Introduction
Acute kidney injury (AKI) after endovascular aneurysm repair (EVAR) is present in 6.7% of patients. The etiology is unclear and multifactorial in the majority of cases [1]. Urinary obstruction due to ureteral entrapment is a rare cause of AKI after EVAR, which should be considered after inflammatory abdominal aortic aneurysms (IAAAs) treatment [2].

Case Presentation
An 81 year-old man was admitted in the emergency department with AKI (serum creatinine 4.4 mg/dl). He performed an ultrasound and a CT scan that showed a ruptured IAAA measuring 6.5 cm of maximum anteroposterior diameter and left hydronephrosis (Figure 1). He underwent non-elective endovascular repair of the IAAA with an aorto-uni-iliac graft Endurant® and femoro-femoral bypass. No complications were reported. After the intervention, his creatinine normalized (0.89 mg/dl). Two months later, he was readmitted with AKI (creatinine 8.1 mg/dl), without fever, abdominal or lumbar pain. The CT scan with contrast administration showed the graft in a suitable position, patency of the renal arteries, no endoleak. However retroperitoneal fibrosis and bilateral ureteric obstruction were present (Figure 2). A ureteric double J stent was inserted with subsequent renal function improvement (creatinine 2.3 mg/dl). Corticosteroids were not administered.

A CT scan was acquired 6 months after ureteral stenting, showing EVAR patency without endoleaks or fracture. The ureteral stents were in place and unobstructed. No hydronephrosis was found and renal function has remained stable.

Discussion
This case reports a rare cause of AKI after EVAR, which should be taken into account after IAAAs treatment.

AKI after EVAR is usually multifactorial [1]. The most frequent causes are: contrast nephropathy; renal injury caused by ischemia-reperfusion and renal artery occlusion, which can occur as a result of embolization, supra-renal endograft fixation and graft misplacement [1,3].

In the case reported in this paper, we excluded the hypotheses of contrast nephropathy and renal injury caused by ischemia-reperfusion based on the temporal relation between the kidney exposure to the aggressors and renal dysfunction.
Contrast media leads to hypoxia in the outer renal medulla, causing oxidative stress and the generation of oxygen free radicals [1]. In contrast nephropathy, renal function starts to deteriorate within 24 hours, peaking at about 3 to 5 days following contrast administration [1].

Ischemia-reperfusion injury is a consequence of ischemia limb time. During the EVAR, there is limb tissue hypoperfusion, with anaerobic metabolism, base deficit and neutrophils activation which produces renal damage after limb reperfusion [1]. In this case, the kidney function deteriorates in the immediate post-operative time [1].

AKI caused by renal artery occlusion was considered in this case. One of the mechanisms of renal artery occlusion is embolization, which occurs during guidewire use, sheaths and endograft manipulation. This may be a cause of AKI in 3-5% of EVAR patients [1,3]. In some cases, renal artery stenosis due to plaque embolization can be treated by catheterization and aspiration of the plaque [3]. In our case, the second episode of AKI was not preceded by any type of manipulation, so this etiology was rejected.

However, graft misplacement or migration with impairment of renal perfusion was our initial hypothesis, when the patient was re-admitted with kidney dysfunction. Occlusion of the renal artery immediately after the deployment of a stent graft occurs in 2-4% of cases and is more frequent in short neck aneurysms [3]. The occlusion may also occur later after aneurysm remodeling [3]. Renal artery occlusion caused by these mechanisms can be treated by “pulling-down” the endograft or inserting a bare stent in the renal artery [3].

Impairment of the renal artery perfusion was excluded in our patient with a CT scan. The CT scan also showed us the true etiology of AKI: ureteric obstruction. Due to the inflammatory and fibrotic behavior of the IAAA, ureteral entrapment should be ruled out as a cause of AKI after endovascular IAAA repair. In fact, hydronephrosis is present in 20% of patient with IAAAs [2].

In IAAAs, which represent 3-10% of all abdominal aortic aneurysms, the arterial wall is infiltrated by inflammatory cells and the elastic and muscular fibers of the media are replaced by fibrotic tissue [2,4]. As a result, there are perianeurysmal adhesions, which frequently involve the duodenum, inferior vena cava, left renal vein and the ureters [2].

The presence of adhesions increases the risk of iatrogenic injuries during open surgical repair (OSR), which could be an extra argument for the use of EVAR in this group of patients. Even so, it has not been proved in randomized controlled trials that EVAR is more advantageous than OSR in patients with IAAAs [2].

In a review which analyzed the outcome of IAAAs after OSR and EVAR, it was concluded that both options can be safely performed [2]. Stone et al. [4] demonstrated that EVAR should be considered as a first-line therapy in IAAAs. Despite this, peri-aortic inflammation does not recede completely after OSR or EVAR [2]. Stone et al. [4] found that the reduction in peri-aortic fibrosis is about 55% in EVAR. However, according to Puchner et al. [2], in 42% of patients who underwent EVAR there was no change in the inflammatory process and in 7% there was an inflammation increment. The resolution of hydronephrosis seems to be higher after OSR and is apparently a slow process after EVAR [4]. Moreover, the hydronephrosis progressively worsened in 21% of the patients in the EVAR group [4].

It is thought that the endografts material can increase the inflammation, even when used in the treatment of non-inflammatory aneurysms, due to a reaction to the graft material [4]. It is known as “post implantation syndrome” consisting of fever, leukocytosis and C-reactive protein increment. This means that in IAAAs there is an exacerbation of the natural inflammatory process, after the endovascular treatment. The fact could explain the progressive development of retroperitoneal fibrosis in our case, with hydronephrosis installation and deterioration of renal function after EVAR, which normalized with ureteric stenting. In spite of this, we cannot exclude that this inflammatory and fibrotic process is not related to the EVAR, but it is simply the natural course of this kind of abdominal aortic aneurysms.

This case raises the possibility of renal impairment after EVAR as the consequence of periaortitis and the need for multidisciplinary approach of this complication.

References