0.625 per cent.
Total nitrogen	0.42 per cent.
Chlorids	0.38 per cent.

Extract after evaporation—

Solids	22.8 gm. in 1,000 c.c.
Organic matter	18.3 gm. in 1,000 c.c.
Inorganic matter	4.5 gm. in 1,000 c.c.
Fat	1.5 gm. in 1,000 c.c.

The urine also contained lecithin and cholesterolin.

The microscopic examination showed many white cells, a few hyaline casts and bladder epithelium, but no filaria. A diagnosis was made of chyluria.

During the first week of the child's stay in the hospital, under the administration of urotropin and sodium benzoate her condition improved somewhat and at times the urine almost cleared up, but it always contained some fat. Then the temperature began to rise and reached as high as 104.6. The headache, nausea and anorexia increased in severity, the urine became loaded with pus, markedly ammoniacal, and the fat increased in quantity. The stools contained some blood. The cervical glands became enlarged, although there was no congestion of the throat, and some ecchymotic spots were noticed all over the body and extremities. The pulse became very weak and at times irregular.

The eyes were examined by Dr. Ervin Török on January 15 and he found that the pupils reacted only sluggishly to light. The whole of both disks was covered with a whitish exudate and was moderately swollen. There were several hemorrhages on and around the disks. The blood vessels were large and tortuous. The diagnosis was albuminuric retinitis.

A blood culture gave a negative result. The same evening and on the following day the patient had a number of convulsive attacks, which were most marked on the right side of the body. These were controlled by chloroform. A lumbar puncture was done, but the spinal fluid was normal.

On January 17 the patient became comatose and the pulse was at times imperceptible. The cervical glands became more enlarged and death ensued from cardiac failure.

Postmortem and microscopic examination was performed by Dr. Eli Moschowitz, pathologist to the Beth Israel Hospital, to whom I am indebted for the following report:

A necropsy was performed on the child Jennie G. (Case 6690) eight hours after death. The body was very much emaciated. The cervical, axillary and groin glands were enlarged. Some ecchymotic and petechial spots were noticed over the body and extremities. The lungs were normal, except some slight congestion. The heart was much enlarged, the muscle of left ventricle being about $2\frac{1}{2}$ cm. thick, firm and pale. The left ventricle was dilated, and the muscle of right ventricle was 1 cm. thick. The valves were normal, with the exception of a few atheromatous patches on the ventricular surface of the mitral valve. The liver was slightly enlarged and on section was of a pale, brownish red and cloudy. The stomach mucosa was covered with mucus, but otherwise normal. The right kidney was 8 by 3 by 2 cm. It was very irregular in contour, showed fetal lobulations, and was fairly firm, with the capsule adherent. The surface beneath was very irregular, with numerous depressions and sulci between prominences seeming to represent remnants of kidney tissue. These prominences were paler than the surrounding portions, project above the surface and, on section, were grayish and sharply defined from the surrounding tissue. Section through the kidney proper showed a complete obliteration of all markings, so that it was impossible to differentiate cortex from medulla. The cut section was pale red. There were numerous ecchymoses on the surface, the pelvis was somewhat dilated, and the papillae were flattened. The ureter was somewhat dilated and thickened. The left kidney was 6 by 3 by 2 cm., was somewhat smaller than the right and presented practically the same picture, except for the absence of fetal lobulations. Only the left adrenal was removed; it was slightly enlarged, otherwise normal. The pancreas was enlarged. The spleen was also enlarged and measured $12\frac{1}{2}$ by 6 by 3 cm. The surface was smooth. It was deep red on section and the pulp was firm. The malpighian bodies were prominent. There was a fresh infarct of a dirty brown color, about the size of a bean in the lower pole, soft in texture and sharply circumscribed from surrounding tissue. The intestines were normal, the mesenteric lymph nodes enlarged. The bladder was dilated, the walls being extremely hypertrophied, the mucosa smooth, and presenting many ecchymotic areas.

An anatomical diagnosis was made of chyluria; chronic diffuse nephritis (contracted kidney); chronic congestion of lungs; hypertrophy and dilatation of left ventricle; splenic hyperplasia with fresh infarct; parenchymatous degeneration of liver; hypertrophy and dilatation of bladder; hypertrophy and dilations of ureters.

Microscopic examination of the lungs showed the epithelium of the bronchi to be desquamated and the lumen to contain an exudate of polymorphonuclear cells. Around the smaller bronchi the alveoli contained an exudate consisting of polymorphonuclears and round cells. The walls of the alveoli in the remainder of lung were thickened and the capillaries were markedly dilated. The muscular fibers of the heart were normal and there was no infiltration. The cells of the liver were slightly granular, and the nuclei somewhat pale. There was a very moderate amount of fat in the cells.

Examination of the spleen showed the splenic veins to be dilated and filled with blood. An infarct showed profound necrosis. There were many shadow cells and nuclear detritus. Some of the blood vessels in the necrosed area were still preserved and contained red thrombi; others showed beginning hyaline degeneration. The splenic veins surrounding the infarct were dilated to an enormous size. The capsule and trabeculae of the spleen were normal.

The pancreas showed a few areas of autodigestion. Scattered here and there throughout the organ were small focal areas of polymorphonuclear infiltrations. In these areas the pancreatic cells stained less deeply than in the remainder of the organ. The islands of Langerhans were normal.

The microscopic picture of the kidneys, even on superficial inspection, was seen to be completely disorganized. The interstitial connective tissue was enormously increased and in areas showed a profound infiltration with round and plasma cells. The tubules varied greatly in size and in shape; no distinction could

be made between any of the various portions of the tubules. The majority of the tubules were dilated, the epithelium was flat and stained poorly. Most of them contain hyaline casts, and some of these casts contained polymorphonuclears. The glomeruli revealed various forms, some being completely hyaline, some partially so; some containing new connective tissue, others enormously hypertrophied, and still others showing a polymorphonuclear infiltration. The Bowman capsules were uniformly intact. A few of the glomeruli contained bacterial thrombi. The capillaries were very prominent and dilated.

In the adrenals the cortical cells did not stain uniformly, some being extremely pale. Just beneath the capsule were many small foci of polymorphonuclear infiltrations. In the center of the majority of these foci were small bacterial emboli. There were a few bacterial emboli just beneath the capsule that were not surrounded by polymorphonuclear infiltration. The capsule of the adrenal showed marked extravasation of blood. The medullary portion of the adrenal revealed nothing abnormal.

The intestines and the mesenteric lymph nodes were normal. In the submucous connective tissue of the bladder there was a marked blood extravasation, but the organ was otherwise normal.

REMARKS

This case presents a number of interesting features:

1. It is possible that the severe attack of measles at the beginning of the third year was the causative factor of the kidney derangement and also the cause of the chyluria. While a serious degree of nephritis is supposed to be rare after measles, still Dr. E. Henoch states that as in other infectious diseases, cloudy swelling of the cortex of the kidney is by no means infrequent. Dr. Henoch met with three cases and Dr. G. F. Still saw two cases of nephritis following measles.

2. Had the parents allowed us to tap the abdomen when the ascites was most marked, we probably would have found chylous fluid.

3. There was a disappearance of the ascites under tonic treatment and a marked improvement of the chyluria by rest and the administration of urotropin, salol and belladonna.

4. The course of the disease during the last few days before death and the necropsy findings certainly point to chronic diffuse nephritis, chyluria and some general septic infection. Still, the blood culture failed to show any organisms.

Chyluria (nonparasitic), as already stated, is excessively rare in childhood and a survey of the literature fails to locate any special article which considers it at this period of life. Of five patients who might be classed as children, two were 16, one was 15 and in another no age is mentioned, the patient being termed "a young girl," who was doubtless at least in her teens. The remaining case occurred in a child of 18 months and it is not known that chyluria existed for more than a single day.

It is therefore highly probable that the author's case is almost if not quite unique, at least as far as age incidence is concerned. Four of

the patients might be ranked as adults and the remaining patient as an infant.

A few words concerning these cases may be of interest. The case¹ in the infant was one of combined chyluria and glycosuria, such as has been encountered at times in adults. There is said to have been familial diabetes in the families of both parents. The attack of glycosuria lasted several days and could not be traced to the ingesta. As already stated, the urine was milky on one day only and only the forenoon urine.

Bouchut² saw chyluria of a week's duration in a very hysterical girl of 15. The urine separated into two strata on standing, the upper one thick and creamlike, the lower shallow and clear.

Pasteau's³ case in the "young girl" was associated with right nephroptosis and left renal tuberculosis. Chyluria was most marked in the night urine.

Phillips'⁴ case in a boy of 16; chyluria appeared after recumbency, whether at night or in the daytime.

Becigneul's⁵ case was much like the preceding, occurring in a 16-year-old boy and only upon passing urine after recumbency. The condition was intermittent and lasted eight months.

In none of the above cases was any parasite found.

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1. Brandenburg: *Deutsch. med. Wchnschr.*, 1909, xxxv, 934.

2. Bouchut: *Gaz. d. hôp.*, 1879, No. 36, lii, 874.

3. Pasteau: *Asso. française d'urologie*, 1908, xii, 256.

4. Phillips: *Tr. Clin. Soc.*, London, 1898-1899, xxxii, 216.

5. Becigneul: *Gaz. med. de Nantes*, 1903, Series 2, 403, 405.