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Type AA Amylosis Secondary to Tuberculosis Revealed by Rectal Bleeding and Diarrhea: A Case Report

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INTRODUCTION

Amyloidosis are a rare disorders related to the accumulation of insoluble substance, consisting of a fibrillar structure (in a proportion of 85% to 95%) and an extra-fibrillary structure (component P and polysaccharide fractions) [1,2]. The fibrillar part defines the type of amyloidosis [1,3] of which the most frequent are AL amyloidosis (immunoglobulinic), secondary reaction or AA amyloidosis, AB2M hemodialysis amyloidosis and ATTR senile amyloidosis.

Secondary AA amyloidosis can complicate chronic inflammatory disease. Rheumatoid arthritis, chronic infection, chronic inflammatory bowel diseases and Mediterranean fever are among the most commonly described etiologies [4].

We report the particular case of a 30 year old patient, with no significant medical history, diagnosed in our department of systemic amylosis secondary to lymph node tuberculosis, revealed by non-specific digestive symptoms.

CASE REPORT

Mr A.S is 30 years old, with no particular medical history, mainly no tuberculosis disease, nor recent tuberculosis contagion in the surroundings, neither inflammatory nor systemic diseases nor primitive nor acquired immunodeficiency.

The patient had diffused abdominal pain, cramps type, associated with early postprandial food vomiting, liquid diarrhea (3 stools/day) and rectal bleeding, all of which occurred for 9 months prior to admission in our facility in a context of febrile sensations, alteration of the general state and weight loss.

The whole clinical picture was aggravated 3 months before his admission to our department. An increased number of stools (8 stools/day) and abundance of rectorragies were noticed. First clinical examination revealed a stable hemodynamic and consciousness state, although the patient had a tachycardia at 100 beats/min, was dehydrated with a BMI (body mass index) at 20 kg/m^2 and had a painful right axillary lymphadenopathy measuring 4 * 2 cm.

The blood tests showed an inflammatory syndrome (an accelerated sedimentation rate at 100 mm in the 1st hour, a C reactive protein -CRP- level at 120 mg/l, hypochromic microcytic anemia at 7 g/dl and a thrombocytosis at 750000/ul), associated with leukocytosis at 12000/ul. Renal failure (creatinine at 112 mg/l, urea at 1.7 g/l) was also found as well as cholestasis (gamma-GT at 160 IU/l, alkaline phosphatase -ALP at 242 IU/l) without cytolysis.

Nephrotic syndrome also showed in biologic analysis with 24 h proteinuria positive at 3.6 g/l (i.e., 5, 5 g/24 h) associated with hypo-albuminemia at 25 g/l.

Parasitological stool examinations (EPS) and stool culture performed to identify the etiology of the diarrhea were sterile.

A proctological examination demonstrated a fragile mucosa bleeding easily on contact, with an AA-type amyloidosis and non-specific chronic inflammatory changes in rectal biopsy; same results also were obtained in the histological analyzes of the labial biopsy and those of the liver biopsy.

Cardiac exploration (electrocardiogram and cardiac ultrasound) found neither rhythm or conduction disturbances nor cardiomyopathy in favor of cardiac amyloidosis. The paraclinical exploration supplement by an upper digestive tract endoscopy had objectified a petechial antral gastritis with in the histological analysis a moderate non-atrophic chronic antro-fundic gastritis of light activity HP+. Colonoscopy was normal and the pathology study revealed only non-specific inflammatory changes. Whereupon, the

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diagnosis of systemic AA amyloidosis was retained.

In order to investigate the underlying etiology, the ultrasound showed axilliary necrotic lymph nodes that were biopsied revealing an active tuberculoid granulomatous adenitis without caseous necrosis. The abdominal ultrasound **(Figure 1)** also showed several necrotic coelio-mesenteric lymphadenopathies, the largest of which measured 2.5 cm of minor axis. Both kidneys were increased in hyper-dedifferentiated size (in favor of acute functional renal failure). And in thoraco-abdominopelvic CT **(Figure 2)**, mediastinal and abdominal polyadenopathies with mild hepatomegaly, splenomegaly and coelio-mesenteric and hilar hepatic adenopathies. Although the search for Koch bacilli in the sputum was negative, however the chest x-ray showed a hilar opacity and the tuberculin intradermoreaction was positive at 20 mm of phlyctenular appearance. Thus, the diagnosis of pulmonary and hematopoietic tuberculosis was carried out.

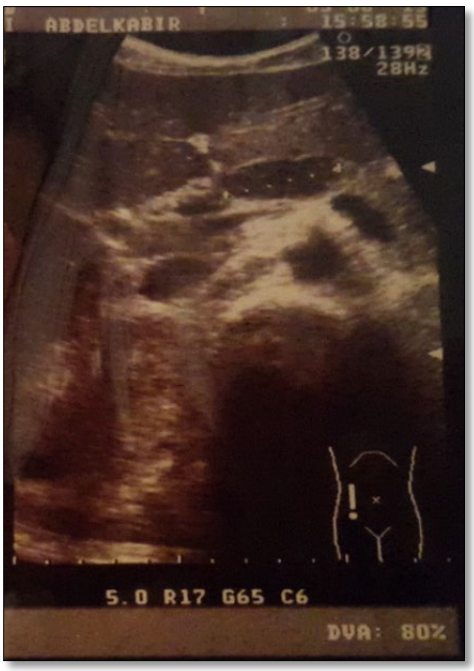

Figure 1. Coelio-mesenteric adenopathies.

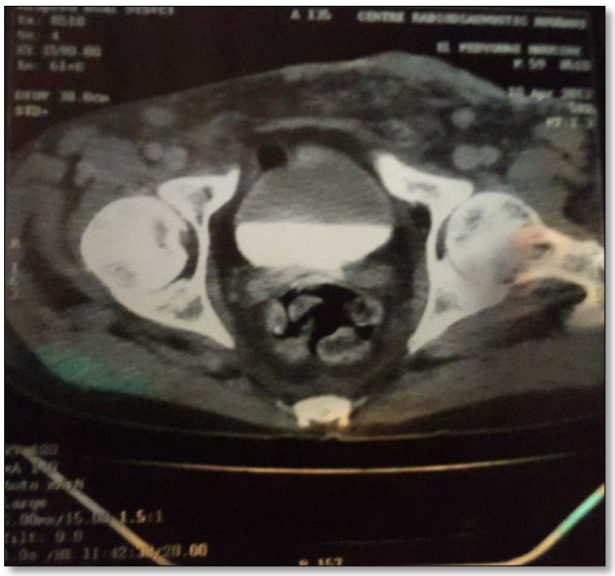

Figure 2. Poly-lymphadenopathy.

The patient had two hemodialysis sessions with transfusion. Diuresis was resumed two days after dialysis with an improvement in renal function (urea at 1.5 g/l and creatinine at 41.7 mg/l).

The anti-bacillary treatment was started according to the 2RHZE/4RH regimen (R: rifampicin, H: isoniazid, Z: pyrazinamide and E: ethambutol) associated with colchicine.

The clinical outcome was favorable with a follow-up of 7 months, clinically by the disappearance of the axillary lymphadenopathy, abdominal pains and diarrhea and a resumption of the weight (gain of 14 kg) and biologically by the improvement of the renal function (urea at 0.63 g/l and creatinine at 18 mg/l) and cholestasis (gamma-GT at 92 IU/l and PAL at 192 IU/l). Proctologic examination had even shown normal rectal mucosa and biopsy, sub-acute and chronic inflammatory changes with no specificity and no amyloid deposits.

DISCUSSION

Systemic amyloidosis is related to the extracellular deposition of amyloid proteins with abnormal fibrillar structure. The clinical presentation is variable and non specific but the digestive tract lesions are frequent which symptoms could even mimic several pathologies of the digestive tract thus posing a real problem of differential diagnosis, in which the biopsy yield, up to 80%, is of a major contribution [5]. The key molecular abnormality is a folding defect with a modification of the spatial conformation of the protein and a tendency to aggregation [2,3].

The inflammatory process, in the context of chronic inflammatory diseases and chronic suppuration, involving apolipoprotein SAA, is one of the mechanisms activating amyloid deposits [2].

The list of etiologies responsible for these structural protein modifications is exhaustive. Rheumatoid arthritis is now the most common cause, followed by ankylosing spondylitis, juvenile chronic arthritis, inflammatory bowel diseases and familial Mediterranean fever. Pulmonary infections occurring on a background of cystic fibrosis precede even tuberculosis [6]. In some patients, AA amyloidosis can be considered as the result of the combination of several inflammatory diseases [7].

AA amyloidosis can complicate the evolution of certain chronic infectious diseases, thus worsening the initial prognosis by the inevitable evolution towards end-stage chronic renal failure. The frequency of these clinical pictures remains particularly high in developing countries. Khedhiri et al reported a series of 7 patients whose lesions secondary to amyloid deposits regressed after treatment of the causal pathology dominated by tuberculosis [8].

The heterogeneous distribution of the deposits in the different organs could be related to the variability of the interactions between the different amyloid proteins and the accessible tissue surfaces according to the specific physicochemical peculiarities of each organ with possible phenomenon of self-accentuation facilitating the formation of new deposits as soon as an organ is reached [2]. AA amyloidosis is therefore mainly renal, hepatic and splenic [2]. This distribution will determine the clinical expression.

The amyloid deposits are found in 51% to 72% at the oesophageal level, 89% to 95% at the gastric level, 96% at 100% in the small intestine, 83% to 91% at the colorectal level and 93% at 100% in the salivary glands [2].

Gastrointestinal manifestations in amyloidosis are variable: abdominal pain, chronic diarrhea, nausea, vomiting, malabsorption, and can be disabling [2].

The onset of spleen involvement is early, as shown by experimental data and P-scintigraphy. This splenic involvement is observed in 87% of patients with systemic amyloidosis. Splenomegaly is clinically observed in only 5% of cases of AL amyloidosis. Biologically, thrombocytosis greater than 500000/mm is noted, however hematologic signs of hypersplenism are unusual in AA amyloidosis [2]. The liver involvement occurs later on and is almost constant during AA or AL amyloidosis (97% to 100%) and results in hepatomegaly with cholestasis, usually without clinical consequence; more rarely ascites, jaundice, portal hypertension and liver failure are reported [7]. Even more exceptional and more pronounced cases have also been reported, such as macroglossia, achalasia, dyspeptic syndrome, gastrointestinal bleeding and subclinical syndrome and even peritonitis by organ perforation [2,3]. Biologically, the increase in ALP is the most frequently reported abnormality during hepatic amyloidosis, it is more marked for AA type than AL type (60% to 80%), cytolysis is generally low (<2 times normal) throughout the course of the disease. Bilirubin above 34 µmol/l (20%) is a poor prognostic factor. The prothrombin level is low in 25 to 35% exposing to a hemorrhagic risk. Hypercholesterolemia greater than 5.2 mmol (80%) is an early manifestation of liver injury. The decrease in albuminemia is only observed in cases of nephrotic syndrome [2].

In the case of our patient, the digestive symptoms dominated the clinical picture, essentially made of diarrhea and rectorrhagia that helped to initially guide the diagnosis of rectal amyloidosis which had pushed the investigations and the assessment of systemic amyloidosis.

Liver biopsy was performed despite the poor clinical presentation, with radiologically discovered hepatomegaly and biological cholestasis, identifying thereby amyloid deposits and ruling out the diagnosis of hepatic tuberculosis simulating the above-described table.

The signs of tuberculous impregnation and the radiological signs (deep lymphadenopathy and pulmonary opacities) had pushed the investigations confirming the diagnosis of bifocal tuberculosis on a set of empirical arguments reinforced by the favorable therapeutic response.

The prognosis of amyloidosis depends on the affected organ. Thus, renal impairment, manifested by a nephrotic syndrome and renal failure, and cardiac involvement, expressed by heart failure and electrical abnormalities like micro-voltage, are determinant of the prognosis [9,10]. Renal amyloidosis was suspected in our case before the nephrotic syndrome regressing under colchicine and etiological treatment.

Certainly the clinical signs direct the biopsies towards the organs involved and accessible such as the liver, the kidney, the digestive tract, the skin and the bone marrow. However, in the absence of orientation, biopsies are performed on sites known for their frequent infiltration by deposits. Given the diffuse nature of amyloid deposits and the invasive nature of renal biopsy, the current trend is to practice first-line salivary gland biopsies (sensitivity of 80% to 100%) [2]. These were also sites of amyloid deposits in our observation. All amyloid deposits share the characteristic of apple-green birefringence in polarized light after staining with Congo red, but the amyloid protein is identified by immunohistochemistry using anti-SAA antibodies [2]. Iodine-123 scintigraphy, which exploits the high affinity of SAP for amyloid fibrils, has been proposed as a diagnostic test. Several studies have shown that it is a non-invasive technique that can detect, locate and quantify amyloids in vivo. Its sensitivity is 90% for AA amyloidosis [3]. However, such a review could not be adapted to the socioeconomic base of the population we are dealing with.

The spontaneous prognosis of AA amyloidosis is poor with a median survival of 12 months after diagnosis [3]. Joss et al have found that blood and urinary albumin levels as well as creatinine clearance hold a major prognostic value, given the risk of infection and the resulting poor immunosuppressive response [11]. Severe gastrointestinal involvement was also considered to be a leading cause of death in 6.4% to 10% of cases because of bleeding complications or malnutrition charts that are secondary to malabsorption [2]. Cardiac dysfunction, amyloidosis of the peripheral vessels and the autonomic nervous system also considered a vital risk [10].

The acute renal failure that our patient had presented was of functional origin, related to the dehydration related to the loss of water and electrolytes during diarrhea and deglobulization secondary to rectorrhagia. The numbers of urea and creatinine were improved after a hemodialysis session. However, nephrotic syndrome could well be explained by renal amyloidosis. The renal biopsy puncture was avoided because of the invasiveness of the procedure and the diagnosis of systemic amyloidosis was made by more accessible and less risky biopsy specimens, as well as the favorable response after initiation of the etiological treatment.

The treatment of AA-type amyloidosis is based on the treatment of the causative disease [3,9,11,12]. Colchicine may regress or stabilize amyloid type AA and may prevent the occurrence of amyloid nephropathy [13]. The therapeutic response is monitored using non-invasive markers (CRP, creatinine and SAA) [3,4,11]. Our patient had evolved favorably on clinical level. The clearance of amyloid deposits at the end of anti-bacillary treatment was assessed by rectal biopsy, although this attitude is unconventional.

CONCLUSION

AA amyloidosis constitutes a real derogatory turn in the natural history of the underlying disease, with a worrying prognosis. It complicates infections and chronic inflammatory diseases whose incidence should be decreasing thanks to the advent of powerful immunosuppressive and antibacterial/viral therapeutics. Stopping the inflammatory process in its undifferentiated phase is essential to fight against the installation of amyloid deposits, hence the interest of early management of the incriminated etiologies. Such a picture remains a problem in the developing countries, which is life-threatening, in the hope of progress, particularly in the needy therapeutic arsenal of this morbid disease, especially the development of drugs targeting amylogenesis.

REFERENCES

1. Cazalets C, Cador B, Mauduit N, Decaux O, Ramee MP, et al. (2003) Epidémiologie descriptive des amyloses diagnostiqués au CHU de Rennes. La revue de médecine interne 24: 424-430.
2. 2, Clavière C (2008) Amyloses digestives. Gastro-enterologie (9-089-C-15).
3. Dao T, Bouhier K, Gloro R, Rouleau V, Rousselot P (2005) Amylose hépatique. EMC - Hépato Gastroentérologie 2: 330-338.
4. Prayman T, Gillmore JD, Pinney JH, Gibbs SD, Wechalekar AD, et al. (2013) Inflammatory bowel disease and systemic AA amyloidosis. Dig Dis Sci 58: 1689-1697.
5. Béchade D, Algayres JP (2007) Amylose digestive. Hépato-Gastro Oncol Digestive 14: 447-457.
6. Lachmann HJ, Gilbertson JA, Gillmore JD, Hawkins PN, Pepys MB (2002) Unicentric Castleman's disease complicated by systemic AA amyloidosis: A curable disease. Q J Med 95: 211-218.
7. Stankovic K, Grateau G (2008) Amylose AA. Nephrologie Therapeutique 4: 281-287.
8. Khedhiri A, Harzallah A, Kaaroud H, Amri N, Jerbi M, et al. (2015) Amylose rénale secondaire à des maladies infectieuses chroniques: Rémission après traitement de la maladie causale. Néphrologie Thérapeutique 11: 379.
9. Falk RH, Comenzo RL, Skinner M (1997) The systemic amyloidoses. N Engl J Med 337: 898-909.

10. Abdallah E, Waked E. (2013) Incidence and clinical outcome of renal amyloidosis: A retrospective study. Saudi J Kidney Dis Transpl 24: 950-958.

11. Joss N, McLaughlin K, Simpson K, Boulton-Jones JM (2000) Presentation, survival and prognostic makers in AA amyloidosis. QJ Med 93: 535-542.

12. Kim YJ, Kim HS, Park SY, Park SW, Choi YD, et al. (2012) Intestinal amyloidosis with intractable diarrhea and intestinal pseudo-obstruction. Korean J Gastroenterol 60: 172-176.

13. Béji S, Kaaroud H, Moussa FB, Goucha R, Abderrahim E, et al. (2004) Amylose rénale compliquant l'évolution des colites inflammatoires chroniques. Presse Med 33: 862-865.