**Left kidney: an unusual site of cocaine-related renal infarction. A case report**

F. FABBIAN, M. PALA, A. DE GIORGI, R. TISEO, C. MOLINO, A. MALLOZZI MENEGATTI, F. TRAVASONI, E. MISURATI, F. PORTALUPPI, R. MANFREDINI

Institute of Clinical Medicine, School of Medicine, University of Ferrara, Ferrara (Italy)

**Abstract.** Acute renal infarction is a well known, although relatively unfrequent, cause of flank pain resistant to administration of spasmyotic and nonsteroidal anti-inflammatory drugs.

We present an original case of a 41-year old man, complaining of acute severe left flank pain, resistant to common analgesic therapy, who was diagnosed of segmental renal infarction of a branch of left renal artery. Pathophysiology of renal damage in cocaine users is multifactorial, and it has been postulated that the right kidney was more prone to ischaemia. Left kidney represents an extremely unusual site of cocaine-related renal infarction.

**Key Words:** Renal infarction, Cocaine-related diseases.

**Introduction**

Although usually the symptom pain is managed on an outpatient basis, the number of patients presenting to the Emergency Department (ED) because of back or flank pain is high. Acute renal infarction is a well known, although relatively unfrequent, cause of flank pain resistant to administration of spasmyotic and nonsteroidal anti-inflammatory drugs. Usually it presents with nonspecific symptoms, and diagnosis may be often delayed, but with the improvement in computed tomography (CT) in the recent years, this examination has a reliable accuracy in the differential diagnosis between flank pain due to renal infarction and abdominal pain due to different conditions. The majority of patients suffering of renal infarction have also cardiac diseases, and renal infarction is usually due to acute embolic occlusion of an end-artery vessel. Atrial fibrillation, valvular heart disease, dilated cardiomyopathy, and myocardial infarction are established risk factors for renal infarction. Cocaine abuse is frequent, and its renal toxicity is well known, besides it could be a cause of renal infarction. We report here a case of a patient who presented a left renal infarction, but only after some days of hospital stay admitted his cocaine abuse.

**Case**

A 41-year old man was hospitalized complaining of acute severe left flank pain, resistant to common analgesic drugs. His medical history included non-dipping arterial hypertension, treated with thiazides, and smoking habit. On physical examination: arterial blood pressure 150/95 mm Hg, axillary temperature 36.5°C (95.9°F), regular heart beat 72 beats/min, with no heart murmur. The patient had no nausea or vomiting, examination of the chest was unremarkable, and there was tenderness at the upper left quadrants of the abdomen. Electrocardiogram did not show significant alterations. Laboratory examinations showed normal renal function (glomerular filtration rate 78 ml/min/1.73 m²), white blood cells 5.07 × 10³/µl, serum lactate dehydrogenase (LDH) 259 U/L, and presence of antiphospholipid antibodies. On urinalysis haemoglobin 2+, and albuminuria 255 mg/24 hour. Abdominal ultrasound was normal, but abdominal computed tomography revealed the presence of a segmental renal infarction of a branch of left renal artery (Figure 1). Only several days after admission he told doctors he was alcohol and cocaine (nasal route) abuser. The patient was treated with cal-
abusers. In Italy, 4.6% of people aged between 15-64 years reported to be (at least once during lifetime) cocaine abusers\(^5\). Thus, even if nephropathy due to cocaine abuse represents an uncommon condition, the high number of cocaine abusers could make this condition relatively frequent in emergency settings dealing with renal pain. Rhabdomyolysis, and its life-threatening complications, such as acute renal failure, is not so rare in cocaine abusers, and it has been suggested that cocaine nephropathy should be considered a cause of end-stage renal disease\(^6\). In cocaine users, pathophysiology of renal damage is multifactorial, and seems to be due to hemodynamic changes, glomerular matrix synthesis and degradation, and oxidative stress leading to ischemia\(^7\).

Based on the excellent table by Madhrira et al\(^8\), in the present updated version (Table I) we report 12 cases of cocaine-related renal infarction\(^11-20\). We decided to exclude the reports by Antonovych et al\(^21\), reported in an abstract published in 1990, dealing with 2 cases with “unilateral” renal infarction (not more precisely defined) with no mention of route of cocaine assumption. Out of the total 12 cases (10 men), 8 had a right kidney infarction, 2 had a bilateral involvement, and 2 (including the present case) had a left infarction. As for gender predilection, one possible explanation might be that cocaine use is prevalent in males. In fact, renal infarctions secondary to thromboembolic or atherosclerotic causes have been described in both sexes\(^2,22-25\), as well as idiopathic cases\(^25,27\). As for site of infarction, the right kidney is highly preferred. In their interesting paper, Bemanian

**Table I.** Sites of cocaine-related renal infarction.

<table>
<thead>
<tr>
<th>Case</th>
<th>Kidney involved</th>
<th>Sex</th>
<th>Route of cocaine</th>
<th>Year of publication (ref)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bilateral</td>
<td>M</td>
<td>Nasal</td>
<td>2008(^{11})</td>
</tr>
<tr>
<td>2</td>
<td>Bilateral</td>
<td>M</td>
<td>Nasal</td>
<td>2009(^{10})</td>
</tr>
<tr>
<td>3</td>
<td>Right</td>
<td>M</td>
<td>L.V.</td>
<td>1984(^{12})</td>
</tr>
<tr>
<td>4</td>
<td>Right</td>
<td>M</td>
<td>L.V.</td>
<td>1987(^{13})</td>
</tr>
<tr>
<td>5</td>
<td>Right</td>
<td>M</td>
<td>L.V.</td>
<td>1993(^{14})</td>
</tr>
<tr>
<td>6</td>
<td>Right</td>
<td>M</td>
<td>Nasal</td>
<td>1995(^{15})</td>
</tr>
<tr>
<td>7</td>
<td>Right</td>
<td>M</td>
<td>Nasal</td>
<td>2001(^{16})</td>
</tr>
<tr>
<td>8</td>
<td>Right</td>
<td>F</td>
<td>Nasal</td>
<td>2003(^{17})</td>
</tr>
<tr>
<td>9</td>
<td>Right</td>
<td>M</td>
<td>Nasal</td>
<td>2005(^{18})</td>
</tr>
<tr>
<td>10</td>
<td>Right</td>
<td>M</td>
<td>Not reported</td>
<td>2004(^{19})</td>
</tr>
<tr>
<td>11</td>
<td>Left</td>
<td>M</td>
<td>Intestinal transport</td>
<td>2007(^{20})</td>
</tr>
<tr>
<td>12</td>
<td>Left</td>
<td>M</td>
<td>Nasal</td>
<td>Present case</td>
</tr>
</tbody>
</table>

**Figure 1.** Abdominal computed tomography: segmental ischemia of a branch of left renal artery.
et al. provided an elegant hypothesis to explain such preference. Based on Poiselle equations on fluids and vessels, since the radius of the right and left renal arteries are similar in size, they postulated that the right kidney might be more prone to ischaemia, due to the increased resistance that it encounters by the longer length of its artery due to the longer length of its artery. However, this seems to be valid only in the case of cocaine-related renal infarction, since in the general population it was not possible to detect a clear preference for the right or the left kidney.

In our patient, on one hand many of the classical biochemical signs of renal infarction, e.g., white blood cells and LDH, were lacking. On the other, we detected the presence of antiphospholipid antibodies, a finding reported since in classical biochemical signs of renal infarction, e.g., white blood cells and LDH, were lacking. On the other, we detected the presence of antiphospholipid antibodies, a finding reported since 1991 in cocaine abusers, and provided with predictive value for ischaemic disease. To the best of our knowledge, this is the second case in literature of left kidney localization of a cocaine-related renal infarction. Thus, although an evident preference for right kidney is reported, the possibility that also the left kidney may be interested by infarction should be considered in cocaine abusers as well. In patients who present to the ED with abdominal or flank pain resistant to treatment, independent of the site of pain, renal infarction must be considered, especially if they have thromboembolic risk factors.

Acknowledgements

Supported, in part, by a scientific Grant from the University of Ferrara (FAR – Fondo Ateneo Ricerca –).}

References


