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Osler's Subacute Infective Endocarditis on Rheumatic Heart: A Complicated Clinical Case That Reflects Four (4) Major Public Health Issues in Sub-Saharan Africa

Pêngd-Wendé Habib Boussé Traore[1,2*], Aliou Alassane Ngaide[1], Jean Augustin Diégane Tine[2], Alioune Kane[1], Akouètè Jean Paul Degue[1], Alassane Mbaye[1], Bouna Diack[1], Abdoul Kane[3]

[1]Cardiology Department, Idrissa Pouye General Hospital, Faculty of Medicine, Pharmacy and Dentistry, Cheikh Anta Diop University, Dakar, Senegal
[2]Institute of Health and Development, Public Health Service, Faculty of Medicine, Pharmacy and Dentistry, Cheikh Anta Diop University, Dakar, Senegal
[3]Cardiology Department, Dalal Jamm Hospital, Faculty of Medicine, Pharmacy and Dentistry, Cheikh Anta Diop University, Dakar, Senegal
Email: *traore.habib1990@gmail.com

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Abstract

Endocarditis is an inflammation of the endocardium and its structures (valves), most often of infectious origin, described by William Osler in 1885. In the 21st century, infective endocarditis remains a reality in our countries. We report a complicated case of infective endocarditis (IE). This is a 53-year-old woman, obese and passive smoker who died on the 5th day of her hospitalization following an infective endocarditis (IE) with bacterial strains resistant to the usual antibiotics: daughter of acute lithiasic cholecystitis, mother of major mitral valve perforation, brain abscess, ischemic stroke and atrial fibrillation. All were responsible for septic shock and fatal coma. Surgical management of the infective endocarditis in the first hours of her admission could have improved her prognosis. To conclude, in addition to its interests and its clinical particularities, our present observation has highlighted major public health problems specific to our sub-Saharan African countries, namely: The problem of the double health burden, the problem of delays in seeking care, the problem of resistance to antibiotics and the problem of the insufficiency of reference health technical platforms.

Keywords

Infective Endocarditis, Rheumatic Heart Disease, Cardiovascular Risk Factors,

Antibiotic Resistance, Acute Lithiasis Cholecystitis, Neurological Complications, Delayed Consultation, Senegal

1. Introduction

Infectious endocarditis (IE) is a septicaemia state due to the graft and proliferation of a pathogenic microorganism circulating in the blood, on an initially sterile fibrino-platelet vegetation developed on a healthy or previously damaged endocardium or on any prosthetic structures and various pacemaker or defibrillator leads [1] [2] [3]. This definition also includes infections developed on heart defects [1] [2] [3].

IE positive diagnosis is aided by the modified Dukes diagnostic criteria [1] [2] [3]. Its prognosis remains dark and its management is medical and sometimes surgical [1] [2] [3].

We report the case of a singular IE due to the rarity of its portal of entry (acute lithiasic cholecystitis), due to the presence of multiple complications (valvular perforation, cerebral abscess, ischemic stroke, septic shock), and finally due to the multiple antibiotic resistances of isolated bacterial strains. In addition to these clinical particularities, our observation reminds us of the existence of four (4) major Public Health issues in Sub-Saharan Africa.

2. Observation

This was a 53-year-old patient, with passive smoking at home as a cardiovascular risk factor, menopause, grade I obesity, physical inactivity, with no known history of heart disease who had been admitted to the Cardiology department of the Idrissa POUYE General Hospital.

The reasons for consultation and hospitalization evolved for three weeks with abdominal pain of increasing intensity in the right hypochondrium, with vomiting of food, permanent fever and palpitations. To this was added suddenly in less than 24 hours, Wernick's motor aphasia and psychomotor agitation. It was these last two signs that worried the relatives and motivated the emergency consultation in the middle of the night.

On admission, the patient was very agitated, obtunded, feverish with a body temperature of 39.8˚C, blood pressure was 145 mmHg/90mmHg, heart rate 115 beats per minute, respiratory rate 32 cycles per minute with an ambient air saturation of 94%.

Physical examination found regular auscultatory tachycardia with an aortic insufficiency murmur, with no signs of peripheral heart failure. The patient systematically rejected the examiner's hand at each attempt to palpate the right hypochondrium (clinical Murphy sign). The rest of the examination (including ENT) was normal apart from effacement of the right nasolabial fold with contralateral deviation of the mouth and positive pipe sign.

The resting surface 12-lead electrocardiogram (Figure 1) showed: regular sinus tachycardia, PR interval at 12/100 of a second, normal heart axis, Q3T3 pattern, left ventricular hypertrophy according to overload index ventricular of Sokolow Lyon with secondary disorders of the repolarization in lower side.

Emergency transthoracic resting echocardiography showed dilated heart chambers, with a RV/LV ratio < 1, free of spontaneous contrast and thrombus. Biventricular function was preserved with a Simson biplane ejection fraction measured at 65% and a TAPSE at 18 mm. Left ventricular filling pressures were normal with a Type 1 mitral profile. The inferior vena cava was dilated, measuring 22 mm and poorly compliant. The pericardium was dry and the diaphragmatic pleuro-costal pouches were free of effusion. The aortic and mitral valves were thickened, remodelled calcified, rheumatic in appearance (Figure 2(c)). Color Doppler revealed moderate aortic insufficiency (Figure 2(a)) and moderate

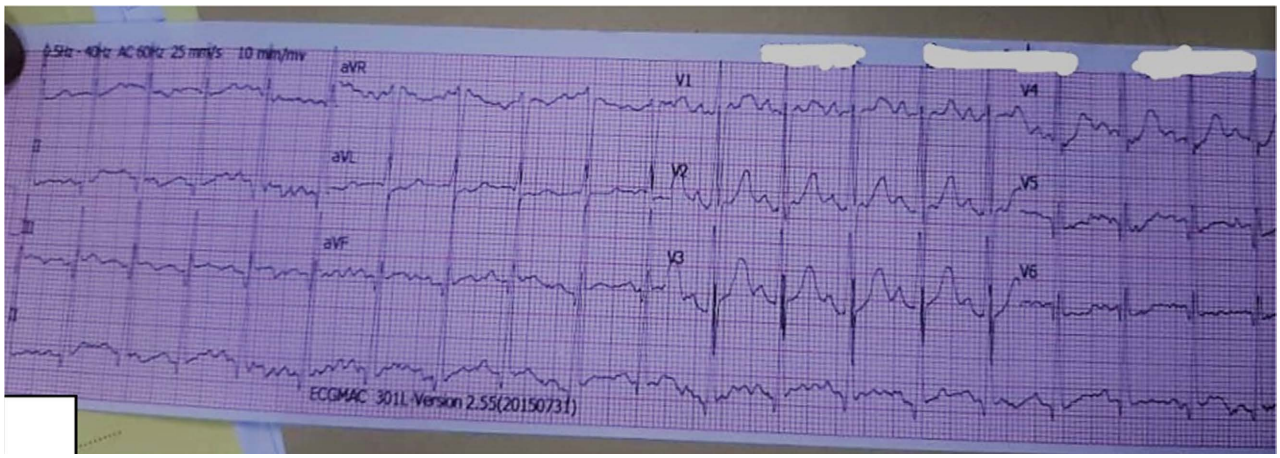

Figure 1. 12-lead resting surface electrocardiogram tracing on patient admission.

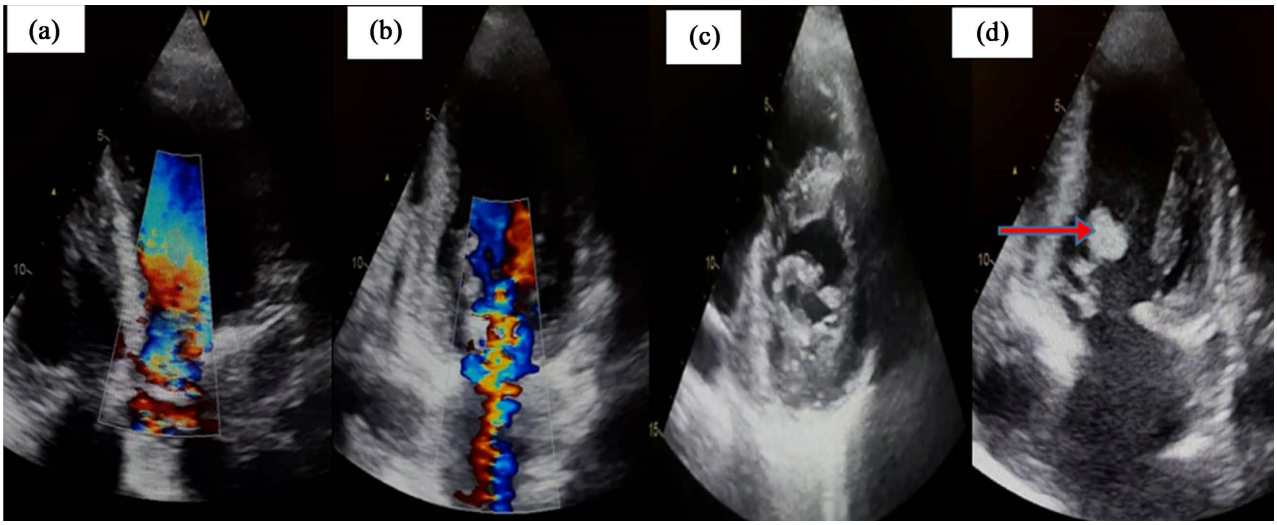

Figure 2. Resting transthoracic echocardiography images: (a) Apical 5-chamber two-dimensional section coupled with color Doppler showing aortic regurgitation; (b) Apical 2-chamber two-dimensional slice coupled to color Doppler showing mitral regurgitation; (c) two-dimensional parasternal trans mitral short axis section showing a remodeled calcified mitral valve; (d) Apical 2-cavity two-dimensional section showing the vegetation on the large mitral valve (red arrow).

to severe leak-predominant mitral disease with two (2) jets, one eccentric and one central by perforation of the anterior mitral valve and restriction of the posterior mitral valve (**Figure 2(b)**). Finally, it especially revealed a hyper-echoic image in clusters; very mobile, crumbly on the large mitral valve (image of vegetation) (**Figure 2(d)**).

Despite pain in the right hypochondrium with each passage of the probe (Murphy ultrasound sign), the emergency abdominal ultrasound revealed a dilated gallbladder with a thickened wall measured at 6mm. In the fundus of this gallbladder, there was a rounded hyper-echoic image measuring 13 × 12 mm in axis with a posterior shadow cone (**Figures 3(a)-(c)**). Abdominal CT angiography found a hyperdense image measuring 11.7 mm × 13.7 mm in the gallbladder (macrolithiasis) (**Figure 3(d)**).

Cerebral CT angiography revealed a hypodense right frontal parenchymal lesion with peri-lesional peripheral enhancement (image in rosette). This lesion measured 12.5 mm × 11.5 mm with no mass effect (midline structures in place) (**Figure 4(a)**, **Figure 4(b)**).

The biology performed came back in favor of a major non-specific biological inflammatory syndrome with hyperleukocytosis at 36.960 × 10^3 elements/LU; neutrophil predominance at 94.7%; a C-Reactive Protein at 332 mg/L; a sedimentation rate of 128 mm in the first hour, anemia at 7.6 g/dl hypochromic (26 pg) microcytic (79.3 fl) inflammatory with serum iron (150 ug/dl), ferritenaemia (14,962.22 ng/ml). Renal function, blood ionogram, blood filth were normal, HIV 1 and 2 serology, HBs Ag were negative.

Faced with the presence of a major criterion (vegetation and mitral perforation on Doppler echocardiography) and three (3) minor criteria (rheumatic valve disease, 39.8˚C fever and the biological inflammatory syndrome), we had from the admission evoked and retained the positive diagnosis of certain infectious endocarditis. According to the diagnostic criteria of Duke University of

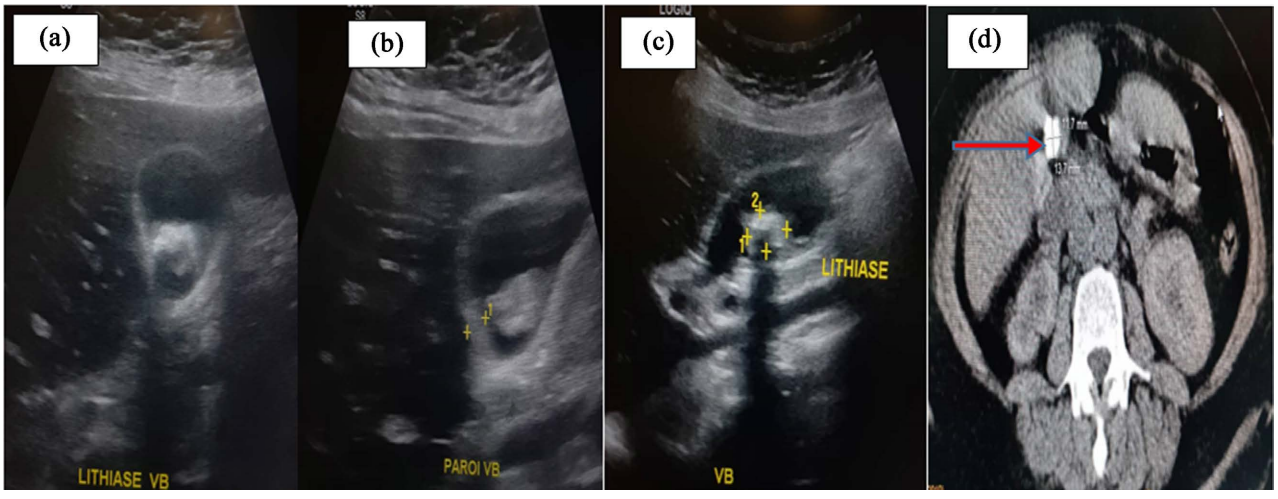

Figure 3. Abdominal ultrasound (a)-(c) and computed tomography (d) images showing acute lithiasic cholecystitis, (a) Enlargement of the gallbladder with hyper echogenic material within it, (b) Thickening of the wall gallbladder, (c) Measurement of the gallstone and posterior cone of shadow, (d) Measurement of the gallstone on the CT scan.

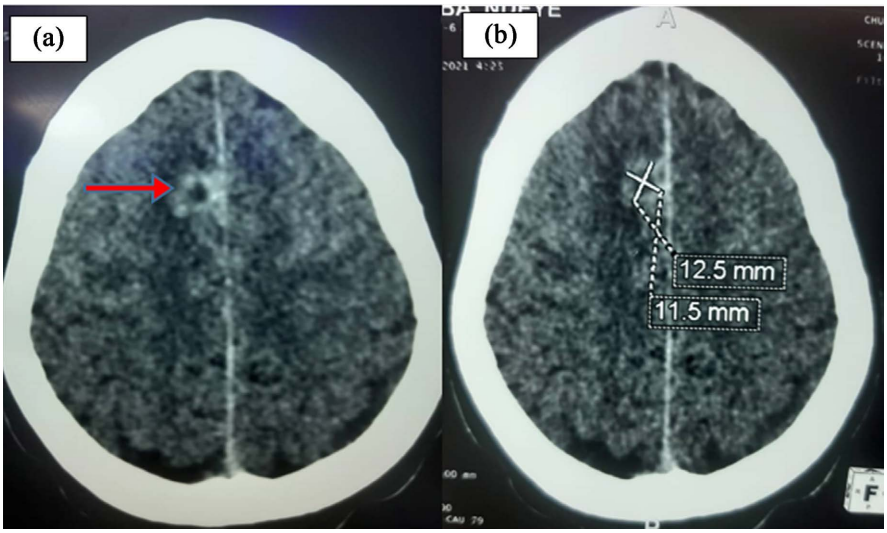

Figure 4. Cerebral computed tomography images with injection of contrast product on admission of the patient showing the cerebral abscess, (a) Demonstration of a hypodense parenchymal lesion with peri-lesional peripheral enhancement (red arrow), (b) Measurement of lesion.

1994 modified by Li in 2000 and adapted by the ESC in 2015.

It was an Osler's subacute infective endocarditis on rheumatic heart with digestive entry gate (acute lithiasic cholecystitis) and with multiple complications (valvular mutilations, cerebral abscess).

Admitted to the cardiological intensive care unit, the continuous monitoring of the constants made it possible to detect at H5 of hospitalization, a state of cardiovascular shock with a blood pressure at 70 mmHg/40mmHg; a break in diuresis, hot extremities, profuse sweating, tachycardia at 125 beats per minute and polypnea at 50 respiratory movements per minute. This septic shock required Noradrenaline 0.5 gamma kilo per hour with an electric syringe pump with an intravenous probabilistic dual antibiotic therapy comprising ceftriaxone at a meningeal dose (4 g/day in a single dose) and gentamycin (160 mg/day).

Blood cultures were collected before any antibiotic therapy and at the peak of fever in an aerobic and anaerobic environment and sent to biology.

On D2 of hospitalization, the patient presented with a supraventricular arrhythmia such as complete tachyarrhythmia by paroxysmal atrial fibrillation with an average ventricular response of 180 beats per minute (**Figure 5**), with more pronounced obnubilation, but without hemodynamic failure (BP: 110/60mmHg). This arrhythmia justified digitalis injection 0.5 mg IV. In the absence of results, amiodarone was administered as a loading dose (1800 mg per os) for a drug cardio version, which was unsuccessful.

On day 4 of hospitalization, the patient presented proportional right hemiplegia with a Glasgow score of 9/15 subject to aphasia. The emergency cerebral computed tomography returned in favour of a semi-recent ischemic cerebrovascular accident in the superficial territory of the left Sylvain valley (**Figure 6**).

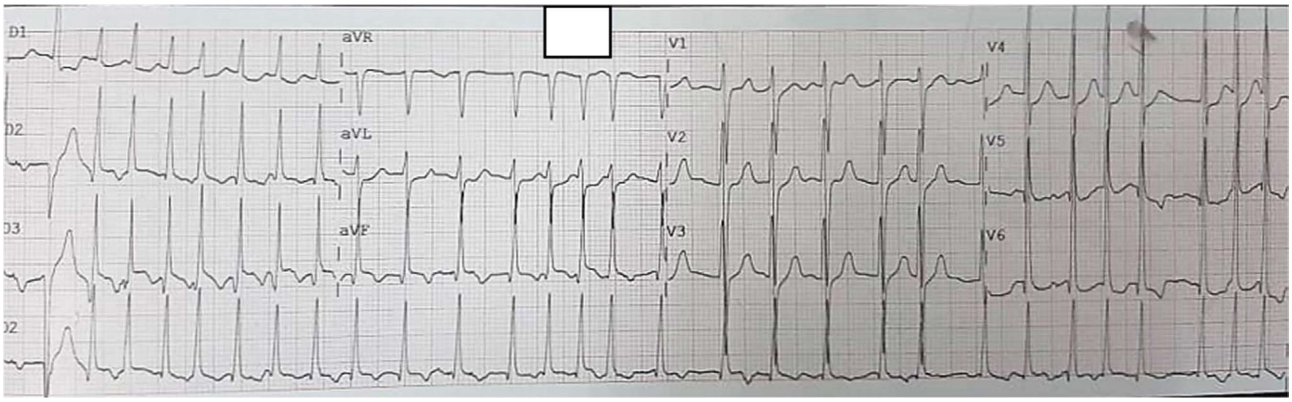

Figure 5. 12-lead resting surface electrocardiogram tracing during hospitalization of the patient showing complete tachyarrhythmia due to atrial fibrillation.

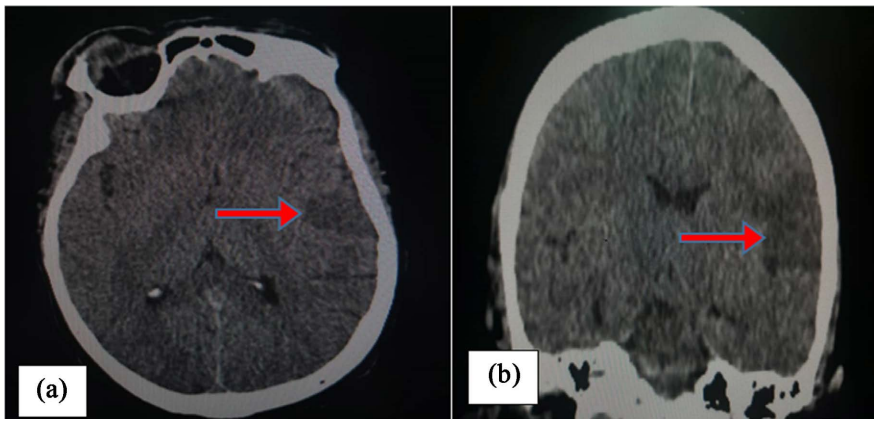

Figure 6. Cerebral computed tomography images without injection of contrast product during the patient's hospitalization, in cross section (a) and frontal (b) highlighting the ischemic cerebrovascular accident in the form of a superficial hypo dense image of the left Sylvain valley (Red Arrow).

Blood cultures were positive on day 5 of hospitalization and isolated strains of non-fermentative Gram-negative bacilli and anaerobic Gram-positive Cocci. The antibiogram showed resistance to ceftriaxone and gentamycin.

On D6 of hospitalization, the patient presented a cardio-respiratory arrest that was fatal to her despite the resuscitation measures.

3. Discussion

1) Circumstances of discovery of infective endocarditis

They are many and varied. In our present observation, it was a digestive and neurological context. Other circumstances of discovery have been reported such as acute pulmonary oedema [4], intracranial haemorrhage [5] and even splenic infarction [6]. Whether pulmonary, digestive or neurological, the circumstances of the discoveries aroused by infective endocarditis had in common to evolve in a frank febrile context. In our case the fever was 39.8˚C, it was 38.9˚C in the case of acute pulmonary oedema [4], 39˚C in the case of intracranial haemorrhage [5] and 39.5˚C in the case of splenic infarction [6]. This reminds us that a tempera-

ture ≥ 38˚C is one of the diagnostic criteria for infective endocarditis [7] [8]. Indeed, the Duke University diagnostic criteria of 1994 modified by Li in 2000 and adapted by the ESC in 2015 [7] [8] [9] [10] [11] make fever (≥38˚C) a minor diagnostic criterion.

2) Positive diagnosis of infective endocarditis

IE was retained in our patient [7] [8] [9] [10] [11] before the following major criteria: two positive blood cultures showing strains of non-fermentative Gram-negative bacilli with anaerobic Gram-positive Cocci and images intra cardiac vegetations on the calcified mitral valve remodelled on resting transthoracic echocardiography (**Figure 2**). This made any recourse to transesophageal echocardiography unnecessary [7] [10]. In addition to the two major criteria, there were minor criteria, which are fever at 39.8˚C and incidental discovery of rheumatic-like mitral disease (**Figure 2**). All this made it possible to retain in our patient the certain positive diagnosis of Osler's Sub Acute bacterial IE on the native mitral valve with a rheumatic appearance [7] [8] [10] [11]. Involvement of the valvular structures of the left heart are more frequent in IE than those of the right heart, which accounts for only 5% to 10% of cases [12] [13] [14] [15].

3) Etiological diagnosis of infective endocarditis

The etiological investigation was exhaustive in our patient. It made it possible to retain acute lithiasic cholecystitis as the entry point (**Figure 3**). The digestive gateway of IE is rare. It is generally dominated by oral diseases [16] [17]. There is also a particular entry point, often forgotten, which is intravenous drug addiction and which is particular because of its predominant male involvement, its occurrence in young adults most often, its development on a healthy heart most often and its location on the right heart [13] [18].

4) Medical treatment of infective endocarditis

It is essentially based on prolonged bactericidal antibiotic therapy. On admission, in the absence of any blood culture and antibiogram, we had instituted systematic probabilistic dual antibiotic therapy based on a 3rd generation cephalosporin type beta-Lactamine (Ceftriaxone: 4 g/day by IVD in a single dose) and an aminoglycoside (Gentamycin 160 mg/day in single infusion). This is in accordance with the IB recommendation of the European Society of Cardiology (ESC) of 2015 for infective endocarditis on native, uncomplicated valves and preserved renal function [7].

However, our case was far from uncomplicated. In addition, the mere suspicion of a digestive portal of entry raised the indication of adding at least injectable metronidazole to the treatment in anticipation of anaerobic bacteria, which most often are naturally resistant to aminoglycosides [19].

Ultimately, this serious case of IE at the digestive entry point could have benefited from probabilistic antibiotic therapy based on four molecules: Cephalosporins (3rd or 4th generation) + Aminosides + Metronidazole + Fluoroquinolones [20]. Then be adapted to the results of the antibiogram.

5) Surgical treatment of infective endocarditis

With hindsight, it appears that surgical treatment of the infective endocarditis,

from the first hours of her admission, could have improved the functional and vital prognosis of the patient. Indeed, on admission, she presented with a left heart IE with an embolic episode (brain abscess) and moderate to severe acute mitral regurgitation, without coma, shock or massive ischemic cerebrovascular accident. These were indications for surgical treatment of IE with an urgent (within the first few days) and even extremely urgent (within 24 hours) turnaround time IB recommendation of ESC 2015 [7] [10].

6) Complications of infective endocarditis

They are many and varied. They can be isolated cardiac type of valvular mutilation [4] [12] [21] [22] such as perforation of the large mitral valve in our patient. They can also be isolated extra cardiac by splenic involvement [6] [21] [22], limb ischemia by septic embolism [22] [23]. Of all isolated extra cardiac complications, cerebral complications are the most frequent [22] [24] [25] [26]. Symptomatic neurological complications occur in 15 to 30% of patients with left heart IE [7] [22] [26]. This was the case in our patient, who upon admission already presented signs of neurological localization due to a haematogenous cerebral abscess diagnosed on cerebral computed tomography (**Figure 4**). This type of complication represented 14.3% of neurological complications due to IE in Burkina Faso from 2009 to 2012 [24], 12.5% at the Center Hospitalier Universitaire de Libreville from January 2013 and December 2017 [16] and up to 30.8% in Dakar Main Hospital between January 2005 and December 2014 [27]. Alongside isolated complications, IE can be the cause of systemic complications such as immunological vasculitis responsible for Osler's whitlow, Osler's nodules, Janeway's palmoplantar erythema, Roth's spots and or even acute renal failure due to immune complex glomerulonephritis [7] [22] [28] [29] [30], but also and above all cardiovascular collapse/septic cardiovascular shock [22]. In the context of multiple infectious foci (digestive, cardiac, cerebral and a severe biological inflammatory syndrome), our patient presented a septic shock at H5 of her admission to the CICU. In addition to the cerebral abscess and the state of septic shock, the patient had further worsened her vital prognosis with the appearance of an atrial fibrillation with ventricular response at 180 beats per minute (**Figure 5**) and a massive ischemic cerebrovascular accident (**Figure 6**) at D2 and D4 of hospitalization respectively. Indeed, complications such as arrhythmias are possible in IE as reported by the Senegalese series of 39 cases of IE from January 2004 to December 2008 at the Aristide Le DANTEC Hospital, which found six cases of atrial fibrillation [31].

7) Prognosis of infective endocarditis

It remains dark. Hospital mortality in the acute phase of IE varied between 15 and 35% in recent series [14] [15] [17] [27] [32] [33]. The study carried out at the Libreville University Hospital Center from January 2013 to December 2017 found 57.1% lethality [16]. The poor prognostic factors described in IE are anemia, cardiovascular comorbidities [7] [10], immunosuppression [7] [10], the presence of a symptomatic neurological complication [7] [10] [22], the virulence

of the germ [7] [10], the antibiotic resistance of the germ, the severity and the refractory nature of the biological infectious syndrome [33], the installation of septic shock [7] [10], the appearance of congestive heart failure [4] [7] [10], localization of the bacterial graft on intracardiac material (valvular prosthesis, pacemaker probe, defibrillator) [33]. Our patient was sedentary with grade 1 obesity, passive smoking, severe anemia, significant biological infectious syndrome and two major neurological complications responsible for aphasia, hemiplegia and coma, bacterial strains that proved to be resistant to the molecules of our probabilistic antibiotic therapy. Death was the evolutionary modality in our patient on D6 of hospitalization in a context of refractory septic shock and coma.

8) Public Health issues arising from our observation

In addition to its clinical interests and particularities, our present observation highlights major public health problems which are specific to our countries in Sub-Saharan Africa (SSA) and which should be mentioned:

The problem of the double health burden: This case is an illustration on an individual scale of the fact that SSA countries are experiencing both the galloping morbidity and mortality of non-communicable diseases and the persistence of major endemics of transmissible pathologies [34] [35]. Indeed, our patient had both risk factors for non-communicable disease (passive smoking, obesity, physical inactivity) and an infectious pathology, namely Acute Articular Rheumatism complicated with rheumatic heart. Rheumatic heart disease remains a hard-skinned reality in our countries [31] while it is almost non-existent in developed countries.

The problem of delays in seeking care: Consultation at the stage of complications is common in SSA. Indeed, the patient had already consulted with a neurological complication and a valvular perforation. Preventive medicine and early consultations are sorely lacking in the SSA country.

The issue of antibiotic resistance: It is a growing public health problem in SSA [36] [37]. The widespread and misguided excessive use of antibiotics is at the origin of resistant or even multi-resistant bacterial strains [36] [37]. This was the case in our patient, rendering ineffective the only curative treatment (probabilistic antibiotic therapy) that we had proposed to her.

The problem of the insufficiency of reference health technical platforms: We should have a reference center for the management of IE [7] [10]. With teams made up of cardiologists, cardiovascular surgeons, microbiologists, radiologists, infectiologists, neurologists, neurosurgeons, anesthetists-resuscitators, with a cutting-edge imaging unit, operational cardiovascular surgery and a laboratory dedicated, for exhaustive and efficient medico-surgical management of our complicated cases of IE [7] [10] [38].

This is a necessity especially when surgical treatment is often indicated in half of patients with IE [14] [30] [32].

4. Strengths and Limitations

Our observation presents a strong point. It proves the need to set up Infectious

Endocarditis Teams in our SSA countries for rapid diagnosis and efficient medico-surgical management of complicated IE cases. This will save more lives.

However, two limitations should be noted and commented on. On the one hand, it is the absence of precise identification of the bacterial species isolated in the blood cultures: This had not been made possible due to a lack of reagent in the laboratory.

On the other hand, the lack of evidence of a match between the bacterial strains isolated from the blood cultures and the strains in the gallbladder: This could not have been done by the lack of cholecystectomy during the patient's lifetime. In addition, the proposed autopsy had been declined by the family.

5. Conclusion

Infectious endocarditis on rheumatic heart disease remains a reality in our countries. The precise and rapid identification of the germ(s) in question by blood cultures and antibiogram are of vital importance in the management because antibiotic therapy must always be adapted. IE surgery must be more available in our countries.

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Cardiology Department of the Idrissa POUYE General Hospital, Institute of Health and Development (ISED).

Consent

Informed consent was obtained from the patient's family to report this case.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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