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Acute kidney injury during anticoagulant treatment

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ABSTRACT

Acute kidney injury represents a medical emergency with high mortality rate even with proper medical care; the survival chances are higher if the diagnosis is early established and an adequate treatment administered. We present the case of a patient with acute kidney failure induced by post-renal mechanism, in the context of a chronic anticoagulant treatment, with a particular development due to bilateral kidney involvement.

KEYWORDS acute kidney injury, anticoagulant treatment, urinary blood clots

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Introduction:
Acute kidney injury (AKI) was first defined as an acute loss of kidney function characterized by metabolites retention and important water-electrolyte and acid-base imbalances; in 2004 were published RIFLE criteria (Risk, Injury, Failure, Loss, and End stage disease) for staging and diagnosis of acute kidney lesions [1]. The classification include three degrees of severity and two possible complications considering the urine output and glomerular filtration rate. These criteria were later completed and modify by Acute Kidney Injury Network (AKIN) with less severe injury elements; there is also recommended to avoid using the glomerular filtration rate as a single marker in AKI since there is no dependable way to measure glomerular filtration rate [2,3]. In 2012 the Kidney Disease Improving Global Outcomes (KDIGO) released their clinical practice guidelines for acute kidney injury (AKI), which build off of the RIFLE criteria and the AKIN criteria, The KDIGO consensus classification has yet to be validated.[4]

Acute kidney injuries may be divided (based on the apparition mechanisms) in pre-renal injury (caused by important reduction in renal perfusion), renal injury (caused by intrinsic kidney lesions) and post-renal injury (caused by obstacles of the urinary tract). The post-renal causes are the rarest, inducing under 5% of all cases of AKI – a possible argument for the late diagnosis of these cases.

We will present the case of a patient with AKI installed by postrenal mechanism, a case associated with diagnostic and therapeutic management problems.

Case study:
We present the case of a 41 year-old male, with chronic VHC hepatitis, diagnosed two years before with with thrombosis of a superior mesenteric artery branch (under treatment with acenocumarolum 1mg daily); currently the patient received 4 days also ibuprofenum (400mgx4) and chlorzoxazonum (250mgx3) for a muscle contracture.

The patient arrived at the hospital, presenting colicative pain in the left flank, haematuria and nausea.

Day 1: Biologically he had moderate inflammatory syndrome (ESR 80 mm/h, fibrinogen 693 mg/dl), minor thrombocytopenia (150000/cmm), hypochromic microcytic anemia (Hb 9.4 g/dL), indirect bilirubin 0.3 mg/dL, direct bilirubin 0.5 mg/dL, slight elevation of serum creatinine (1.3 mg/dL) and urea (46mg/dL) and a high prothrombine time (24.6 s); an urine culture test was also recommended.

In order to exclude/confirm an eventual left renal lithiasis was performed an ultrasound exam, which revealed presence of left renal microlithiasis, without dilatation of calyceal system or left ureter.

The patient received spasmolytic and analgesic treatment: ondasetronum i.v. 4mgx3, drotaverine hydrochloride i.v. 2mLx3 (40mg/mL), paracetamolum i.v. 100mLx3 (10mg/mL) and ranitidine hydrochloride 2mLx3 (25mg/mL) with slight amelioration of the symptomatology.

Day2: the patient was transferred in our clinic for further treatment and investigations.

Biologically: persistent inflammatory syndrome (ESR 69 mm/h, fibrinogen 641 mg/dl), minor thrombocytopenia (143000/cmm), hypochromic microcytic anemia (Hb 9.1 g/dL), indirect bilirubin 0.4 mg/dL, direct bilirubin 0.6 mg/dL, increased serum creatinine (1.5 mg/dL) and urea (49mg/dL) and a high prothrombine time (26.1 s); results for the urine culture test (received form the initial hospital) were negative.

We repeated the ultrasound exam and found:
- normal aspect of the right kidney

- left calyceal dilatation II/V degree, with increased ecogenity of the calyceal system, suggesting a solid/tisular content (for which we performed a CT scan later same day)

CT scan revealed:
- left kidney with diminished perfusion and absence of excretion; enlarged calyceal system with blood densities on the unenhanced scan(Fig. 1)

- right kidney with normal perfusion and excretion, slightly enlarged calyceal system and presence of a lacunary image in the renal pelvis; the volumetric endoureteral reconstructions shows a protrusive image in the right renal pelvis (blood cloth) (Fig. 2)

Two hours after the CT scan, on the plain radiography we observed the lacunary image in the right renal pelvis and lack of left renal excretion; no particularities were detected in the right ureter and the bladder.

The patient received plasma for normalization of coagulation times (acenocumarolum treatment was stopped) and was performed hemodialysis on central venous catheter in order to correct the metabolic acidosis (pH 7.31, HCO3 17 mEq/L), hyperpotassemia (5,6 mEq/L), the retention of fluids and to prevent the aggravation of renal insufficiency after iopromidum administration (during CT exam).

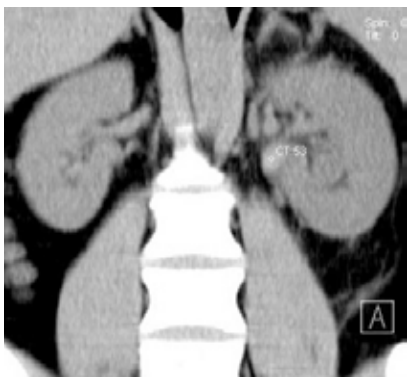

Fig. 1 – unenhanced CT scan – coronal reconstruction - blood-like densities in the left renal pelvis (UH=53)

Fig. 2 – CT – volumetric endoureteral reconstruction - protrusive image in the right renal pelvis and right ureter (blood cloth)

During the evening the patient installed anuria and presented colicative pains also in the right flank; an additional ultrasound exam was performed and it evidentiate an enlarged right caliceal system with increased ecogenity (Fig. 3), similar to the left kidney, suggesting bilateral blood cloths in the renal pelvis. Biologically was registered a higher serum creatinine (7.3 mg/dL) and urea (78mg/dL).

The patient was transferred into a urology clinic and after a second hemodialysis on central venous catheter, he underwent ureteral catheterization with aspiration of blood cloths, lavage and bilateral installation of Cook ureteral catheters, with favorable evolution.

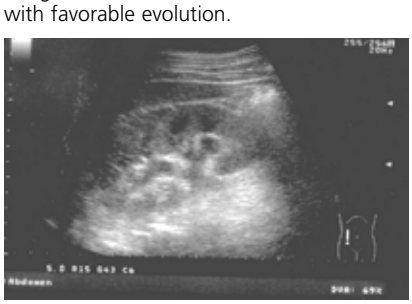

Fig. 3 – Right kidney (ultrasound), coronal image – enlarged calyceal system with increased ecogenity

Discussions:

Renal function is commonly evaluated by measuring the glomerular filtration rate (GFR), but serum creatinine does not reflect always accurately GFR in first stages of kidney failure, because it necessitates a certain time to reach a required diagnosis level; furthermore when GFR decreases, serum creatinine clearance may overestimate GFR by increasing the tubular secretion of creatinine – urinary creatinine is represented by the creatinine filtered at glomerular level and the creatinine secreted at tubular level (physiological the secretion of creatinine is very low, but when GFR is decreasing and its serum level increases, the tubular secretion of creatinine also increases). Based on these facts, in order to improve the diagnosis process, monitoring the urine output is essential.

AKI by postrenal mechanism is caused by obstacles of the urinary tract. Most causes that reduce renal excretion are chronic pathologies (prostate adenoma, bladder or prostate tumors, urotelial tumors, retroperitoneal fibrosis), causing a slow increase of the retrograde hydrostatic pressure, which leads to a chronic kidney injury (CKI).

In order to induce AKI, the obstacle must occur suddenly (trauma, urinary stone, blood cloth) and in case of a severe AKI (associated with high mortality rate) is mandatory that both kidneys are involved. In our case the patient presented a rare cause of bilateral blockage of urinary tracts – bleeding of the urotelial mucosa and cloth-blockage initially of the left kidney followed by the right kidney. An acute complete block in the urine excretion leads to a retrograde increase of the hydrostatic pressure in the calyceal system and the renal tubules with egalisation of the glomerular filtration pressure.

What caused in our patient the urinary bleeding and apparition of blood cloths followed by AKI?

First variant would we should consider in this case is a renal pathology (kidney stones, glomerulonephritis etc), that may induce haematuria, which in condition of anticoagulant treatment may increase greatly causing also apparition of urinary blood cloths. Although first ultrasound exam described left renal microlithiasis, it was not confirmed by the other ultrasound exams and the CT scan; also the left microlithiasis can't justify the blood cloths in the right calyceal system. Patient had no fever and the urine culture test was negative so we can exclude also an infectious cause (glomerulonephritis, pielonephritis) which could generate a haematuria.

Second variant we should consider in this case is an overdosage of the anticoagulant treatment – in our case the patient and its relatives confirmed that he followed the prescription and used only 1mg daily.

From all considered aspects we could find only one element which could explain the evolution of the pathology: the patient followed a treatment with nonsteroid anti-inflamatory drugs (ibuprofenum) for the muscle contraction, which could generate, in a patient undergoing anticoagulant treatment, haematuria and development of blood cloths in left renal pelvis; haematuria was accentuated and occurred also in the right side after administration i.v. of paracetamolum in first day of treatment.

Another aspect which should be discussed is what type of CT scan do we perform? The contrast medium can accumulate into the kidney and may aggravate an existent renal insufficiency, but the information it provides are essential in depicting the cause and if a haemodialysis can be performed, the CT scan should be with contrast medium – we can identify the thrombotic processes, eventual arterial stenosis (as pre-renal mechanism), inflammatory changes in the kidney parenchyma (as renal mechanism) or blocks of the urinary tract (postrenal mechanism)

Ultrasound exam has more a screening value in diagnosis of AKI by renal mechanism, providing limited information in prerenal and postenal causes. MRI is ideal in depicting the cause

– prerenal/renal/postrenal and can also provide information regarding the renal function (perfusion, secretion, excretion). In our case the patient had a metallic osteosinthesis material in the left leg (with no information about the type of metal) so MRI exam was excluded.

***Conclusion*:**

The particularity of the case is represented by the etiology of the AKI – bilateral calyceal blood-cloths with involvement of both kidneys, caused by association of nonsteroid anti-inflammatory medication and oral anticoagulants.

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