Resolution of page kidney-related hypertension with medical therapy: A case report

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A 24-year-old man developed systolic hypertension as a result of renal contusion, perinephric fluid collection, and renal compression (Page kidney) after blunt renal trauma. The patient was treated with an angiotensin-converting enzyme inhibitor for 30 days, after which his blood pressure normalized and the medications were discontinued. Follow-up clinical and laboratory examinations at 3, 6, and 12 months showed normal results. Transient hypertension may develop in patients after blunt renal injury as a result of Page phenomenon. Appropriate medical therapy is warranted and may be successful because spontaneous resolution may be delayed or not occur at all. (Heart Lung® 2007;36:377–379.)

Hypertension secondary to blunt renal injury is an uncommon complication of abdominal trauma, especially if observed within a short period of time after abdominal trauma.[1] Mechanisms involved include renal vascular damage, renal parenchyma compression, and contusion (Page kidney) leading to renal ischemia and increased renin production.[2] Hyperreninemia is the major pathogenetic mechanism of hypertension in these patients, which is caused by renal hypoperfusion and thus renal ischemia.[3] The duration of hypertension varies from transient to chronic and is related to the reversibility of the cause.[3] However, Page kidney is often associated with the worst outcome, especially in patients requiring surgical interventions.[2,3] We report on a patient with severe systolic hypertension after blunt abdominal trauma who was effectively treated with medical therapy until spontaneous resolution was observed.

CASE REPORT

A 24-year-old previously healthy man was admitted to the hospital after being involved in a car accident. He had abdominal pain, distension, tenderness, and macroscopic hematuria. He was hemodynamically unstable (systolic blood pressure [SBP] of 80 mm Hg, heart rate of 120 beats/min, diaphoresis, hematocrit of 24.9%, and platelet count of 113,000/mm^3). He had no rib, long bone, spine, or pelvic fractures on clinical and radiography examination. Intraperitoneal lavage was positive for intra-abdominal bleeding. He underwent emergency surgery for abdominal exploration, which revealed left lobe liver injury and mesentery rupture requiring suture repair. Postoperatively, he was admitted to the intensive care unit (ICU) for a mild closed head injury. He was in stable condition and placed on mechanical ventilation. The patient became hemodynamically stable; the electrocardiogram, chest radiography, and arterial blood gases were normal, and there was gradual stabilization of the hematocrit, platelets, coagulation screening, and liver function tests. He had normal urine output and renal function test results, but he continued to have mild microscopic hematuria and a moderate amount of fluids from the right perinephric drain. He had no renal parenchyma and vascular damage on imaging studies for both kidneys (ultrasound and computed tomography, including two sequential intravenous pyelographies, Fig 1).

While the patient was in the ICU (eighth day), severe systolic hypertension developed (SBP 210 mm Hg, diastolic blood pressure [DBP] 80 mm Hg)

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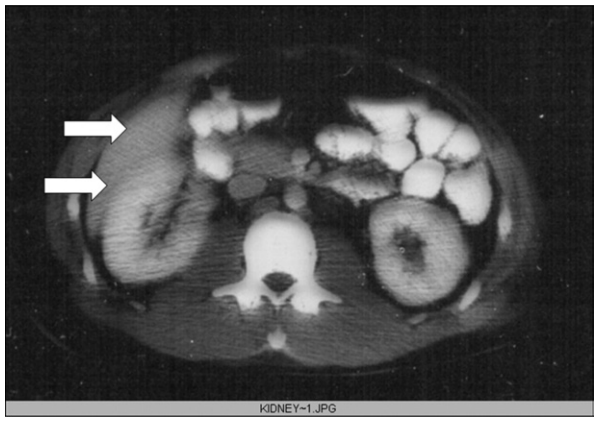

Figure 1 Computed tomography of the abdomen. Perinephric fluid collection and renal compression (*white arrows*). *ICU,* Intensive care unit; ACE, angiotensin-converting enzyme.

without significant tachycardia (heart rate 85-100 beats/min) and mild hyponatremia (range 129-134 mmol/L). His electrocardiogram was normal, and creatine phosphokinase, alanine aminotransferase, aspartate aminotransferase, and lactate dehydrogenase levels were within normal range. Sequential troponin test results were negative, and cardiac echocardiography studies revealed normal ventricular function. Thus, myocardial contusion, infarction, and ischemia were ruled out. The patient had no signs of elevated intracranial pressure that could explain the hypertension after a head injury due to Cushing's reflex, and his initial head computed tomography showing mild cerebral edema was significantly improved to resolution. Also, there were no signs of inappropriate antidiuretic hormone secretion, such as hypervolemia and hyponatremia, or of diabetes insipidus, such as polyuria and severe hyponatremia. At first he was given clonidine intravenously in bolus doses (50-100 μg in 10 mL NaCl 0.9%) and then in continuous intravenous infusion (at the highest recommended doses, 600 μg in 500 mL dextrose in water 5% for continuous infusion for 24 hours up to 750 μg daily) for 24 hours, but he was unresponsive. He was then given an angiotensin-converting enzyme inhibitor, captopril, at first sublingually (25 mg) and then enterally 25 mg four times per day with significant response (SBP 160 mm Hg, DBP 70 mm Hg). A beta-blocker was added (propranolol hydrochloride, 20 mg three times/day enterally for 9 days). The patient was extubated on day 12 and had normal cerebral function (Glasgow Coma Scale 15, normal head computed tomography), and normal gastrointestinal, liver, and renal function. His S_{AO_2} was 99%, and P_{AO_2} with a supplemental 3 liters per minute of O_2 through nasal prongs was 87 mm Hg. He had no signs and symptoms of tachycardia or diaphoresis observed in sympathetic stimulation (dysautonomy) after head trauma. He continued to have high blood pressure controlled by captopril alone (25 mg four times/day, SBP 140 mm Hg, DBP 70 mm Hg) until his discharge on day 24 when he was transferred to the surgical ward and was given captopril 25 mg four times per day by mouth. Captopril was discontinued on day 38 (after 30 days). The patient was discharged from the hospital after 51 days of hospitalization in stable condition and with normal blood pressure. Follow-up at 3, 6, and 12 months revealed his blood pressure to be 120/75 mm Hg with normal clinical examination results. Renal imaging studies and laboratory findings (urea, creatinine, and urinalysis) were normal. Fig 2 shows the time frame of hypertension development after ICU admission, management with medical therapy, spontaneous resolution, and follow-up.

DISCUSSION

Posttraumatic renovascular hypertension may present several days to years[1] after a blunt or penetrating renal injury,[1,2] which may be spontaneously

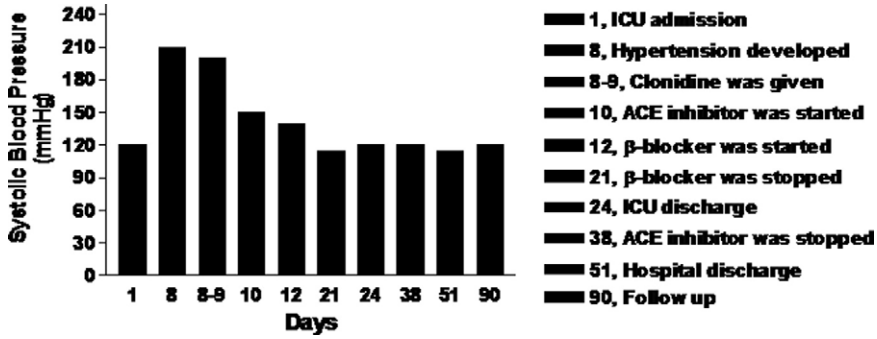

Figure 2 The time frame of hypertension development, management, and spontaneous resolution.

resolved after a variable duration of time.[2] Types of renal injury related to hypertension include[3] renovascular trauma (renal artery stenosis, occlusion, or external compression), severe external renal parenchyma compression (Page phenomenon–Page kidney), and arteriovenous fistula, all of which lead to overall reduction in renal blood flow and renal ischemia, which is the primary stimulus for increased renin production[4] and secondary hypertension. Secondary hypertension at any age should alert physicians to a previous trauma and to consider an undiagnosed renal injury as a possible cause.[4]

Our patient may have developed the "Page phenomenon,"[4,5] which is renal parenchyma compression (perinephric fluid collection) with concomitant renal parenchyma contusion (initially gross and then microscopic hematuria) leading to renal ischemia, increased renin production, and hyperreninemia-induced hypertension. He had normal renal function test results (urea, creatinine, and glomerular filtration rate), normal kidney imaging studies with perinephric fluid collection, mild hyponatremia, and systolic hypertension responsive to an angiotensin-converting enzyme inhibitor, findings that are closely related to increased renin production. We speculate that subsequent renal compression was the main mechanism of hypertension because renal parenchyma contusion was present from the beginning of the ICU admission, but hypertension was observed on the eighth day of hospitalization and the perinephric drainage was positive. However, we should keep in mind that the patient was under sedation for the first 4 days.

The gold standard for the diagnosis of "Page kidney" is selective renal arteriography to exclude renovascular lesions and renal-vein renin assays[3] to confirm hyperreninemia, tests that were postponed for our patient because of his significant clinical improvement after medical therapy and the absence of evidence of vascular damage from imaging studies. Appropriate surgical therapy[2,3] for Page kidney is nephrectomy, drainage, and renal decapsulation. However, close observation and appropriate medical therapy are warranted[3] for a reasonable period of time before surgical treatment because spontaneous resolution may occur. Medical therapy alone may be successful in less severe cases of renal compression-ischemia with mild hyperreninemia, as in our case.

CONCLUSION

Posttraumatic renovascular hypertension after blunt abdominal trauma is an uncommon complication,[1] and this diagnosis is based only on a high index of suspicion.[3] The major pathogenetic mechanism is severe hyperreninemia caused by renal ischemia, which may be associated with poor outcome in severe cases. However, the condition requires close monitoring and timely selection of appropriate treatment. Clinicians, ICU specialists, and consultants should be aware of this rare syndrome in patients with blunt renal injury and excess intra-abdominal and perinephric fluid collection for early recognition of hypertension and selection of appropriate treatment.

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