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# Spontaneous Bilateral Renal Subcapsular Hematoma as a Possible Complication of Myeloproliferative Disorders

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A 64-year-old male had bilateral renal subcapsular fluid accumulation evident on abdominal sonography performed during postoperative follow-up after surgery for a hepatoma. There were no clinical symptoms, and he had no history of trauma or receiving anticoagulant medication. Spontaneous bilateral renal subcapsular hemorrhage was diagnosed after a series of examinations. He did have an underlying myeloproliferative disorder (MPD), and had received incomplete cytoreductive treatment. Platelet aggregation in MPD is often abnormal, and both bleeding and hypercoagulation complications have been reported. Here we report a rare case of MPD with possible thrombohemorrhagic complications presenting as spontaneous bilateral renal subcapsular hematomas.

Key words: renal hemorrhage, myeloproliferative disorder, thrombohemorrhagic complication

## INTRODUCTION

Lesions involving the perinephric spaces include primary and secondary solid neoplasms and fluid collections[1]. Perinephric fluid collections may be blood, pus, urine, lymph, exudates or transudates resulting from abnormalities within the kidney or adjacent retroperitoneal structures. Spontaneous renal subcapsular or perirenal hematoma is a relatively uncommon but often diagnostically challenging condition. The most common causes for spontaneous renal subcapsular hematoma are malignant and benign tumors, vascular diseases, infections, nephritis and blood dyscrasias. Here we report a case of asymptomatic bilateral perinephric fluid accumulation subsequently proved to be spontaneous renal subcapsular hematomas. Our patient did not take any anticoagulant medication and had no history of trauma. After other causes of spontaneous renal hematoma were reasonably excluded, the most probable reason for his hematomas was that he had an MPD of the essential thrombocythemia (ET) subtype. Platelet aggregation tests showed defective platelet aggregation in response to the agonists adenosine-5′-diphosphate (ADP) and ristocetin, indicating platelet dysfunction, which might be responsible for the development of the spontaneous bilateral renal subcapsular hematomas.

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## CASE REPORT

The patient was a 64-year-old male with hepatitis B virus-related liver cirrhosis, child-Pugh class A, and type 2 diabetes mellitus. Hepatoma and an angioma of the spleen were found in 2002, and he underwent enucleation of the liver (segment 7) and splenectomy. In addition, an MPD of the ET subtype was proved by bone marrow biopsy in 2005. He was treated with incomplete hydroxyurea cytoreductive therapy for three months. Bilateral fluid collection in the perirenal space was incidentally noted in follow-up abdominal sonography a few days before this admission in June 2006.

On admission, there was no clinical symptom of arthralgia or abdominal pain. His vital signs were normal, and mental status was intact. A full blood count showed a hemoglobin concentration of 11.4 gm/dl, an elevated white cell count of $29\times10^9$ cells/l and a platelet count of $1222\times10^9$/l. Biochemical tests, including renal function and liver function, were unremarkable, but positive hepatitis B surface antigen serology. Urinalysis showed no hematuria or pyuria. The coagulation profile revealed normal prothrombin and activated partial thromboplastin times. Platelet aggregation tests showed aggregation ab-

normalities with decreased aggregation by ADP at 20 μM and an absent platelet response to ristocetin at 0.5-1.0 mg/ml.

Abdominal computed tomography (CT) revealed a heterogeneous lesion of about 8×5×10 cm over the lateral aspect of the right kidney with compression (Fig. 1), and a large amount of fluid accumulation in the left subcapsular space with extrinsic compression of the left kidney. Sono-guided percutaneous drainage of the subcapsular fluid was performed. The fluid was clear and sterile with low concentrations of triglyceride, cholesterol, blood urea nitrogen and creatinine. A high red blood cell count was noted, but cytology showed no malignant cells. The impression of bilateral subcapsular hematomas of the kidney in the process of resolving was favored. A renal arteriogram was arranged for further evaluation, but no significant finding was noted.

Left percutaneous catheter drainage was continued for 10 days and a total of 7000 ml of fluid was drained away, and he also received hydroxyurea cytoreductive therapy at our clinic after he was discharged. Follow-up abdominal sonography and blood examinations were improved, and he was uneventful.

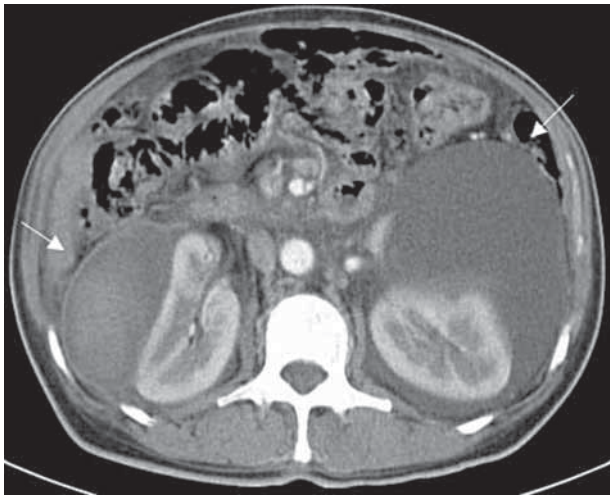

Fig. 1 Axial computed tomography of abdomen showed resolving subcapsule hematoma over the right lateral aspect of the right kidney about 8×5×10 cm, with compression (arrow). In addition, a large amount of pure clear fluid accumulation in the left perirenal space was noted. The fluid also caused extrinsic compression of the left kidney (arrow).

## DISCUSSION

The subcapsular area of the kidney is a potential space where fluid can accumulate, causing compression of the renal parenchyma. The perinephric fluid may consist of pus, urine, blood, lymph, exudate or transudate. Bilateral renal subcapsular hematomas was considered after excluding the possibilities of urine, infection, and lymph by the drainage fluid cytology results.

Renal subcapsular hematoma is defined as renal parenchymal bleeding with dissection of the blood into the subcapsular space. In the diagnosis of renal subcapsular hematoma, ultrasound findings are usually non-specific, and CT scan is a better investigation for the evaluation of a perinephric hemorrhage. Acute hematoma has a higher attenuation value than the renal parenchyma on the unenhanced CT scan, and a lower attenuation value than normal enhancing renal parenchyma on contrast-enhanced CT scan. With time, there is liquefaction of the hematoma with a decrease in its attenuation value approaching the density of water. Angiography is generally more useful in the evaluation of vascular diseases that are associated with spontaneous renal hemorrhage, and diagnostic angiography can be followed by therapeutic embolization of the bleeding vessels in suitable cases during the same procedure.

The most common causes of spontaneous subcapsular hematoma include renal carcinoma followed by angiomyolipoma and vascular disease, of which periarteritis is the most common[2]. Selective renal angiography is mandatory to rule out vascular lesions, such as periarteritis, arteriosclerotic aneurysms, arteriovenous malformations, renal infarction or small renal carcinoma not demonstrated on the CT scan[3]. In our case, abdomen CT showed typical characteristics of renal subcapsular hematoma with no apparent etiology. Further renal arteriogram demonstrated no tumors, intrarenal aneurysms, thromboses, or wedge-shaped filling defect. The possibilities of bilateral coincidental renal tumor bleeding, hepatitis B-related polyarteritis nodosa with renal hemorrhage[4] or renal infarction were less likely. Besides, the spontaneous renal subcapsular bleeding blamed on liver cirrhosis-related coagulopathy was not considered in this case because of normal coagulation profile. The diagnosis of spontaneous bilateral renal subcapsular hematoma in the process of resolving is favored in this case according to the findings of computed tomography, angiography and drainage fluid cytology.

The most possible explanation for the bilateral spontaneous renal subcapsular hematomas in this case was they were caused by the MPD. He had received inadequate cytoreductive treatment, and platelet aggregation tests

showed decreased aggregation by ADP at 20 μM and an absent platelet response to ristocetin at 0.5-1.0 mg/ml. The MPDs are a group of clonal bone marrow stem cell diseases, characterized by qualitative as well as quantitative abnormalities in the various blood elements. Several platelet function defects have been described in MPDs[5], including impaired aggregation. In study by Zeidman[6], 79% of their patients had platelet aggregation abnormalities, compared to 20% in the control group, and 50% demonstrated reduced ADP-induced platelet aggregation, while ristocetin resulted in decreased platelet aggregation in 18% patients. In Wehmeier et al.[7], a retrospective study analyzing vascular complications in 260 patients with MPD, 126 patients had bleeding or thrombotic events, but no cases of perinephric or renal subcapsular bleeding.

Extrahematological features associated with myeloproliferative syndrome presented as bilateral perirenal hemorrhage is rare[8]. Here we reported a case of MPD with abnormal platelet aggregation presenting with asymptomatic spontaneous bilateral renal subcapsular hematomas, which has seldom been previously reported as a thrombohemorrhagic complication of MPD.

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