POSTOPERATIVE ANURIA

ROBERT I. HILLER, M.D., F.A.C.S.

Attending Surgeon, Mt. Sinai Hospital; Surgical Staff Member, Johnston Emergency Hospital

MILWAUKEE, WIS.

POSTOPERATIVE anuria or suppression of urine is a most distressing complication and like acute dilatation of the stomach, may arise after almost any type of major operation or injury. Evans,[1] in 1931, reported a fatality from anuria in a man, aged thirty-five years, which began on the sixth day following an operation for gangrenous appendix. The blood pressure, which had not been noted prior to the onset of the anuria, was consistently low in spite of the progressive increase of nitrogen retention. The anuria in this case was attributed to the fact that the blood pressure was not high enough to cause kidney function. Autopsy disclosed only slight cloudy swelling of the kidneys and liver. Microscopically, the kidneys showed only a small amount of acute parenchymatous degeneration. Segal and Read[2] have recently reported 2 cases admitted to the urologic service of the Coney Island Hospital with symptoms of inability to void, in which the bladder was explored and it was later discovered that one patient was suffering from a ruptured carcinoma of the stomach and the other from a perforated gastric ulcer near the pylorus. At autopsy, in the first case, the kidneys showed only arteriosclerosis. McCandless[3] has just reported a case of oliguria in a man, aged forty-two years, following a hemolytic blood transfusion in which the donor and patient were both type IV. The transfusion was given for severe hemorrhage and after a stormy period of nitrogen retention, the patient finally recovered. A review of the literature by this author disclosed 29 such cases with a mortality rate of 62 per cent. Bordley[4] outlined the clinical syndrome that is characteristic in this type of a case as follows: (1) Immediately after the transfusion there is a sharp febrile reaction, followed frequently by hemoglobinuria and invariably by suppression of urine; (2) there is an interval of several days, during which there is symptomatic improvement but continued oliguria; and (3) after that interval the characteristic uremic symptoms of agitation or drowsiness followed by convulsions and coma usually develop. Profuse diuresis heralds recovery, whereas, the fatal cases terminate by uremia. He offers four possible explanations for the mechanism by which the incompatible blood damages the kidneys: (1) By mechanical blockage of their tubules, the kidneys have been rendered functionless as excretory organs; (2) the kidneys are sensitive to certain bodies contained in the injected blood, and the functional decline results from a local reaction which is in the nature of an anaphylactic shock; (3) the immediate transfusion reaction causes a metabolic disturbance, such as loss of chlorides, that affects renal function; and (4) by the action of toxic substances set free in the blood at the time of transfusion, the functioning renal tissue is so severely damaged that it is unable to perform its duties. An analogy has been drawn between transfusion reaction and blackwater fever to substantiate the latter explanation. At autopsy, he described the kidneys as swollen, the tubular epithelial cells as containing droplets of a peculiar pigmented material and showing advanced degenerative changes, and the tubular lumina filled with various cells, blood pigment and debris. Small necroses were generally found in the liver.

Since little is positively known about the mechanism of development of postoperative suppression of urine, therapy has likewise been speculative. The list of therapeutic measures employed has included hot packs with injections of pilocarpine; intravenous injections of urea, hypertonic glucose, magnesium sulphate, or methenamine; intramuscular injections of the patient's blood; venesection; purging with magnesium sulphate instilled through a duodenal tube; and spinal puncture. In

1925, Bancroft[5] reported the case of a woman, aged forty-nine years, who developed anuria following a blood transfusion by the Unger method. The transfusion had been given for severe hemorrhage after an incomplete abortion. On the ninth day, as a last resort he performed a decapsulation operation on both kidneys. At operation the kidneys appeared "almost necrotic," large, grayish and friable. Recovery followed, although complicated by the development of an intraperitoneal abscess on the right side ascribed to injury of the peritoneum on that side. Decapsulation of the kidneys for nephritis was first performed by Harrison in 1896 and in 1901 by Edebohls, according to Bessessen,[6] who reviewed the literature on this subject. It was a measure to be employed when all others had failed and the following results were to be anticipated: improvement in 65 per cent, failure of improvement in 20 per cent to 25 per cent and death in 5 per cent to 10 per cent of the cases.

Two cases of postoperative suppression of urine are herewith reported.

Case 1. L. G. aged twelve years, white female, was admitted to the hospital on May 12, 1935, with a history of pain in the right lower abdominal quadrant and vomiting beginning twelve hours before admission. A cathartic had been administered. Previously she had had chicken pox, mumps, measles and a tonsillectomy. Her history, otherwise, presented no significant facts.

Physical examination disclosed a blood pressure of 120 systolic and 80 diastolic, a temperature of 98.6°, a pulse rate of 92 and a respiratory rate of 20. The only abnormal physical findings were severe tenderness and muscle spasm in the right lower abdominal quadrant. A blood count disclosed leucocytes in the amount of 21,850 with 84 per cent neutrophilic polymorphonuclears and 16 per cent lymphocytes. The urine was negative for albumin and sugar.

An emergency appendectomy was performed through a McBurney incision under nitrous oxide and ether anesthesia. The appendix was removed by the ligation and over-sewing technique and the abdomen closed in layers without drainage. Free fluid was present in the peritoneal cavity in excess of normal and the appendix was distended, the serosal vessels congested and covered with fibrinoplastic exudate. Gross and microscopic examination of the removed appendix resulted in a pathological diagnosis of "Acute ulcerative and purulent appendicitis."

The postoperative course was almost uneventful until the sixth day. The temperature rose to 100° on the first day and declined to normal on the fourth day. The pulse rate was rather rapid, 140, but declined to 78 on the fourth day. A carminative enema was given on the third day with very good results. The skin clips were removed on the fifth day, and the patient was about to be dismissed, when she began to complain of feeling tired and achy all over. On the sixth postoperative day she passed a small amount of bloody urine and thereafter ceased to void. Vomiting occurred and the patient complained of pain on the left side. In spite of repeated intravenous infusions of hypertonic glucose solutions, hot pilocarpine packs, an intramuscular injection of the patient's blood, and colonic irrigations, the patient failed to void and sank deeper and deeper into uremia as the blood non-protein nitrogen rose from 85.7 mgm. on May 20, 1935, to 129.9 mgm. on May 22, 1935. On the same dates, the blood creatinine readings were 6.1 mgm. and 9.4 mgm. respectively. Convulsions developed and the blood pressure rose to 160 systolic and 110 diastolic. Two cubic centimeters of 50 per cent magnesium sulphate were administered intravenously, but the convulsions recurred and the patient's condition was critical. Anuria had been present for about one hundred hours when a decapsulation operation was performed at the suggestion of Dr. Francis Murphy. A spinal puncture was performed and about 10 c.c. of spinal fluid under greatly increased pressure withdrawn for decompression purposes. Spinal anesthesia with 75 mgm. of novocain crystals was then instituted. An oblique incision was made in the right loin and the lower pole of the right kidney exposed. The kidney was cyanotic, distended with blood and the capsule was tense. A small incision was made through the capsule on the convex surface of the kidney and the finger inserted through this incision to strip the capsule from the kidney on all surfaces. A Penrose drain was inserted into the wound which was then closed in layers. The patient left the operating

room in fairly good condition. A duodenal tube was then passed, magnesium sulphate administered through the tube, and the patient placed in a hot pack. Urination first occurred twelve hours after the operation. Numerous involuntary urinations followed at intervals of a few hours, until the patient emerged from her coma five days after the decapsulation operation. The urine at first contained traces of albumin and many white and red blood cells. By May 28, 1935, only a few leucocytes and a very faint trace of albumin were notable in the urine. The blood non-protein nitrogen on May 25, 1935, was down to 41.7 mgm. The drain was removed from the kidney wound on the fourth day and the stitches on the eighth postoperative day. The patient left the hospital in a wheelchair on the tenth day. The urine was examined regularly following her dismissal from the hospital and by June 22, 1935, it was entirely free of pathological findings.

Case II. Mrs. B. S., a white housewife, aged thirty-four years, was suddenly seized with pain in the abdomen and nausea at 10 P.M. on March 8, 1934, after a heavy, greasy meal. During the week preceding her illness she had partaken daily of mushrooms. Following the onset of this pain and nausea which made her feel rather faint, she vomited most of the food eaten. Two enemas and a cathartic failed to produce a bowel movement. At 3 A.M., she again fainted and vomited, complained of pain in both upper abdominal quadrants and morphine was administered hypodermically. At 8 A.M. the patient was comfortable but weak. At 9 A.M. tenderness in the right lower quadrant was noted, although she complained of pain principally in the right upper quadrant where no tenderness could be elicited. At 11 A.M., there was quite pronounced tenderness in the right lower abdominal quadrant and hospitalization was advised. She was admitted to the hospital at 3 P.M. on March 9, 1934 where a blood count yielded the following results: hemoglobin 66 per cent, red blood cells 3,660,000, white blood cells 45,950 with 36 per cent non-segmented and 54 per cent segmented polymorphonuclears, 5.5 per cent lymphocytes, and 4.5 per cent monocytes.

Inventory of her systems revealed only the fact that she had had a vaginal discharge after her periods for some time, which had been noticeable daily during the preceding month. Her periods had otherwise been regular and normal; last period two weeks prior to her present illness.

Aside from having had two children, seven and five years, respectively, and an attack of "kidney trouble" in childhood, her past history presented nothing of note medically or surgically. Her use of alcoholic drinks had been rather free.

Physical examination on admission to the hospital disclosed a well developed and nourished adult white female of marked pallor. She was restless and appeared acutely ill. Her temperature was 98°, pulse rate 102, respirations 24, and blood pressure 102/60. Her pupils were pinpoint in size and her breath was offensive. Examination of the heart and lungs disclosed no notable abnormalities aside from a rapid heart rate. Her abdomen was distended. There was no muscular rigidity, but considerable tenderness in the right lower quadrant and moderate tenderness in the left lower quadrant of the abdomen. Peristalsis was faintly audible. Vaginal examination disclosed a fullness in the cul-de-sac of Douglas with tenderness in the right fornix. The cervix was closed and painful to manipulation. The urine was yellow with a specific gravity of 1.035, an acid reaction, and was negative for albumin, sugar, and microscopic findings of a pathological nature. A second white blood count two hours after admission showed the leucocytes to number 43,750 with 39 per cent non-segmented and 47.5 per cent segmented polymorphonuclears, 8 per cent lymphocytes, and 5.5 per cent monocytes. The patient's blood was type IV, Moss.

A diagnosis of ruptured ectopic pregnancy was made.

A surgical consultation verified the foregoing findings and the condition was diagnosed as a "surgical abdomen" due most likely to a gangrenous appendix, with ectopic pregnancy as a second possibility.

At operation, under ethylene and ether anesthesia with morphine and atropine preliminary medication, a midline incision in the lower abdomen was made. The peritoneum was opened and a large quantity of free blood encountered, the source of which seemed to be a ruptured cyst of the right ovary and a small perforation of the cornual end of the left tube. The left tube was adherent in the cul-de-sac of Douglas. It was dissected free and removed and the raw area sutured. The right tube and ovary were removed to stop the ovarian bleeding.

Blood was also seen to come from several small rents in the pelvic peritoneum producing a generalized oozing most marked near the right broad ligament and in the cul-de-sac. A Penrose drain was inserted and the abdomen closed in layers. With the patient still in the Trendelenberg position 500 c.c. of blood were administered by the citrate method. At the conclusion of the operation the blood pressure was 150/60, pulse rate 130, and respiratory rate 30.

Pathological Report of Specimens Removed at Operation. "Gross specimen is that of an ovary and attached Fallopian tube. The ovary contains a ruptured follicle cyst measuring 5 × 2.5 × 1 cm. The attached tube is patent and slightly edematous with no evidence of ectopic pregnancy.

"*Microscopic Examination.* The ovary contains several recent follicular cysts. Cortex is moderately cellular. Few primary follicles. The ruptured cyst contains a corpus luteum lined by fibrin and lutein cells of recent origin. Around the margins of the cyst is recent hemorrhage and edema. Section of the tube reveals hemorrhagic reaction on the serosa, generalized congestion, edema and early fibrinous and leucocytic exudate. Mucosa and lumen are free of any pathology. Diagnosis: Ruptured corpus luteum. Acute peri-salpingitis."

Postoperative Course. Because of the generalized oozing at operation a platelet count was deemed advisable, which on the following day showed 40,000 platelets. Hemoglobin was 58 per cent, red blood cells, 2,450,000, and white blood cells 18,200.

At first there was considerable bloody drainage from the wound, but after the administration of thromboplastin, calcium gluconate, 20 c.c. of whole blood intramuscularly, and ceanothyn by mouth, this oozing stopped, especially when the platelet count reached 170,000 on March 12, 1934.

There was some distention during the entire postoperative course, in spite of the fact that gas and liquid fecal material were passed constantly after the second day, either voluntarily or through a rectal tube. In fact, a diarrhea occurred from the eighth through the eleventh postoperative days. The nasal suction and on occasions pitressin were used to combat the distention. Nausea was a more or less constant complication. On two occasions light food was tolerated for a few days, but nausea and vomiting supervened.

The temperature was constantly elevated, maintaining a level of about 103° rectally until the fourteenth day, after which it rose steadily, reaching 106°, on the sixteenth day just before death. The pulse rate was also constantly elevated, hovering between 110 and 140, except the thirteenth day only, when it remained at 100. The respiratory rate, too, was elevated throughout her hospital stay, ranging between 24 and 30 most of the time. The blood pressure on the second day was 148/80. Subsequent readings showed a low pressure of 98/60 to 110/50 until two days before death when it rose progressively to 150/98. Two vaginal examinations postoperatively disclosed no abnormal findings. The blood cultures and Widal tests were negative. On March 13, 1934, the erythrocytes numbered 2,970,000, leucocytes 9900, and platelets 132,000, and a transfusion of 500 c.c. of citrated blood was administered on that day. On March 15, 1934, the erythrocytes numbered 3,400,000, hemoglobin 76 per cent, platelets 220,000, but the leucocytes had risen to 15,600 with 67 per cent non-segmented and 2 per cent segmented polymorphonuclears, 17.5 per cent lymphocytes, 10 per cent monocytes, Turck's cells 2.5 per cent, and myelocytes 1 per cent. The urine, which had previously been negative for albumin was noted to give a two plus reaction and was loaded with bacteria. Catheterization had been necessary only once postoperatively. On March 17, 1934, the hemoglobin was 78 per cent, red blood cells 3,940,000, white blood cells 16,750 with 65.5 per cent non-segmented and 7 per cent segmented polymorphonuclears, 24.5 per cent lymphocytes, and 3 per cent monocytes. On March 20, 1934, during the period of diarrhea, the erythrocytes were noted to have dropped to 2,180,000, hemoglobin 68 per cent, whereas the leucocytes numbered 15,050 with 65.5 per cent non-segmented and 17 per cent segmented polymorphonuclears, 9.5 per cent lymphocytes, 7 per cent monocytes, and 1 per cent myelocytes. The urine still gave a two plus reaction for albumin and many coarsely and a few finely granular casts, a moderate number of red blood cells and epithelial cells were noted. On March 21, 1934 450 c.c. of citrated blood were administered. On March 22, 1934 another transfusion was attempted through the continuous intravenous set-up but some difficulty was encountered and only about 100 c.c. of

blood were actually administered.* Following this, the urine was noted to be bloody, specific gravity 1.020, acid in reaction, albumin three plus, negative for sugar, and loaded with erythrocytes and leucocytes. On March 23, 1934, the urinary output began to decrease. Urinalysis showed specific gravity of 1.016, albumin three plus, erythrocytes and leucocytes in large quantities, and an occasional coarsely granular cast. Blood chlorides were 0.663, non-protein nitrogen was 81.9 mgm. and creatinine 2.84 on examination of the blood. On March 24, 1934, edema began to develop and muscular twitchings were quite pronounced. Small quantities of urine were voided involuntarily. Urinalysis yielded findings similar to those of the previous day with the addition of a great amount of laked blood. Fifteen per cent glucose solution intravenously failed to increase the urinary output or reduce the edema. The patient's condition became progressively worse; coma developed on March 24, 1934 followed by death at 9:30 P.M. on March 25, 1934.

The drugs used during the postoperative course were: thromboplastin, calcium gluconate, glucose and saline, ceanothyn, morphine, pantopon, sodium luminal, pitressin, codeine, sodium bromide, allonal, sodium amytal, digifoline, eschatin, oxygen, caffeine sodium benzoate, and insulin.

At autopsy, the peritoneum was smooth with no evidence of peritonitis. The operative site showed good healing surrounded by recent adhesions; no evidence of thrombosis of pelvic veins. The lungs showed massive edema. The spleen, pancreas, adrenals, uterus and gastrointestinal tract appeared normal. The liver was normal in size, pale yellow, brown and slightly fatty. Microscopically it showed widespread fatty change but no evidence of necrosis. The kidneys were not enlarged but were congested, the capsules stripped easily and small amounts of cloudy urine were found in the pelves. Microscopically there was marked tubular degeneration, hemorrhage into the tubules and hemorrhagic casts, with swelling and edema of the glomeruli and some periglomerular exudate of serum and red cells.

Anatomical Diagnosis. Toxic degeneration of kidney; fatty liver; mushroom poisoning (?).

On November 6, 1935, the left tube was rechecked histologically and evidence of an ectopic pregnancy was found.

* The donor for this transfusion was recently rechecked on November 11, 1935 and he was found to be a Group IV, Moss.

COMMENT

The first case corresponds to what is clinically known as "reflex anuria." The onset of the suppression of urine was comparatively sudden after apparently normal kidney function for six days following appendectomy. Death was impending when the decapsulation operation was done. Resumption of kidney function ensued, so that it is logical to assume that it was the result of the operation.

The second case, however, was one of a progressive development and might well be described as one of "progressive renal failure." Here, there was first a massive intraperitoneal hemorrhage followed by apparent recovery except for constant nausea, fever and elevated pulse rate. Then, on the sixth postoperative day a leucocytosis and albuminuria appeared followed by the development of a diarrhea and a drop in the hemoglobin and erythrocyte count. Finally, with a normal blood chloride reading, a decreased urinary output, bloody urine, edema, retention of nitrogen products in the blood, and a rise of blood pressure terminated the picture on the fifteenth day. Degeneration of the tubular epithelium with periglomerular swelling were the only findings of note at autopsy. These findings correspond with the ones noted in cases of suppression of urine following transfusion. Whether this type of case would respond to a decapsulation operation is a matter of conjecture.

CONCLUSIONS

1. Two cases of postoperative suppression of urine are reported; one in which the suppression was progressive and another in which the onset was comparatively sudden, corresponding to the type of case which is clinically known as "reflex anuria."

2. A decapsulation of the right kidney was performed in the case of "reflex anuria."

3. The operation would appear to be of value in this type of case.

[*For References see p.* 368.]

is with weakness, tenderness and stiffness and sometimes slight pain on forced movement of the wrist. In two or three years the disease becomes stationary with residual limitation of motion and weakness. In the usual anterior type of deformity, extension of the wrist is diminished and flexion is increased; there is an increase in adduction and a diminution in abduction. In other words, those motions which oppose the direction of the deformity are restricted while the opposite motions are increased.

The disease is not occupational. However, it occurs more often in the poor classes suggesting that a nutritional element may be involved. It is bilateral in about 50 per cent of the cases although one wrist usually shows more involvement.

SUMMARY

The outstanding features of a case of Madelung-like deformity presented are the following:

1. There is primary involvement of the ulna producing an atypical form of deformity, which is most unusual.
2. The association of possible endocrine disturbance is suggested by the disturbed menstrual history of the patient from the onset of puberty.
3. The case is observed at a late stage.
4. There is an associated essential hypertension.

REFERENCES OF DR. HILLER*

1. Evans, Theodore S. Azotemia with normal kidneys found postmortem. *Arch. Int. Med.*, 48: 1231 (Dec.) 1931.
2. Segal, Abraham D. and Read, J. Sturdivant. Complete anuria in perforated gastric lesions. Report of 2 cases. *Am. Jour. Surg.*, 30: 276 (Nov.) 1935.
3. McCandless, H. G. A hemolytic blood transfusion reaction with oliguria. *J. A. M. A.*, 105: 952 (Sept. 21) 1935.
4. Bordley, James, III. Reactions following transfusion of blood with urinary suppression and uremia. *Arch. Int. Med.*, 47: 288–315 (Feb.) 1931.
5. Bancroft, F. W. Anuria following transfusion, effect of decapsulation of both kidneys. *Ann. Surg.*, 81: 733 (April) 1925.
6. Bessessen, Daniel H. Renal decapsulation for nephritis; a brief review. *Am. Jour. Surg.*, 38: 264 (Nov.) 1924.

* *Continued from p. 356.*