

Case Report

Acute Ischemia of the Lower Limb in Two Patients with Tuberculosis and HIV Infection

Gogoua D. Raphaël*, Traoré Mactar, Yépié M. Armand, Kouame K. Maurice, and Anoumou Michel

Department of Orthopaedic Surgery and Traumatology, C.H.U de Treichville, Abidjan

***Corresponding author**

Gogoua D. Raphaël, Department of trauma and orthopady at CHU Treichville -18, BP 160 ABIDJAN 18, Abidjan (CÔTE D'IVOIRE), France, Tel: 00-225-05633161; Email: gogouad@yahoo.fr

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Abstract

The authors report two cases of ischemia of the lower limbs observed in the department of trauma at Treichville University Hospital. Both patients presented immunodeficiency with HIV virus 1 and 2 and were treated for pulmonary tuberculosis. No pathological history was found in both patients. The treatment was radical in both cases. It consisted in performing a 1/3 upper leg amputation and a 1/3 lower thigh amputation.

The postoperative course was uneventful in both patients and after a follow up of at least 2 years, the contralateral limb was unremarkable.

These two observations raise the issue of etiologic diagnosis, but also vascular prognosis in patients presenting these 2 conditions.

INTRODUCTION

Gangrene is a serious and irreversible complication of ischemic limbs. Ischemia is usually due to metabolic diseases and certain inflammatory diseases; Sometimes no etiology is found [1,2].

These vascular complications have been related to some infectious diseases that cause immunodeficiency [1,3]. We report two cases of limb ischemic gangrene that occurred in two HIV infected patients and who was treated for pulmonary tuberculosis.

OBSERVATIONS**Observation N°1**

Mrs. S.B, a 49 year-old woman, of precarious socioeconomic status, a non- smoking person and a non-drinker, was a patient who is with the Human Immunodeficiency Virus type 1 and 2. She was also on Tuberculosis treatment for pulmonary tuberculosis. This treatment associated Rifampicin (10mg/kg per day), Isoniazid (5 mg / kg per day) and Pyrazinamide (10 mg / kg per day). On the 7th day of the Tuberculosis treatment occurred an acute ischemia of the lower left limb characterized by a sudden onset of cyanosis from the toes, up to the lower ¼ of the left leg (Figure 1). The patient was first admitted to the cardiology department and then referred to the orthopedic department for surgical treatment. In the history of the patient, there was no notion of lower limb trauma or drug intake except

the above treatment, or history of cardiovascular disease. Physical examination revealed a poor general condition. There was no portal of entry, or skin ulceration that could evoke any etiology. The X-ray of the leg and foot concerned was normal. The chest X-ray showed diffuse macro nodular opacities on both fields. The vascular Doppler ultrasound of the lower limb found acute obliteration of tibial arterial trunks beyond the lower 1/3 of the leg whereas the echocardiogram and electrocardiogram were normal. Biologically, there was a normocytic normochromic anemia with a hemoglobin level of 9 g / dl. The sedimentation rate was not accelerated (8 mm in the first hour and 15 mm in the second hour). Blood dirt and lipids dosage were normal. There was no protein S deficiency.



Figure 1 Acute ischemia of the left foot (first case).

This patient was amputated upper 1/3 of the leg on the 6th day of admission. The intraoperative assessment of the lesions showed a threadlike artery and pedal vein containing blood clots (Figure 2). Pathological examination of the surgical specimen showed a normal artery wall and the presence of emboli due to blood clots.

The postoperative course was uneventful and Tuberculosis treatment was brought to completion. Antiretroviral treatment was started at the end of Tuberculosis treatment. After a follow-up of 24 months, the patient presented an average condition in relation with her serological constitutional susceptibility and the contralateral limb was unremarkable.

Observation N°2

Ms T.Y, a 37-year-old patient, was followed for 3 years for acquired immunodeficiency syndrome virus of type 1 and 2. Treatment was a combination of Combivir and Efavirenz. In her history she had been treated for pulmonary tuberculosis and declared cured. She was not hypertensive or diabetic or obese.

Following intermittent claudication in the right calf she had been treated for occlusive arterial disease stage 4 of the right lower limb with incipient necrosis of toes (Figure 3). It was practiced in emergency arterial unblocking + endarterectomy (from primitive iliac artery to tibial artery). But the persistent development of ischemia required a radical treatment with amputation in lower 1/3 of the thigh. The postoperative course was uneventful. And at the review 15 months later the contralateral limb was unremarkable.



Figure 2 The intraoperative assessment of the lesions showed a thread like artery and pedal vein containing blood clots.



Figure 3 Acute ischemia of the right foot (second case).



Figure 4 The second patient after desobstruction of foot artery: no successful result.

DISCUSSION

Ischemia of the lower limbs usually occurs in organisms with risk factors; particularly vascular and metabolic risk factors. Vascular complications have been little described in patients with both HIV infection and tuberculosis. Kashiwagi [4], have reported a case of cerebrovascular ischemia in an adult who presented in childhood meningeal tuberculosis. Casanova-Romain [5] reported cases of venous thrombosis of the leg in a patient with protein S deficiency and pulmonary tuberculosis. Ischemic gangrene in a patient with infectious defects, especially tuberculosis would occur with predilection for the male elderly on the basis of a sex ratio of 2 /1 (6). Risk factors are usually diseases responsible for hypercoagulability (protein S deficiency, presence of IgG and IgM antibodies) and cardiovascular diseases [6-8]. Our 2 observations involved female young adults. The second characteristic was that these two patients were infected with AIDS (type 1 and 2). One patient had been on antiviral therapy (Combivir + Efavirenz) for 3 years while the second patient had not yet started her antiviral treatment. The origin of these ischemia's raises a question. Is it a common early atheroma or phenomena associated with HIV infection or tuberculosis? It is known that biological abnormalities found in ischemia in organisms with tuberculosis have also been reported in HIV infection particularly the presence of lupus anticoagulant, the increase of plasma factor VIII and tissue activator of plasminogen. Similarly, it is now accepted that HIV infection can cause vascular complications by several potential vascular effects particularly by direct infection of endothelial cells, which express CD4 molecule¹¹⁻¹³. But also by contributing to opportunistic infections by micro- organisms with vascular tropism such as cytomegalovirus suspected to contribute to the development and progression of atherosclerotic plaques.

This combination of two these diseases could increase the risk of occurrence of thrombosis, not only venous but also arterial as suggested by many authors [6-8].

Other authors have suggested the involvement of antiretroviral drugs in the onset of ischemia of lower limbs because of the same anomalies they cause. This involvement of AntiRetroVirals cannot be mentioned in our two observations because if a patient was already on antiretroviral therapy, the other had not yet begun hers.

These 2 observations therefore confirmed with other observations of the literature review that cardiovascular

complications can occur in patients with HIV infection and tuberculosis. It is not known yet whether these complications are due to a common early atheroma or to phenomena associated with these infections. A larger cohort of patients with these symptoms and this constitutional susceptibility could help elucidate this question.

The approach to achieve this goal should consist in a systematic screening of cardiovascular risk factors in all patients with one or both defects and treat them before beginning Antiretroviral and tuberculosis treatment.

CONCLUSION

This article showed the extreme danger of the association of HIV infection and tuberculosis. This danger is represented by the acute ischemia of the limb. Its mechanism remains a question. It is why we need a larger cohort of patients with these dual diseases to clarify the issue.

And then, systematic screening of cardiovascular risk factors should be performed in any patient with one or both defects and treat them before the beginning of treatment or all along Tuberculosis treatment. This approach could help to treat preventively this ischemia.

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