Asynchronous Cortical Necrosis after Bilateral Nephrostomy

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Hemodialysis

Admission

Right nephrostomy

150

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Left nephrostomy

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Dear Sir,

Renal cortical necrosis is commonly bilateral and described as a manifestation of disseminated intravascular coagulation (DIC). Hydronephrosis [1–3] and renal artery stenosis [4] have been reported to protect the kidney from being affected by DIC. It is possible that DIC coincides with hydronephrosis, especially in patients with disseminated cancer in the retroperitoneal area. The following case with bilateral hydronephrosis secondary to metastatic ureteral obstruction is such a case.

Case Report

A 75-year-old woman was admitted on November 18, 1985, because of anuria caused by bilateral ureteral obstruction. Urine obtained later through nephrostomy showed no evidence of bacterial infection. Laboratory data revealed a serum creatinine of 10.1 mg/dl, urea nitrogen (BUN) 157.2 mg/dl, uric acid 11.8 mg/dl and lactic dehydrogenase 478 IU/l. There was no laboratory data suggestive of disseminated intravascular coagulation.

On the day of admission, percutaneous right nephrostomy was performed. Prompt diuresis could be obtained but bleeding through the nephrostomy occurred and required 400 ml of blood

transfusion. Bleeding was managed by thrombin irrigation into the renal pelvis. During 12 h following the nephrostomy, 3,000 ml of urine were excreted; urine output continued to increase on the 3rd day with improvement of BUN and serum creatinine levels to 73.4 and 2.2 mg/dl, respectively (fig. 1). But thereafter urine output decreased gradually, and was not responsive either to extracellular volume expansion or furosemide administration. Because of no evidence of catheter obstruction, percutaneous left nephrostomy was carried out 4 days after right nephrostomy. Marked diuresis was noted again but urine output started to decrease 3 days after the nephrostomy in the same way as in the right side. During two episodes of diuresis and a subsequent gradual fall in urine output, platelet and fibrinogen tended to decrease, although values of PT and activated partial thromboplastine time (APTT) remained within the normal limit. Serum fibrin degradation products (FDP) tested only in the late course of the second episode were elevated beyond normal. She died of respiratory failure on December 8, 1985.

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 November December

Fig. 1. Clinical course of the patient. Right and left nephrostomy were performed as indicated. Normal range: lactic dehydrogenase (LDH) = 120–520 IU/l and FDP = < 100 ng/ml. PT = 70–130%.

Autopsy disclosed cancer of the pancreas with metastasis to liver, thyroid gland and both sides of the ureters. The right kidney was slightly smaller than the left, with a smooth greyish-yellow surface with scattered hemorrhagic spots. On section, the whole cortex was yellowish-white and sharply demarcated from the medulla (fig. 2). Microscopically the cortex was completely necrotic, and every glomerulus contained varying numbers of fibrin thrombi. In the left kidney, intraglomerular fibrin thrombi were noted to the same degree as in the right but cortical necrosis was not evident. Fibrin thrombi were also detected in other organs such as lungs, liver and spleen.

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parity between the two kidneys is not clear. Denervation of the kidney [6], the pretreatment with heparin [7] or α -adrenergic agents [8] have been reported to prevent cortical necrosis. All these treatments were applied in our patient. A difference in clinical events, however, between the two sequential nephrostomies was the usage of thrombin irrigation into the kidney. Because Margaretten et al. [9] reported the induction of cortical necrosis in pregnant rat by intravenous infusion of thrombin, we could not exclude the possibility that part of thrombin might have

entered into the systemic circulation and activated the clotting system with resultant cortical necrosis.

We should be alert to the fact that diuresis subsequent to resolving urinary obstruction could be followed by acute renal parenchymal damage if nephrostomy was done in a patient with DIC or in a pre-DIC condition.

Fig. 2. Gross section: right kidney was slightly smaller than the left and its whole cortex was yellowish-white, sharply demarcated from the medulla, showing cortical necrosis.

Discussion

When the patient was admitted, there did not seem to be any apparent causes for inducing cortical necrosis. The clotting study examined after right nephrostomy, however, revealed a drop in platelet count and an increase in serum FDP, despite normal values for PT, APTT and fibrinogen. This finding is consistent with a laboratory picture in patients with chronic DIC [5], which may occur in cases of disseminated cancer. Since the first nephrostomy (the right side) caused a fairly large amount of bleeding, this could have triggered the evolution of DIC. It is possible, therefore that the kidney temporarily excreted urine following nephrostomy and was subsequently affected by intraglomerular fibrin thrombosis, leading to anuria.

The contralateral kidney (the left side) was still hy-dronephrotic at this stage (the right perinephrostomy period), and therefore could be protected from the DIC involvement. This was proven by the fact that the left kidney excreted urine subsequent to the ipsilateral nephrostomy. But because the patient was in a DIC state, the kidney was again likewise involved by DIC and ceased functioning.

The problem is that cortical necrosis occurred only in the right kidney but not in the left, although the latter, too, was affected by intraglomerular fibrin thrombi. This dis-

References

- Watchi JM, Altman J: Unilateral cortical necrosis of the kidney. *Arch Path* 1970;90:125–128.
- Blau EB, Dysart N, Fish A, et al: Unilateral renal cortical necrosis. *Am J Dis Child* 1971;122:31–33.
- Halleraker B, Høisæter P: Unilateral renal cortical necrosis with contralateral hydronephrosis. *Scan J Urol Nephrol* 1974;8:77–80.
- Solez K: Acute renal failure; in Heptinstall (ed): *Pathology of the Kidney*. Boston, Little Brown, 1983, chap 21.
- McKay DG: Clinical significance of intravascular coagulation; in disseminated intravascular coagulation. Tokyo, University of Tokyo Press, 1983, pp 63–78.
- Palmerio C, Ming SC, Frank E, et al: The role of the sympathetic nervous system in the generalized Schwartzman reaction. *J Exp Med* 1962;115:609–612.
- Good RA, Thomas L: Studies on the generalized Schwartzman reaction. IV. Prevention of the local and generalized Schwartzman reaction with heparin. *J Exp Med* 1953;97:871–888.
- Muller-Berghaus G, McKay DG: Prevention of the generalized Schwartzman reaction in pregnant rats by α -adrenergic blocking agents. *Lab Invest* 1967;17:276–280.
- Margaretten, W, Zunker, HO, McKay DG: Production of the generalized Schwartzman reaction in pregnant rats by intravenous infusion in thrombin. *Lab Invest* 1964;13:552–559.