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Case Report

Aortic infective endocarditis decompensated by alithiasic cholecystitis in a patient on hemodialysis: Case report

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ABSTRACT

Infective endocarditis is an uncommon disease, which most often affects elderly subjects at risk or with favorable factors. Its prognosis is guarded with complications or decompensating factors that are often formidable.

We report the case of an acute endocarditis decompensated by acute alithiasic cholecystitis in a 52-year-old patient, with a history of diagnosed end-stage renal failure (GFR 7 ml/min according to the MDRD) for 4 weeks, of undetermined etiology, undergoing hemodialysis, followed for aortic disease for 6 years (IAO grade II, RAO loose). After 6 weeks, the evolution was favorable under adapted and early antibiotic treatment and associated hygiene measures including gastric rest.

Infective endocarditis remains a serious pathology, due to its high morbi-mortality. The association of acute infective endocarditis and acute alithiasic cholecystitis is of reserved prognosis especially on a ground of immunodepression like the end-stage chronic renal failure. This association requires a rapid and efficient management.

1. Introduction

Our work consists of a single case report and has been reported in accordance with SCARE 2020 criteria [1].

Infective endocarditis is defined as colonization of the healthy or pathological endocardium (valvular or more rarely parietal) or intracardiac prosthetic material by a microorganism, most often bacterial. Most often it is secondary to medical care related gestures and favouring factors having emerged.

The treatment is well codified and is based on broad-spectrum parenteral antibiotic therapy associated with hemodynamic stabilization measures [2]. The evolution and prognosis depend on several parameters: terrain, associated defects, and decompensation factors.

The factors of decompensation are essentially multi-visceral failure, in particular cardiac failure, but other organs can be incriminated.

The authors report a case of infective endocarditis decompensated by acute lithiasis cholecystitis in a chronic end-stage renal failure.

2. Case presentation

A 52-year-old patient of average socioeconomic status with a history of diagnosed end-stage renal disease (GFR 7 ml/min according to MDRD) for 4 weeks, of undetermined etiology (no family history of renal disease). He had been on hemodialysis for 10 days (KTC J right: 2 sessions/week). He has been regularly followed for aortic disease for 6 years (IAO grade II, loose RAO). His risk factor is a chronic smoker at 21 pack-years, weaned one month ago. The patient consulted his nephrologist because of an altered general condition for 2 days with orthopnea, asthenia and fever. The clinical examination revealed: fever at 39 °C, altered general condition (WHO score 3); weight 58 kg/height 1.72 m (BMI: 19.6 kg/m2); hemodynamics: orthopneic (NYHA IV), BP at 130/82 mmHg, HR at 88 bpm, ECG unfolds regular sinus rhythm, with first degree atrioventricular block, electrical left ventricular hypertrophy, 1 mm sub-shift inferiorly, on cardiac auscultation a diastolic murmur at the aortic focus of 4/6 and a systolic murmur at the mitral focus of 2/6 is objectified; respiratory status: SpO2 at 90% in room air (97% under O2, 2L/min), FR: 22 cycles/min, on pulmonary auscultation there are

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crepitus rales at both pulmonary bases, pulmonary RX: normal; neurologically: GSC score at 15/15. Moreover, the examination of the dialysis catheter revealed inflammatory signs (skin redness, with pain on palpation), the dressing was slightly soiled. In emergency, the dialysis KT was removed and sent to the laboratory for bacteriological examination. The rest of the somatic examination was unremarkable. Biologically, the following were found: white blood cells: 16730/µl; CRP: 52.10 mg/L; Procalcitonin: 12.48 ng/ml; hemoglobin: 8.4 g/dl; platelets: 223000/µl; prothrombin time: 45%; AST/ALAT: 275/298 IU/L; Na+/K+: 134/5.2 mEq/L; urea/creatinine: 2.23 g/L/91.1 mg/L; (GFR: 5.7 ml/mn/m2; according to MDRD); GAJ: 1.02 g/L. Troponin Us: 54 ng/ml; Pro BNP: 147 pg/ml; D-Dimers: 100. Blood cultures came back negative, as well as bacteriological examination of the dialysis catheter. Transthoracic cardiac echocardiography showed a left ventricle dilated to 67 mm, with 50% preserved LV function, vegetations on the aortic valve, one on the right anterior cusp and one on the coronary annulus responsible for the worsening of the aortic insufficiency with SOR at 43 mm^2, RV at 61 ml, telediastolic effect at 38 cm/sec. [Fig. 1]. There is a moderate mitral leak by bivalvular restriction and tenting, SOR at 23 mm^2, PISA radius at 6.5 mm, tenting height at 11 mm, without vegetation. There was a moderate tricuspid insufficiency estimating the PAPS at 58 + 15 = 73 mmHg, no vegetation image on the tricuspid valve nor on the pulmonary valve, dilated inferior vena cava, not very compliant and the pericardium was dry. In view of these data, the diagnosis of probable infective endocarditis, in an immunocompromised environment, was evoked. The patient was put on dual antibiotic therapy with vancomycin 500 mg and Gentamicin 160 mg adapted to the renal function.

The evolution was marked at D8 by the persistence of the fever (39°) and the worsening of the PCT (12.48 VS 19.32 ng/ml) and of his general condition. The patient was switched to Imipenem (THIENAM*) 500 mg/day, preceded by a complete new bacteriological assessment. The evolution was marked after 15 days of treatment by the appearance of incoercible food vomiting, with abdominal pain in the right hypochondrium. The physical examination revealed a positive Murphy's sign with no sign of cholestasis. **Abdominal and pelvic ultrasound revealed signs suggestive of alithiasic cholecystitis without signs of pancreatitis and without effusion [Fig. 2].** Complementary abdominal CT scan confirmed the diagnosis of alithiasic cholecystitis, with thickened gallbladder walls, free of any calculus [Fig. 3]. The biological work-up showed lipemia at 571 IU/l; GGT at 234 IU/l; total bilirubin at 30.5 mg/l; conjugated bilirubin at 16.8mg/l; free bilirubin at 13.7mg/l; white blood cells at 24,000/mm3, Procalcitonin at 25.32 ng/ml; CRP at 103 mg/L.

In agreement with the gastroenterologists, the patient was put on analgesics, digestive rest with parenteral feeding, gastric protection (the patient was already under antibiotic coverage), with indication of a Bili-IRM which returned normal. After 5 days of treatment the evolution was favorable. The patient regained apyrexia, with improvement of the digestive signs, and a normal ultrasound appearance of the gallbladder after 10 days. Biologically, PCT was negative at 4 VS 25.32 ng/ml. Lipasemia 70 IU/l; GGT 65 IU/l; total bilirubin 10.5 mg/l; conjugated bilirubin 5 mg/l; free bilirubin 7mg/l; white blood cells 9000/mm3; CRP 30 mg/L. After 6 weeks of treatment, the patient was declared discharged with letters of consultation in cardiology, nephrology and gastroenterology.

In summary: infectious endocarditis decompensated by acute alithiasic cholecystitis on a ground of chronic end-stage renal failure having evolved favorably under medical treatment (digestive rest + antibiotic therapy).

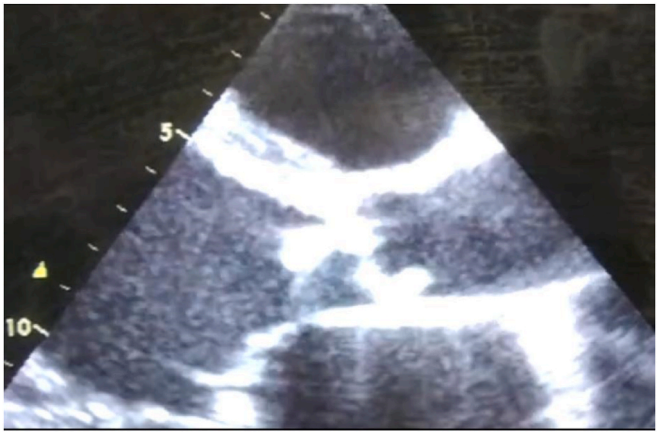

Fig. 1. Cardiac ultrasound showing vegetations on the aortic valve.

2.1. Clinical discussion

Infective endocarditis, described by Sir William Osler in 1855, is defined as colonization of the healthy or pathological endocardium (valvular or, more rarely, parietal) or intracardiac prosthetic material by a microorganism, most often bacterial. It is usually classified into subacute endocarditis ("slow" or Osler's disease), and acute endocarditis with virulent germs. It is an infrequent disease with an incidence of 3–10 per 100,000 persons/year with a significant increase with age. It affects at least two men for one woman, 2H = 1F. The lethality of infective endocarditis in the initial hospital phase varies from 10 to 25% according to studies [2].

Most often, it is secondary to medical care procedures, and favorable factors have emerged (valve prostheses, degenerative valve sclerosis, intravenous drug abuse). The association of aortic and/or mitral murmurs, acute fever and the presence of valvular vegetations on echocardiography in a patient with a dialysis catheter, allows the probable diagnosis of infective endocarditis to be made, despite the absence of identification of germs in the various laboratory tests. On a practical level, it is the association of one major and two minor criteria, according to the modified DUKE classification. In our patient, vegetations are found on echocardiography in aortic disease in a febrile context.

The background of chronic renal failure under dialysis is favorable to the occurrence of an infectious phenomenon. Moreover, the dialysis catheter is a significant entry point, and the favorable evolution of endocarditis under appropriate treatment confirms the diagnosis. The factors of decompensation of acute endocarditis are generally multivisceral failures, in particular cardiac decompensation, according to the literature [3]. In our patient, alithiasic cholecystitis was a decompensating factor.

Alithiasic cholecystitis is an acute necrotic inflammation of the gallbladder, in the absence of gallstones, with an incidence ranging from 2% to 14% of all cases of acute cholecystitis [2,3]. Some authors suggest biliary stasis and ischemia as etiopathogenic phenomena. Risk factors associated with alithiasic cholecystitis include: trauma, recent surgery, shock, burns, sepsis, critical illness, total parenteral nutrition, prolonged fasting, duration of respiratory support, positive end-expiratory pressure, and factor XII activation [2–4] In our patient, the risk factors are chronic end-stage renal disease-which is a critical illness-and infective endocarditis on aortic disease, which can be the starting point for sepsis. The positive diagnosis of acute cholecystitis is based on clinical symptoms, which include right hypochondrial pain, positive Murphy's sign on abdominal physical examination [5]; and imaging findings (abdominal ultrasound, CT scan, and Bili-MRI) showing a thickened gallbladder [6]. Our patient presented vomiting with abdominal pain localized in the right hypochondrium and Murphy's sign on physical examination. On ultrasound, an uncomplicated alithiasic gallbladder was found, confirmed by abdominal CT. The best choice of treatment for AAC is cholecystectomy, in a clinically stable patient [6,7] Clinicians should be wary of elderly patients with severe leukocytosis, which is a clinical predictor of severe gallbladder complications. In this case, the treatment of acute alithiasic cholecystitis is medical and consists of

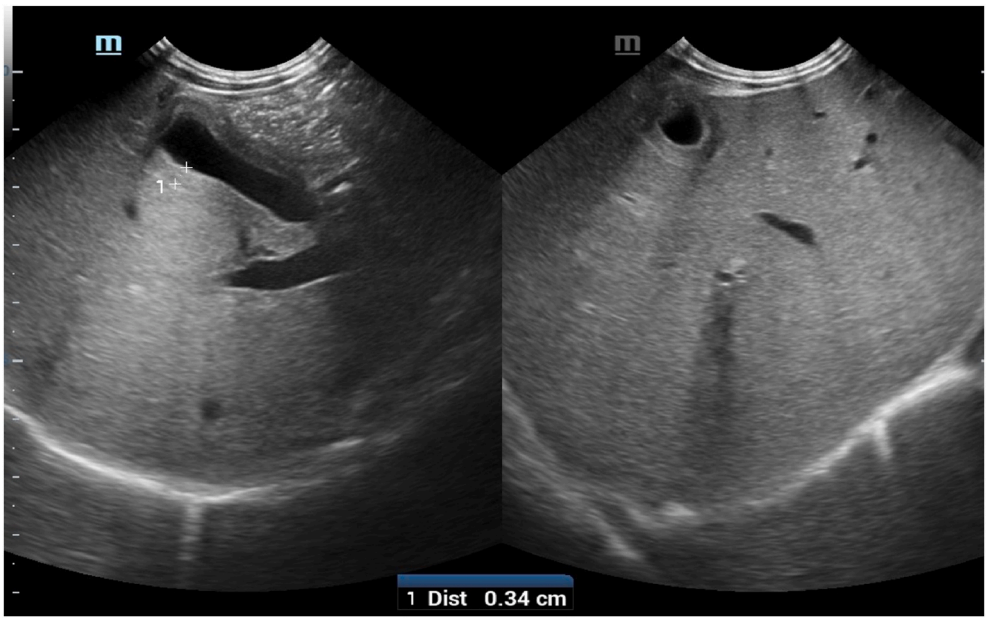

Fig. 2. Ultrasound image of alithiasic cholecystitis.

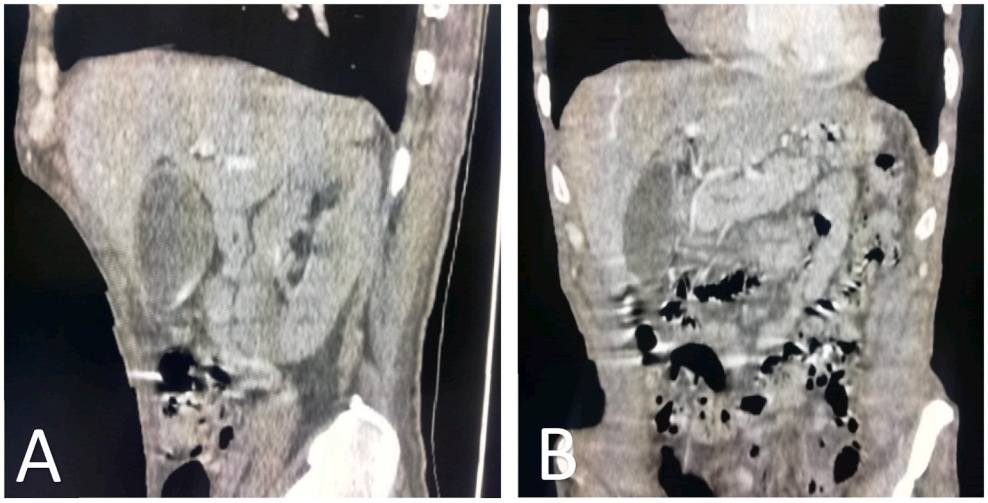

Fig. 3. Abdominal CT scan in sagittal (A) and coronal (B) sections showing alithiasis cholecystitis.

digestive rest with gastric protection and parenteral antibiotic therapy. The indication for medical treatment was retained in our patient because of his critical cardiac and renal condition and his infectious workup with worsening hyperleukocytosis and PCT. Alithiasis cholecystitis is a potentially fatal and critical complication [6,8–10]. Laurila and colleagues [7] found that alithiasic cholecystitis was associated with severe disease, long ICU stay and multi-visceral failure with a high mortality rate of 44%. However, early and adequate management can improve prognosis and survival. In our patient the evolution was favorable with digestive rest and antibiotic therapy.

3. Conclusion

Infective endocarditis remains a serious pathology, due to its high morbi-mortality. The association of acute infective endocarditis and acute alithiasic cholecystitis is of reserved prognosis and requires a fast and effective management. It is important to look for its decompensating factors because the prognosis depends on the precocity and the quality of the management.

Conflicts of interest

Authors of this article have no conflict or competing interests. All of the authors approved the final version of the manuscript.

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Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Consent

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Author contribution statement

Barage Amadou: Corresponding author writing the paper.
Harouna Idrissa. Seydou: writing the paper.
Mounaouir Karim: writing the paper.
Nouamou Imad: writing the paper.
Drighil Abdennaser: writing the paper.
Azzouzi Leila: writing the paper.
Habbal Rachida:: Correction of the paper.

Registration of research studies

1. Name of the registry: researchregistry
2. Unique Identifying number or registration ID: 6818
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

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