Available online at www.scientiaresearchlibrary.com

Scientia Research Library

ISSN 2348-0416
USA CODEN: JASRHB

Journal of Applied Science And Research, 2019, 7 (1):30-33

(*http://www.scientiaresearchlibrary.com/arhcive.php*)

NEPHROGENIC ASCITES : A CASE REPORT

F.Belabbes, K.Benjouad, K.Gharbi, S.Oubaha, Z.Samlani, K.Krati

---

ABSTRACT

Nephrogenic ascites is a clinical entity defined as refractory ascites, which complicates the development of chronic end stage renal failure, often with hemodialysis, and which in practice has sometimes very serious diagnostic and therapeutic difficulties. of this study is to specify these difficulties, through a new observation. We report a case of nephrogenic ascites associated with end-stage renal failure in a 42-year-old patient who had been on hemodialysis for 1 year and who had consulted for exudative and leucocyte-free high ascites ascites diagnosed with ascites. nephrogenic was retained after eliminating the other etiologies of protein-rich ascites (peritoneal tuberculosis, peritoneal carcinomatosis, ascites infected with cirrhosis, Budd-Chiari syndrome, hypothyroidism), the treatment was based on increasing the number of sessions hemodialysis with a decrease in the volume of ascites waiting for kidney transplantation. We conclude that this entity is rare, that its diagnosis is a diagnosis of elimination based on the exclusion of other causes of ascites, especially exudative, that its treatment is based mainly on kidney transplantation and that its prognosis is rather poor.

Keywords : *Nephrogenic ascites; Chronic renal insufficiency; Hemodialysis*

INTRODUCTION

Nephrogenic ascites is a clinical entity defined as refractory ascites of unknown etiology that occurs in a patient with chronic end stage renal disease, often on hemodialysis. This entity is particularly rare and frequently poses diagnostic and therapeutic difficulties (1).

We report in this work a case of nephrogenic ascites and we try, through the data of the literature, to specify the diagnostic, evolutionary and therapeutic peculiarities of this pathology.

MATERIALS AND METHODS

Observation:

This is Mrs. FB aged 42, followed for chronic end stage renal failure, of undetermined etiology, on hemodialysis, at 2 sessions per week, for 1 year, having risk factors for viral contagion (hemodialysis , blood transfusion, dental care at the traditional practitioner). The patient consults for an abdominal distension gradually increasing in volume, evolving for 5 months, without abdominal pain or associated transit disorders, external digestive bleeding or other digestive signs, all evolving in a context of slimming and apyrexia.

The clinical examination found an apyretic patient, pulse at 70 beats / min, eupneic with an earthy complexion, discolored conjunctiva, edema of the lower limbs and a BMI at 23Kg / m. The abdomen is distended, without collateral venous circulation, showing diffuse dullness. The examination of the liver and spleen is hampered by ascites of great abundance. There are no signs of hepatocellular insufficiency (no evidence of hepatic encephalopathy, jaundice, stellate angiomas, palmar erythrose, or digital clubbing). Pleuro-pulmonary and cardiovascular examination are normal; in particular, there are no signs of right heart failure). There are no clinical signs of hypothyroidism.

Ultrasonography supplemented by abdominopelvic CT shows ascites of great abundance with small kidneys. The liver is homogeneous, of normal size and regular contours. The trunk door is of normal caliber. There is no deep lymphadenopathy or digestive thickening. The ovaries are normal in appearance. The ascites puncture results in a citrus-yellow fluid, rich in protein (50 g / l), low in leucocytes (100 leucocytes / mm3) without significant eosinophilic polynuclear cells. Direct examination and culture for bacteria and Koch bacillus in the ascites fluid were negative. We carried out a tuberculosis assessment (tuberculin IDR, chest x-ray, BK sputum test, adenosine deaminase (ADA) determination in the ascites fluid) which was negative. The abdominopelvic scan is normal. Histopathological examination of the fluid showed no neoplastic or atypical cells. . Doppler ultrasound of hepatic veins is normal

The blood biology highlighted:

- normochromic normocytic anemia at 9.8g hemoglobin / 100ml

a normal leucocyte formula with, in particular, no blood eosinophilia.

a serum creatinine elevated to 663 m mol / l

- a normal liver test, with in particular absence of signs of hepatocellular insufficiency (88% TP), and negative B and C viral serologies. TSHus is normal at 2 mIU / l. Upper gastroscopic fibroscopy and colonoscopy were normal and did not specifically find oesophageal or cardiotuberotic varices or tumoral processes.

A diagnostic laparoscopy should have been performed, but was refused by the patient. The evolution was marked by the resistance of ascites to diuretics and its frequent recurrence after macro puncture.

The diagnosis of nephrogenic ascites was retained in this patient with the combination of exudative ascites and chronic renal failure and the elimination of other causes of ascites, particularly rich in proteins (ascites infected with cirrhosis, insufficiency heart disease, Budd chiari syndrome, peritoneal tuberculosis, peritoneal carcinomatosis, hypothyroidism ...). After discussion with the nephrologists, we decide to increase the number of hemodialysis sessions.

At present, and after a retreat of almost ten months, the patient maintains a relatively conserved general state and ascites of low free abundance and no peritoneal nodules.

RESULTS AND DISCUSSION

Nephrogenic ascites was formerly called dialysis ascites or ascites associated with hemodialysis. However, the term nephrogenic ascites seems more appropriate since ascites may precede dialysis (2).

The appearance of ascites during end-stage renal failure was reported for the first time in 1970 by Cinque T.J, Mahony J.F. and their collaborators (3,4). At the beginning, we spoke of dialysis ascites

or ascites associated with hemodialysis. Currently, the term nephrogenic ascites is preferred because ascites may precede dialysis.

The exact pathogenesis of this entity is still unknown and several mechanisms are mentioned:

-Alteration of the permeability of the peritoneal membrane during end-stage renal failure.

-The decrease in the passage of peritoneal fluid to the lymphatic channels, due to vascular hepatic and portal overload secondary to previous peritoneal dialysis sessions. (2)

-The obstruction of the peritoneal lymphatic channels responsible for an alteration of the resorption of peritoneal fluid in chronic renal failure (5).

And hypoalbuminemia, which may be secondary to renal involvement and contribute to the formation of ascites (2).

Clinically, ascites is of variable abundance and is usually associated with lower limb edema, anorexia, and weight loss. The absence of signs of portal hypertension, hepatocellular insufficiency and right heart failure on this site should already attract the clinician's attention. The exploratory puncture of ascites typically returns a protein-rich, low-leucocyte fluid.

Our observation meets these clinical and biological criteria.

The diagnosis of nephrogenic ascites is retained after elimination of other etiologies of ascites, in particular exudative and low in leucocytes.

Peritoneal carcinomatosis is the main differential diagnosis in this situation. Its elimination relies mainly on laparoscopy or diagnostic laparoscopy with peritoneal biopsies. In some cases, evidence of neoplastic cells in the ascitic fluid or percutaneous peritoneal biopsy of possible peritoneal nodules on ultrasound may be an alternative to laparoscopy. In our patient, there were no signs pointing to primary neoplasia, ultrasonography and CT did not show any peritoneal nodules, the search for neoplastic cells in the ascites fluid was negative and diagnostic laparoscopy was refused. Clinical and morphological evolution has allowed us to virtually eliminate peritoneal carcinomatosis.

Right heart failure, pericarditis, and Budd chiari syndrome may also pose diagnostic problems with nephrogenic ascites. They are eliminated by the clinic, the ultrasonographic explorations and if necessary the angio scanner, to see the magnetic resonance imaging. In our patient, these three diagnoses have been formally discarded.

Infected ascites of the cirrhotic are eliminated in the absence of signs of hepatocellular insufficiency and portal hypertension, the absence of hepatic dysmorphia and the negativity of cytobacteriological examinations of the ascites fluid.

Peritoneal tuberculosis, a diagnosis that must always be considered in the presence of ascites in our country, is eliminated by diagnostic laparoscopy, which ideally should be combined with peritoneal biopsies. The presence of signs of tuberculous impregnation, a richness of ascites fluid in leukocytes predominant on the lymphocytes with positive ADA dosage, peritoneal cakes and the cloisonne character of ascites are arguments which point towards the diagnosis. peritoneal tuberculosis. None of these arguments were present in our patient; and here too, evolution without specific treatment allowed us to rule out this diagnosis.

Eosinophilic ascites is eliminated in the absence of blood eosinophilia and ascites fluid. Finally, hypothyroidism is eliminated by a normal rate of TSH us.

The treatment of nephrogenic ascites poses significant difficulties in practice. In fact, albumin-perfused evacuation puncture, peritoneal shunts and peritoneal instillation with corticosteroids are not effective in the medium and long term [6]. Daily hemodialysis results in a loss of protein and worsens malnutrition and can not be proposed despite its effectiveness on ascites. Paracentesis with reinjection of ascitic fluid during hemodialysis [7] and continuous ambulatory peritoneal dialysis can control ascites but pose practical problems [2].

As a result, kidney transplantation remains the only definitive treatment for ascites; it corrects renal function and improves the survival and quality of life of patients (8).

The prognosis for nephrogenic ascites is poor, with an average survival of 7 to 10.7 months and a mortality rate of 44% after 15 months of diagnosis (9). Our patient is still surviving with a relatively well preserved general condition after one year of evolution.

In our patient, and the treatment was based on hemodialysis sessions and ascites punctures with intravenous passage of albumin when needed.

CONCLUSION

Nephrogenic ascites is a rare and serious complication of end-stage renal failure.

Its exact pathogenesis is unknown and certainly multi factorial. Its diagnosis is a diagnosis of elimination and is based on the exclusion of other etiologies of ascites, especially exudative ones. His treatment is based mainly on kidney transplantation, which improves the quality of life and increases patient survival.

REFERENCES

[1] Han, Steven Huy B. MD, Reynolds, Telfer B. MD, Fong, Tsc-Ling MD Nephrogenic ascites : Analysis of 16 cases and review of the literature. Ovid. Han. Medicine (Baltimore). **1998** ; 77 : 233-45

[2] Debbech R, Salem M. L'ascite néphrogénique, un diagnostic rare auquel il faut penser. A propos d'une observation. La Tunisie Médicale **2010**;88:437-39.

[3] Cinque TJ, Letteri J. Idiopatic ascites : complication of extra corporeal dialysis. Am.Soc.Nephrol.**1970** ; 4 :16

[4] Mahony JF. , Guth CF., Holmes JH. Intractables ascites in chronic dialysis patients. Am. Soc – Nephrol. **1970** ; 4 : 51

[5] Hela Elloumi, Dalenda Arfaoui, Ahlem Zayane, Salem Ajmi. Ascite néphrogénique. A propos d'une observation. Ann. Med. Interne. **2002** ; 153 : 478-80

[6] Jones BF, Trevillian PR. Idiopathic ascites of hemodialysis. Risponse to treatment. Br Med J **1976**;1:877.

[7] Touam M, Orozoo R. Refractory ascites in hemodialysis. Treatment by Paracentesis-reinjection during dialysis. Nephrologie **2001**;22:25-8.

[8] Popli S., Chen WT., Nakamoto S., Dangirdas JT., Cespedes LE., Ing TS. Hemodialysis ascites in anephric patients. Clin. Nephrol. **1981** ; 15 : 203-5

[9] Feingold LN, Gutman RA., Walch Fx, Gunelles C. Control of cachexia and ascites in hemodialysis patients by binephrectomy. Arch. Intern. Med. **1974**; 134 : 989-97.