Spontaneous Perirenal Hematoma due to Periarteritis Nodosa

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Dear Sir,

Spontaneous perirenal hematoma (SPH) of the kidney is a rare entity and SPH of both kidneys is even more unusual. In this letter we present a patient who underwent bilateral nephrectomy 1 month apart due to retroperi-toneal hemorrhage as a result of kidney rupture.

A 50-year-old white female was referred to Ege University Hospital because of progressive renal insufficiency, and hypertension. Her previous history revealed intermittent skin eruptions of her upper extremities and trunk since 4 years. Physical examination showed a blood pressure of 150/110 mm Hg, and mild tenderness in right hypochondrium and loin. Hematocrit (Htc) 40%, blood urea nitrogen (BUN) 70 mmol/l (175 mg/dl), creatinine 222 µmol/l (2.52 mg/dl), ESR 26 mm/ h, urine protein (+ + +), and 10-15 RBCs in urine sediment. While these investigations were being completed, the patient suddenly developed agonizing right flank pain and went into shock. A rapidly growing mass in the right loin was palpated. Upper abdomen ultrasonogram (US) and computerised tomo-gram (CT) revealed a perirenal mass having liquid ecogenity. The patient was immediately operated. At operation, a large retroperi-toneal hematoma was found and continuous bleeding from lacerations on the kidney surface which could be controlled only by right nephrectomy. Light microscopy showed fi-brinoid necrosis of the small and medium-sized arteries with numerous neutrophils in and around the walls (fig. 1). Several aneurysms and hematomas were also present. The majority of the glomeruli had crescents. The overall picture suggested a necrotizing vasculitis, especially periarteritis nodosa (PAN). Postoperative laboratory analysis displayed: Htc 21%, BUN 71.6 mmol/l (179 mg/dl), creatinine 222 µmol/l (2.52 mg/dl), ESR 26 mm/ h, urine protein (+ + +), and 10-15 RBCs in urine sediment. While these investigations were being completed, the patient suddenly developed agonizing right flank pain and went into shock. A rapidly growing mass in the right loin was palpated. Upper abdomen ultrasonogram (US) and computerised tomo-gram (CT) revealed a perirenal mass having liquid ecogenity. The patient was immediately operated. At operation, a large retroperi-toneal hematoma was found and continuous bleeding from lacerations on the kidney surface which could be controlled only by right nephrectomy. Light microscopy showed fibroplastic necrosis of the small and medium-sized arteries with numerous neutrophils in and around the walls (fig. 1). Several aneurysms and hematomas were also present. The majority of the glomeruli had crescents. The overall picture suggested a necrotizing vasculitis, especially periarteritis nodosa (PAN). Postoperative laboratory analysis displayed: Htc 21%, BUN 71.6 mmol/l (179 mg/dl), creatinine 222 µmol/l (2.52 mg/dl), HBsAg (-), ANA (-), anti-DNA (-). Chest and sinus radiographies were normal. In skin biopsy specimen perivascular lymphocyte infiltration in addition to C4q accumulation in der-moiidermal junction was observed. Methyl-prednisolone 1 g i.v./day together with cyclo-phosphamide 500 mg i.v./week as pulse therapies were begun. Her clinical condition improved gradually.

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On postoperative day 15, there was again a sudden onset of agonizing left loin pain followed by profound hypotension necessitating blood transfusions. Some bloody urine was passed followed by profound anuria. US and CT revealed a left perirenal mass. Upon opening the abdomen a similar picture as at the first operation was observed and nephrectomy was again inescapable. Histologic examination of the specimen displayed exactly the same findings as the previous ones. Regular hemodialysis treatment was started in addition to combined immunosuppressive regimen consisting of 60 mg/day prednisolone and 50 mg/day cyclophosphamide. The patient gradually improved and was discharged from the hospital.

SPH is generally caused by the rupture of intrarenal aneurysms formed by vasculitis. US and CT can detect the site of perirenal hematoma, but provide no information on the cause of it. On the other hand, angiography discloses arterial wall irregularities, aneurysms and hypoperfusion distal to aneurysms which are rather characteristic but not pathognomonic for PAN [1]. Therefore histologic examination of renal tissue remains the only way to ascertain the diagnosis of classical PAN. Additionally, the optimal management of SPH due to PAN is yet unclear. Combined therapy of corticosteroids and cyclophosphamide appears to have a better survival rate as compared to that surgical and medical therapies provide alone [2]. But to find the optimal dosage of immunosuppressive drugs is a challenge, since adequate monitoring methods are unavailable.

Therefore we conclude that relying alone on the widely accepted but still empirical drug dosages may sometimes be deceptive in regard to adequate immunosuppression, and that close follow-up of the patients is still essential to reduce the rate of fatal outcomes.

References

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