Acute Renal Failure Associated with Increased Intra-abdominal Pressure

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Anuric renal failure developed in four patients in association with increased intra-abdominal pressure from postoperative hemorrhage. Polyuria and resolution of the renal failure occurred in each patient in response to operative decompression of the abdomen. Renal failure secondary to increased intra-abdominal pressure has been previously produced experimentally in dogs by the intraperitoneal installation of graded amounts of saline. This is the first report of this type of renal failure in clinical practice.

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ANURIA AND RENAL failure can be produced in dogs by instilling saline into the peritoneal cavity. The primary cause of anuria and renal failure in this model seems to be related to the level of abdominal pressure since pressures greater than 15 mmHg induce oliguria and pressures greater than 30 mmHg induce anuria.[10] There is little data concerning renal function and abdominal pressures in patients.

Four patients are described with anuric renal failure associated with tense abdominal distention. Polyuria ensued in each patient after the intra-abdominal pressure was reduced. Experimental data confirming previous reports is discussed.

Patient Population and Methods

Four patients treated by the General Surgery Services at the University of Maryland Hospital during 1981 are presented. Two of the patients had penetrating trauma of the abdomen, one patient had adenocarcinoma of the colon with liver metastases, and one patient had cirrhosis with splenomegaly and persistent thrombocytopenia. There were three males and one female, ranging in age from 26 to 64 years. Characteristically, these patients had required an average of 6 liters of blood during their first operations and subsequently developed severe coagulopathy with intraperitoneal bleeding in the early postoperative period. Although all these patients ultimately recovered renal function, two subsequently succumbed to other medical problems.

Three adult mongrel dogs weighing 10 to 20 kg were used in the experimental portion of the study. Intravenous pentobarbital sodium (30 mg/kg) and controlled ventilation (15 mg/kg body weight tidal volume) were used for anesthesia. A midline incision provided exposure to cannulate both ureters with 20-gauge polyethylene tubing; the left cannula being advanced into the renal pelvis and the right being advanced only 4 cm proximal to the ureterovesical junction. The purpose of this maneuver was to insure that compression on the ureter did not itself cause anuria. A catheter was tunneled through the abdominal wall and the incision closed in three layers of O-nylon suture to form a watertight seal. A femoral vein catheter was positioned at the level of the renal veins. Arterial blood pressure was monitored through a catheter placed in the carotid artery.

Pressure measurements from the carotid artery, inferior vena cava, and peritoneal cavity were monitored using a 12-channel Hewlett Packard recorder. Physiologic Kreb's solution at 37° was introduced by syringe into the peritoneal cavity via the implanted catheter.

Case Reports and Results

Case 1

A 26-year-old female accidently shot herself in the abdomen with a hand gun. She arrived in the Emergency room with a systolic blood pressure of 75 mmHg. Urinalysis was normal, the preoperative creatinine was 1.0 mg/dl, and the blood urea nitrogen (BUN) was 10 mg/dl. Prompt fluid resuscitation and operative repair of a through-and-through injury of the portal vein and inferior vena cava was accomplished.

Intraoperatively, the patient required 56 units of blood and 13.5 liters of crystalloid and colloid solution to maintain her blood pressure.

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Submitted for publication: May 7, 1982.

0003-4932/83/0200/0183 $01.05

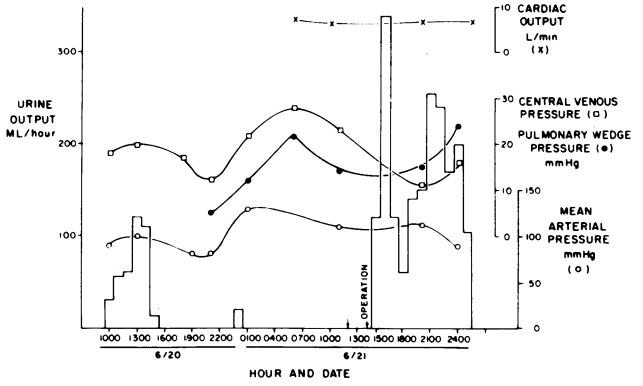

FIG. 1. Urine flow in ml/hr (□), mean arterial pressure (○), central venous pressure (□), pulmonary wedge pressure (●) in mmHg, and cardiac output in L/min (×) in patient two. Operation to decompress the abdomen was performed 6/21/81 beginning 1130 hours and ending 1400 hours as depicted by the arrows.

Even with such massive fluid administration, the first operation was complicated by hypotensive periods requiring the institution of cardiac message. At the end of the operation, clotting studies were grossly abnormal and significant oozing continued from the retroperitoneal tissues. The abdomen was closed without drains, and increasing abdominal girth was soon apparent. Her urine output was now 95 ml/hr and the serum creatinine was 2.6 mg/dl.

Anuria developed 16 hours after operation, by which time the abdomen was tensely distended. The vital signs at this time were adequate with a pulse of 120 per minute, a blood pressure of 120/80 mmHg, and a central venous pressure (CVP) of 13 mmHg. The Foley catheter was irrigated to ensure that there was no obstruction to urine flow. Fluid challenges and diuretics were given, but there was no significant increase in urine output. Eleven hours later the abdominal cavity was decompressed in the operating room with removal of large amounts of blood and clot. Multiple bleeding sites were identified. During the two-hour operative procedure, the patient excreted 850 cc of urine and the serum creatinine was 2.8 mg/dl. The abdomen was again closed without drains.

The patient continued to show a brisk diuresis until 32 hours after operation when oliguria resistant to fluid challenges and diuretics again developed. Coagulopathy once more contributed greatly to postoperative intraperitoneal bleeding. Blood pressure was 140/105 mmHg, pulse was 130 per minute, and CVP was 19 mmHg. Hemoglobin was 9.3 mg/dl. Over the next 11 hours, the patient had an output of 115 cc of urine. Operative decompression was performed, removing a large amount of clot from the peritoneal cavity, but no single bleeding site was identified. This time multiple closed suction drains were positioned in the retroperitoneal space to prevent further accumulation of blood. The patient excreted 130 cc of urine during the three-hour procedure and continued a brisk diuresis thereafter. The creatinine clearance was 3.4 ml/minute just before the third operative procedure and rose to 10 ml/minute during the second postoperative day, finally rising to 70 ml/minute 24 days later.

Case 2

A 29-year-old male was stabbed in the abdomen with a butcher knife. He was transported with inflated antishock trousers to the emergency room, but had a systolic blood pressure of only 60 mmHg upon arrival. The urine was free of red cells and the serum creatinine was 1.0 mg/dl. Vigorous fluid resuscitation and prompt control of intra-abdominal bleeding was carried out. Four small-bowel mesenteric veins and the inferior mesenteric artery were ligated to control bleeding. Intraoperatively, he received six liters of blood and 13.5 liters of crystalloid and colloid. The abdomen was closed without drains.

After operation, he had a blood pressure of 120/90 mmHg, a CVP of 20 mmHg, a pulse of 140 beats per minute and a urine output of 60 ml/hour. He continued to make urine for 22 hours at which point anuria, unresponsive to fluid challenge, developed. The serum creatinine was now 4.2 mg/dl, and a two-hour creatinine clearance was 7.7 ml/min. Bilateral ureteral catheters were introduced to rule out obstruction as a cause for the anuria. During this period of time, the patient's abdomen became tense and distended. Dorsalis pedis and posterior tibial pulses could no longer be palpated, and pitting edema developed over the petibial areas.

He was returned to the operating room, and a large amount of blood was removed from the abdomen. No single bleeding site was identified. Closed suction drains were placed in the retroperitoneium at closing. After operation, polyuria was noted. (Fig. 1 illustrates his clinical course.) The serum creatinine was 3.9 mg/dl with a creatinine clearance 16 ml/min. These values returned to normal seven days after operation. The pitting edema over the pretibial area resolved within 24 hours following the procedure.

Case 3

A 35-year-old male underwent an elective splenectomy because of persistent thrombocytopenia (7,000 to 15,000 platelets per mm^3). His splenomegaly was secondary to cirrhosis from heavy alcohol abuse. The intraoperative blood loss was 300 cc and there were no immediate complications. Eight hours following surgery, he suddenly complained of abdominal pain, and then developed a cardiac arrest five minutes later. He was resuscitated and quickly taken back to the operating room where significant bleeding was found to be coming from the short gastric vessels. The estimated blood loss was 5000 cc during this second procedure. No drains were used. He was taken to the intensive care unit where persistent coagulopathy was demonstrated. The urine output over the first 12 hours was 97 ml/hour. This decreased to 14 ml/hour over the next ten hours. His abdomen became tensely distended. Blood pressure was 110/90 mmHg with a pulmonary wedge pressure of 21 mmHg. Urinalysis showed a small level of bilirubin, negative protein, negative sugar, rare white blood cells (WBC), rare red blood cells (RBC), and some epithelial cells. The serum creatinine was 2.1 mg/dl. A renal arterial isotope study demonstrated normal bilateral renal artery perfusion. Venous flow studies of the lower extremities by Doppler examination revealed bilateral sluggish venous flow, worse on the left than on the right. An arterial doppler showed abnormal wave forms at the ankle, suggestive of a leg compartment syndrome. The ankle branchial index was 0.76 on the left and 1.1 on the right.

Operative decompression of the abdomen was undertaken, with removal of a large amount of blood that had accumulated from diffuse intraperitoneal oozing. Almost immediately the urine flow increased to 441 ml/hour (Fig. 2). The serum creatinine was 1.9 mg/dl eight hours after operation. Unfortunately, despite excellent response to surgical decompression of the abdomen, this man succumbed to persistent severe metabolic acidosis unresponsive to treatment approximately 18 hours after the laparotomy.

Case 4

A 64-year-old white male had undergone sigmoid resection for adenocarcinoma of the colon one month prior to admission. He was referred to University of Maryland Hospital for evaluation and treatment of his hepatic metastases. These were treated by hepatic dearterialization, cholecystectomy, and resection of small bowel metas-

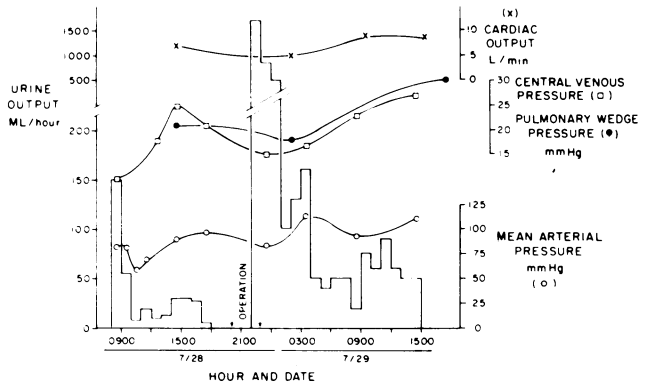

FIG. 2. Urine output in ml/hr (□) mean arterial pressure (○), central venous pressure (□), pulmonary wedge pressure (●) in mmHg, and cardiac output in L/min. (×) in patient three. Operative decompression of the abdomen was carried out on 7/28/81 from 2000 hours to 2300 hours as indicated by the arrows.

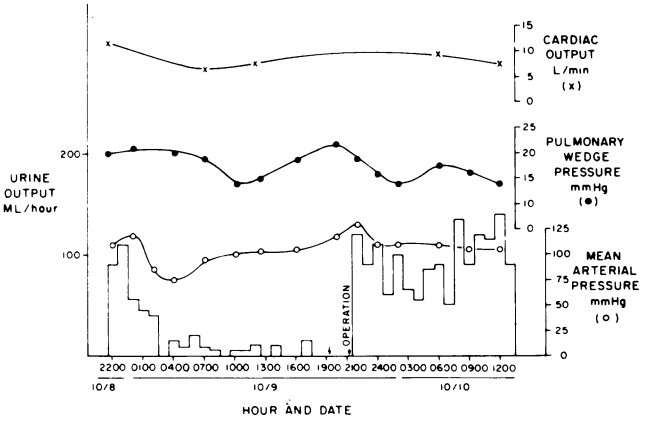

FIG. 3. Urine flow in ml/hr (□), mean arterial pressure (○), pulmonary wedge pressure in mmHg (●), and cardiac output in L/min (×) in patient four. Operation to decompress the abdomen was performed 10/9/81 starting at 1930 hours and ending at 2130 hours as indicated by the arrows.

tases. The operative procedure took three hours and 45 minutes, and the blood loss was 1700 ml. After operation, the serum creatinine was 2.2 mg/dl and the BUN was 22 mg/dl. The patient was stable for ten hours when the urine output fell to 7 ml/hour. An indwelling pulmonary arterial catheter was inserted. This showed the pulmonary arterial pressure (PAP) to be 29/3 mmHg. The pulmonary wedge pressure (PWP) was 2 mmHg and the central venous pressure (CVP) was 0 mmHg. The patient was resuscitated with crystalloid and packed red blood cells (PRBC). The urine output increased to 80 cc/hour, the PAP increased to 36/18, the PWP to 26, and the CVP to 16 mmHg on positive end-expiratory pressure of 10 mmHg. The blood pressure was now 150/80 mmHg, the pulse was 120/minute, and the cardiac output was 7.92 L/minute. The serum creatinine was 3.2 mgm/dl, BUN was 30 mgm/dl, and creatinine clearance was 17 ml/min. Urine chemistries showed a sodium of 88 mEq/L, a creatinine of 48.4 mg/dl, and an osmolality of 270 mOsm/kg. The urinalysis was clear at this point in time.

The patient's abdomen increased in circumference from 51 cm to 53½ cm during the early postoperative period. The urine output fell to 4 ml/hour. At re-exploration 6000 ml of blood were found within the peritoneal cavity. A bleeding point near the ligated hepatic artery was noted.

After operation, the urine output immediately increased to 70–120 ml/hour. Brisk diuresis continued and there was a moderate increase in the creatinine clearance (Fig. 3). The PAP was 52/5 mmHg, the PWP was 14 mmHg, and the cardiac output was 7.5 L/minute. The blood pressure was 180/80 mmHg and the pulse 110/minute. Urinary sodium was 94 mEq/L, and the osmolality was 284 mOsm/kg. The creatinine clearance was 41 ml/min, with the serum creatinine 5 mg/dl. The patient died 15 days after surgery of progressive pulmonary and hepatic failure.

Table 1 illustrates the physiologic parameters before and after operation for patients 2, 3, and 4. Cardiac index and total peripheral resistance were virtually unchanged before and after operation. There was a small drop in pulmonary wedge pressure, central venous pressure, and mean arterial pressure in the three patients after surgical decompression. Case 1 is not listed because a Swan–Ganz catheter was not in place.

Experimental Results

Results from dog 3 (Fig. 4) are typical for the animal studies done in this laboratory and those previously performed in 1923 by Thorington and Schmidt.[10] Intra-abdominal pressure of 15 to 20 mmHg produces oliguria, with concomittant increases in inferior vena cava pressure, although the arterial pressure never falls below 90 mmHg. Anuria ensues with intra-abdominal pressure greater than 20 mmHg. The urine output increases im-

TABLE 1. *Physiologic Parameters During Anuria Associated With High Intra-abdominal Pressure and During Polyuric Phase After Reduction of Intra-abdominal Pressure*

	Anuria and High Intra-abdominal Pressure					Polyuria and Normal Intra-abdominal Pressure				
Case	Cardial Index $L.min^{-1}.m^{-2}$	Pulmonary Wedge Pressure mmHg	Central Venous Pressure mmHg	Mean Arterial Pressure mmHg	Total Peripheral Resistance dyne.sec. $cm^{-5}.m^{-2}$	Cardial Index $L.min^{-1}.m^{-2}$	Pulmonary Wedge Pressure mmHg	Central Venous Pressure mmHg	Mean Arterial Pressure mmHg	Total Peripheral Resistance dyne.sec. $cm^{-5}.m^{-2}$
2	3.5	14	23	110	1986	3.68	15	11	110	2149
3	3.3	21	20	95	1816	2.7	19	15	85	2021
4	4.24	21	16	125	2094	4.33	16	19	110	1679
mean	3.68	18.7	19.7	110	1952	3.57	16.7	15	101	1966

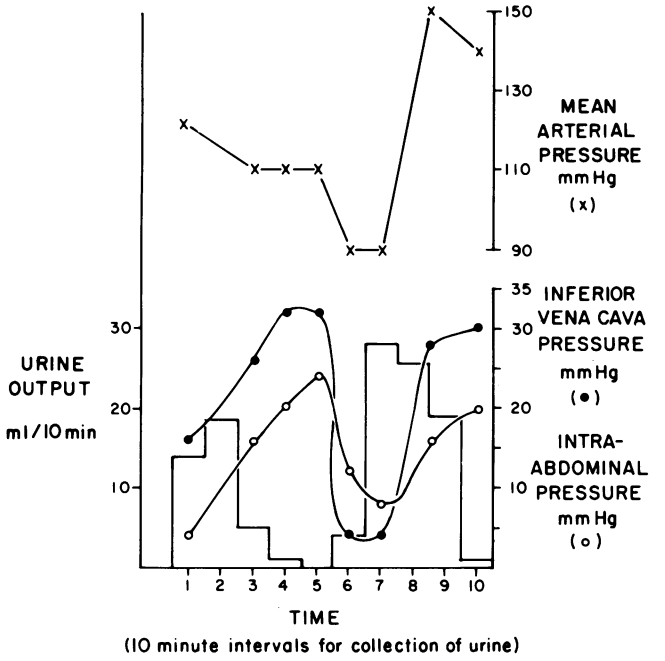

FIG. 4. Urine output in ml/10 min. (□), mean arterial pressure (×), intra-abdominal pressure (○), inferior vena cava pressure in mmHg (●) in dog three. The urine output charted is the sum from both catheters.

mediately with reduction of the intra-abdominal pressure, and the inferior vena cava pressure drops. No difference was noted in the urine output from the left and right ureteral catheters, indicating that no ureteral obstruction occurred during periods of increased intra-abdominal pressure. (Fig. 4)

Discussion

The etiology of acute oliguria and anuria in these four patients is unknown. Restoration of urine flow in all four patients was temporally related to reoperation and release of intra-abdominal pressure. These findings in man are in close agreement with the experimental findings of studies done in 1923 by Thorington and Schmidt[10] and also confirmed by animal studies included as part of this report. In all four patient studies, the mean arterial blood pressure and central venous pressure were either normal or high normal while anuria was present. Bilateral ureteral catheters were placed in one patient (case 2), but this did not reverse the anuria. Ureteral obstruction in the others cannot be totally ruled out, however, no evidence of an obstructing lesion near the ureters was noted in any of the four cases at the time of operative decompression. Reduction of renal arterial blood flow cannot be excluded as an etiologic factor in these patients, however a renal scan done in one patient (case 3) demonstrated normal flow bilaterally. Direct compression of the kidneys might be causally related to anuria in these four patients with increased intra-abdominal pressure. Renal cortical pressures conceivably could be increased in association with increased intra-abdominal pressure. Stone and Fulonwider[9] have suggested that decapsulation of the kidney will improve the prognosis in patients with evolving acute tubular necrosis. He suggests that the kidney could develop a compartment syndrome much like that observed with injury to the brain or lower extremities, with resultant reduction in arterial flow and ischemic injury.

An as-yet-unidentified hormonal factor such as antidiuretic hormone may play a major role in this type of renal failure. High renal pressures may set off delicate sensors within the kidney to produce oliguria and even anuria.[1-3,5] Plasma antidiuretic hormone (ADH) levels have been found to be markedly elevated in dogs in response to elevated intra-abdominal pressures produced by inflation to 80 mmHg of a large sphygomanometer cuff placed around the abdomen. ADH levels rose to two and a half times normal during periods of increased abdominal pressure and fell to normal a half hour after the cuff pressure was released. No correlation between urine output and ADH levels was made in this experiment.[6] In addition, the response to decompression in these patients was too rapid to be satisfactorily attributed to changes in the circulating levels of ADH. The role of other hormonal factors such as renin and angiotensin is speculative in nature.

There is some evidence to support the role of obstruction of the vena cava and renal vein in the etiology of the anuria observed in these patients. Noninvasive Doppler studies indicated sluggish venous flow in the lower extremities of one patient (case 3). Two patients (case 2 and 3) developed severe edema of both legs that resolved within 24 hours after decompressing the abdomen.

Experimental studies performed on human volunteers by Bradley[3] give some insight into the pathophysiology of renal failure from increased intra-abdominal pressure. Renal plasma flow, glomerular filtration rate, and tubular glucose reabsorption were studied in 17 normal human subjects as abdominal pressure was elevated, using a rubber bladder beneath a girdle fitted around the subject's waist. The bladder was inflated with air to pressures of 70 to 80 mmHg which raised inferior vena cava pressure to 20 mmHg. Renal plasma flow, glomerular filtration rate, and glucose reabsorption were all decreased during periods of increased intra-abdominal pressure. The decreased flow rates could be directly attributed to elevated venous pressure in the renal veins and inferior vena cava. These results suggest that a shunt of blood away from the cortex and functioning glomeruli (to medulla) accounts for the decreased renal plasma flow and glomerular filtration rate. Others have corre-

lated increased inferior vena cava pressure and disturbed renal function in alcoholic patients with cirrhosis.[8] Partial renal vein occlusion was the etiology proposed for shunting of blood around glomeruli (and to the medulla), thus reducing the effective renal plasma flow. The disturbed renal dynamics seen in these patients was attributed to partial renal vein occlusion. Baldus et al.[2] found that renal vascular resistance was proportional to renal impairment in 23 adult cirrhotics. At least a portion of the increased vascular resistance was due to increased renal vein pressure, although sympathetic tone and unnamed hormonal factors associated with liver disease were also proposed as major contributors to the increase in renal vascular resistance.

Compression of the intra-abdominal aorta by the high intra-abdominal pressure may increase local arterial vascular bed resistance and lower renal blood flow. Oliguria could progress on to anuria with enough pressure on the aorta. Total peripheral resistance may remain unchanged because of the relatively localized area of increased vascular bed resistance. Kashtan et al.[4] have shown experimentally increased total peripheral resistance and mean arterial pressure in hypervolemic dogs with artificially induced intra-abdominal pressure of 40 mmHg. This experiment did not measure urine output or other renal parameters. High pulmonary wedge pressures and central venous pressures in these patients is at least partially due to large amounts of intravenous fluids during resuscitation efforts. The central venous pressure and mean arterial pressure in the authors' patients and experimental model fell slightly after reduction of intra-abdominal pressure. This may indicate a significant effect of intra-abdominal pressure on the aorta with locally increased vascular bed resistance.

It is clear that increased intra-abdominal pressures were linked to the development of anuria and renal failure in all of these patients. In four documented instances, reduction of the elevated abdominal pressures resulted in an almost immediate polyuria, followed by an increase in creatinine clearance over the next several days. Early experimental studies in dogs have shown that modest increases in intra-abdominal pressure produced by saline infusion lead to oliguria, and larger pressure increases result in anura.[10] These results have been reproduced by others, but no clearly related clinical entity has been described. The hepatorenal syndrome may result, in part, from vena caval obstruction produced by the pressure of ascitic fluid, but decreasing the intra-abdominal pressure through paracentesis does not routinely reverse this type of renal failure.[7,8]

Operative decompression of the abdomen was very effective in the treatment of acute renal failure produced by tense abdominal distension in four patients. Laparotomy is indicated once oliguria unresponsive to fluid therapy develops in association with early postoperative abdominal distension. Patients with massive blood loss, hypotension, and other factors contributing to consumptive coagulopathy need vigorous treatment of coagulation deficits by component therapy. Even with such treatment, there is a likelihood that oozing will continue even after repeated evacuations of blood clot. One method of preventing reaccumulation of blood is by establishing a closed drainage system. Use of such drains was quite effective in two of the patients. Earlier institution of closed suction drainage might have resulted in reduced morbidity for these individuals.

Acknowledgments

The authors thank Herbert Ormsbee III, PhD., and Frank Hardy for their advise and technical assistance during the experimental portion of this paper. We would also like to acknowledge Derryl Johnson and Susan Leap for valuable assistance typing the manuscript.

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