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Acute Stent Thrombosis: The Reality of a Young Cameroonian Center from a Case Report

Amalia Owona[1*], Hans Mbock[2], Afassinou Yaovi Mingnazonzon[3], Hermann Yao[4], Alain Patrick Menanga[1], Maboury Diao[5]

[1]Cardiology Unit, Department of Internal Medicine and Subspecialties, Yaoundé General Hospital, Yaoundé, Cameroon
[2]Foumban Regional Hospital Annex, Foumban, Cameroon
[3]Cardiology Unit, Sylvanus Olympio University Hospital, Lomé, Togo
[4]Abidjan Institute of Cardiology, Abidjan, Côte d'Ivoire
[5]Cardiology Unit, Aristide Le Dantec Hospital, Dakar, Senegal
Email: *amowona@yahoo.fr

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Abstract

While interventional cardiology has been developing since 1977 in northern countries, Yaoundé has its first interventional cardiology center and its only interventional cardiologist established in November 2022 at the Yaounde General Hospital. Coronary angioplasty, the gold standard in the treatment of coronary syndromes, is not without formidable acute complications such as stent thrombosis that can be rapidly fatal without an immediate and optimal management. We report a case of acute stent thrombosis in a patient admitted for late presentation of an acute coronary syndrome with persistent ST-segment elevation myocardial infarction (STEMI), whose management was limited by restricted technical resources.

Keywords

Thrombosis, Stent, Acute Coronary Syndrome, Interventional Cardiology, Yaoundé

1. Introduction

Since its debut in 1977 with Dr. Gruentzig [1] [2], coronary angioplasty has revolutionized the management of coronary artery disease patients. Both its indications and techniques are in perpetual evolution. While angioplasty allows for symptomatic improvement in chronic coronary syndromes, its performance in emergency settings improves the vital prognosis in cases of acute coronary syndrome. Vascular endoprostheses, or stents, were developed during the 1980s to

address the shortcomings of balloon angioplasty. The latter was associated with 2% to 3% of occlusive dissections, up to 2% mortality, 3% to 5% myocardial infarction, 3% to 7% emergency surgery, and 30% to 40% thrombosis [3]. Since then, drug-eluting stents (DES) have taken a predominant role in coronary angioplasty and now represent the gold standard for procedures performed worldwide [4]. Although it has become rare (0.6% at 1 year, then 0.15% per year) thanks to improvements in active endoprostheses [5], stent thrombosis (ST) remains the primary potentially serious complication of angioplasty. Enhancements in adjunct pharmacological treatment, stent implantation techniques, and the devices themselves have reduced the occurrence of this life-threatening complication. The Academic Research Consortium (ARC) has classified stent-related thrombosis into acute (<24 hours), subacute (<30 days), late (between one month and one year), and very late (>1 year) [6]. We are reporting a case of an acute DES thrombosis.

2. Patient and Observation

We report the case of a 67-year-old male patient whose risk factors included male sex, hypertension, sedentary lifestyle, and obesity. He was in good health until his admission four days earlier to the emergency department of a regional hospital located 500 km from the capital, where he was admitted for precordial pain. The patient presented with prolonged constrictive pain, rated 6/10 on the visual analog scale, which occurred while walking on flat ground. The pain irradiated to the left arm and was accompanied by sweating and dyspnea, evolving over 16 hours. Upon admission, he was hemodynamically and respiratory stable, with a blood pressure of 117/87 mmHg, a heart rate of 71 bpm, a respiratory rate of 26 cpm, and oxygen saturation of 97% on room air, without clinical signs of heart failure. His weight was 97 kg and height was 1.77 m, resulting in a body mass index (BMI) of 31 kg/m^2.

Laboratory assessments revealed as some cardiac necrosis markers were unavailable in that center, the CK-MB was 9.5 ng/ml (normal range: 0 - 5 ng/ml). Total cholesterol at 241 mg/dl (normal: <200 mg/dl) and LDL-C at 201.01 mg/dl (normal: <190 mg/dl). Renal function was normal.

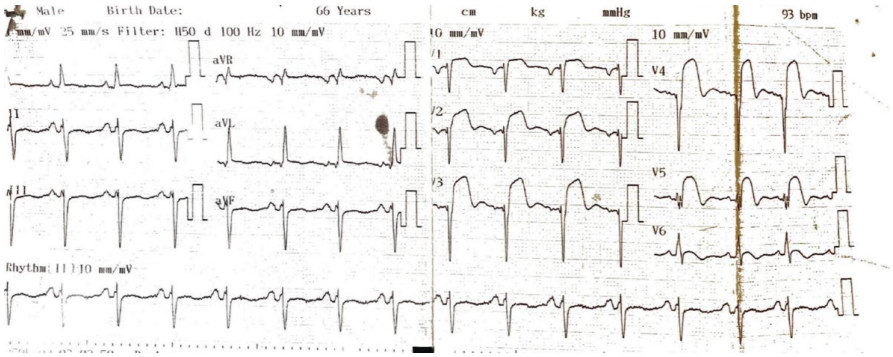

Figure 1. Electrocardiogram showing a regular sinus rhythm at 93 bpm with ST-segment elevation in the antero-septo-apicolateral leads and no reciprocal changes.

The admission electrocardiogram (Figure 1) showed a sinus rhythm at 93 beats per minute (bpm), a mean QRS axis of −56˚, and ST-segment elevation in the antero-septo-apicolateral leads without reciprocal changes (mirror images).

Transthoracic echocardiography revealed cardiac chambers of normal size, preserved left ventricular systolic function (LVEF) at 58%, and akinesia of the antero-mid-septal, septo-apical, and apico-lateral walls. Left ventricular filling pressures were estimated to be normal. The inferior vena cava was thin, and the pericardium appeared normal.

The diagnosis of late-presentation ST-segment elevation myocardial infarction (STEMI) was established. Optimal medical therapy made of a loading dose of Aspirin 300mg orally associated with a loading dose of Clopidogrel 600 mg and low molecular weight Heparin 1 mg/kg/12 hours subcutaneous were initiated. The day following his admission, a Bisoprolol 2.5 mg daily and Perindopril 5mg have been associated and the patient was transferred to the Yaoundé General Hospital.

With the approval of the consent form, the patient was admitted to the catheterization laboratory on the fifth day following the onset of chest pain. Coronary angiography was performed via a 6F radial access according to standard protocol. It revealed an atheromatous coronary network with two-vessel disease involving the circumflex (Cx) and the left anterior descending (LAD) arteries. The lesion in the mid-circumflex artery was considered chronic, with the distal portion being supplied by collateral circulation from the right coronary network. The proximal LAD lesion was identified as the culprit vessel, involving a bifurcation lesion with the first diagonal branch (D1), classified as Medina 1.1.1 (Figure 2).

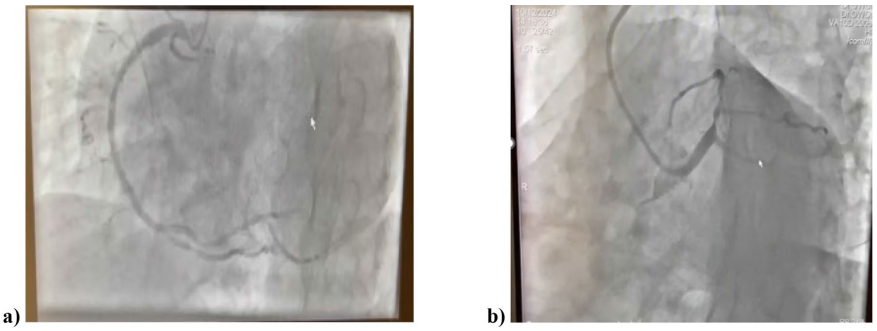

Figure 2. Coronary angiography via 6F radial access showing an atheromatous coronary network: a) LAO: atheromatous right coronary network with ostial stenosis of a small-caliber posterior descending artery; b) LAO 40˚-caudal 40˚, "Spider" view: left coronary network showing an atheromatous left main stem with a long stenosis of the proximal LAD (TIMI 3 flow) involving a bifurcation lesion with the D1, which presents a long stenosis starting from its ostium, and a mid-circumflex (Cx) stenosis with TIMI 0 flow.

Provisional stenting of the bifurcation lesion was decided upon immediately. Two guidewires were advanced into the LAD and the D1. Balloon (2.75 × 15 mm) dilatation at 12 then 14 and 16 atm of the D1 was performed with a good angiographic result. Subsequently, direct implantation of a drug-eluting stent (DES) 3.75 × 18 mm at 12 then 14atm was carried out in the LAD; this yielded a good

angiographic result but was associated with a total occlusion of the D1. This occlusion triggered acute retrosternal chest pain rated 7/10, without electrical or hemodynamic changes, indicating the need for D1 dilatation with a DES. The administration of morphine combined with nitrates led to pain relief.

Initially, we proceeded with a proximal optimization of the LAD stent with a NC balloon 3.5 × 8mm, which resulted in acute intra-stent thrombosis of the LAD with TIMI 0 flow (**Figure 3**). The clinical course was marked on the table by the recurrence of intense chest pain (8/10) accompanied by profuse sweating, without electrical modifications.

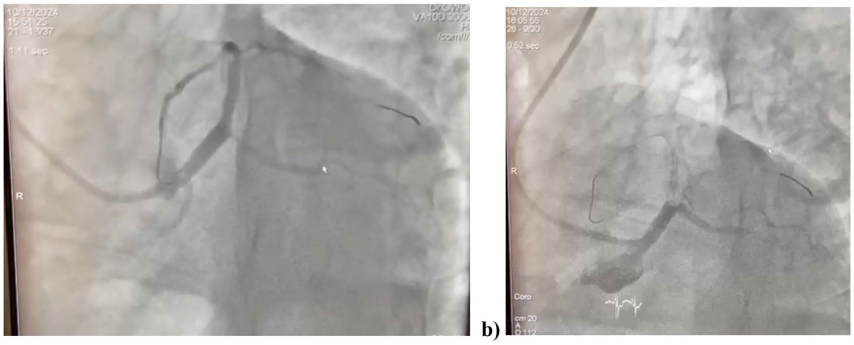

Figure 3. Coronary angiography via 6F radial access: a) LAO 38˚-caudal 39˚, "Spider" view: good angiographic result following direct stenting and total occlusion of the D1 with TIMI 0 flow; b) LAO 38˚-caudal 39˚, "Spider" view: acute intra-stent thrombosis of the LAD with TIMI 0 flow.

The procedure was aborted, and the patient was transferred to the cardiac intensive care unit for intravenous thrombolysis. Intravenous thrombolysis using Streptokinase 1,500,000 IU through electrical syringe over an hour associated with 100 mg of intravenous hydrocortisone was initiated 34 minutes after admission associated with loading doses of Clopidogrel 600 mg and Aspirin 300 mg orally and 6 mg of morphine. Serial electrocardiograms remained unchanged. Echocardiography revealed antero-septo-apico-lateral akinesia with a reduced LVEF of 43%. The pericardium remained normal. The post-thrombolysis period was unremarkable, characterized by pain relief achieved through the use of morphine derivatives.

Further questioning revealed non-adherence to dual antiplatelet therapy (DAPT). Apart from the loading doses, the patient had not taken the maintenance doses, nor had they been administered prior to the coronary angiography.

At 48 hours, Clopidogrel was replaced by Ticagrelor with a loading dose. Follow-up coronary angiographies were performed via 6F radial access on the third and fourteenth days; these demonstrated persistent ST of the LAD with TIMI 0 flow, but recovery of TIMI 3 flow in the D1 (**Figure 4**).

The clinical course in the department was characterized by the resolution of chest pain and the maintenance of hemodynamic stability. Both the electrocardiogram and echocardiography findings remained unchanged.

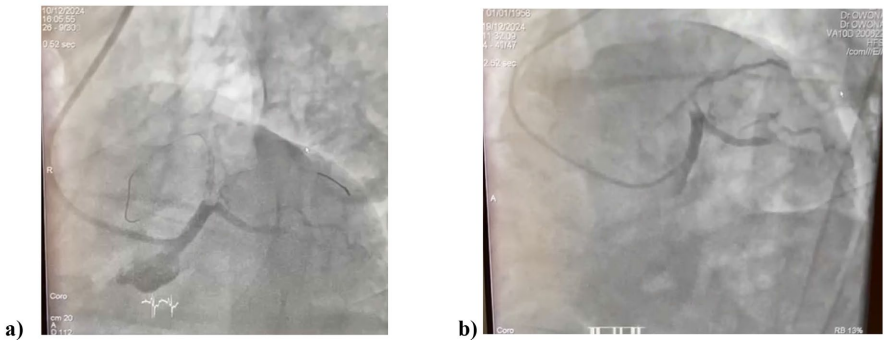

Figure 4. Coronary angiography on Day 14 via 6F radial access: a) LAO 38˚-caudal 39˚, "Spider" view: acute intra-stent thrombosis of the LAD with TIMI 0 flow; b) Follow-up coronary angiography at 72 hours in LAO 38˚-caudal 39˚, "Spider" view: failure of thrombolysis with persistent ST of the LAD (TIMI 0 flow) and recovery of TIMI 3 flow in the D1.

The patient was subsequently discharged on Asprin 100 mg/day, Ticagrelor 90 mg twice a day, Lasilix 80 mg daily, Spironolactone 25 mg/day, Dapalifozin 10 mg/day, Bisoprolo 1.25 mg twice a day and Perindropril 5 mg/day. His follow-up was marked by recurrent episodes of heart failure decompensation, and the patient passed away five months after discharge.

3. Discussion

ST is more frequently observed in the context of acute coronary syndrome compared to elective angioplasty. The most common presentation is myocardial infarction with a frequently significant thrombotic burden, and observational registers report a mortality rate of 20% to 40% for early ST and 5% to 10% for late and very late ST [7].

Beyond the coronary lesion itself and its complexity, the patient remains at the center of this assessment, with risk factors such as diabetes, advanced age, left ventricular dysfunction, renal failure, genetic profile, or adherence to antithrombotic treatment; premature discontinuation or suspension of the latter are recognized as major drivers of ST [8] [9].

Intrastent thrombosis requires urgent management, involving the restoration of effective coronary flow and the intensification of antithrombotic therapy. The recent French PESTO registry, published by the Clermont-Ferrand team, confirms the major benefit of performing endocoronary imaging (OCT: Optical Coherence Tomography) in cases of ST. Abnormalities are very frequently identified, including, in order of frequency: stent malapposition, new progressive atherosclerotic lesions, poor stent expansion, coronary invagination, endothelialization defects with uncovered struts, or intimal hyperplasia [10].

In the acute phase, several reperfusion strategies could have been considered: manual thrombectomy, intracoronary administration of glycoprotein IIb/IIIa inhibitors (GPI), and an attempt to cross the lesion again with a second guidewire in the LAD. Lacking both a thrombectomy device and GPIs, and with the goal of reducing the thrombotic burden, intravenous thrombolysis was our only recourse,

although it proved unsuccessful despite being initiated within 34 minutes. The speed and quality of reperfusion differentiate thrombolytic agents from one another.

In the GUSTO study, the primary criteria for thrombolytic treatment efficacy were: early administration (less than 6 hours), the subject's age (poor prognosis after 75 years), and the absence of cardiogenic shock or a history of coronary artery bypass grafting [11]. The ability of thrombolytics to achieve the highest possible rate of TIMI 3 grade flow as early as possible is decisive, given that beyond 180 minutes, reperfusion rates tend to equalize regardless of the thrombolytic agent used. Indeed, it appears that this type of optimal reperfusion flow is obtained in only 32% of cases with streptokinase, compared to 54% with alteplase and 63% with tenecteplase, which constitutes one of the major limitations of thrombolysis [12] especially in our resource-limited context where Alteplase and Tenecteplase are not only rare but unaffordable for our patients.

Another major factor found in all studies concerns antiplatelet therapy, primarily its premature discontinuation due to medical reasons or poor compliance, as was the case in our experience. The new P2Y12 receptor inhibitors (Prasugrel and Ticagrelor) are first-line treatments after ST due to their superior efficacy. In our context, these drugs had not yet received marketing authorization; furthermore, as there is no established evidence for their indication in conjunction with intravenous thrombolysis, we initially felt comfortable with Clopidogrel, which we later replaced with Ticagrelor.

In our case, the primary cause of ST might be therapeutic non-adherence though other stent thrombosis associated factors such as stent underexpansion, malapposition, and bifurcation-related technical factors that could not be assessed without intracoronary imaging are also plausible. Since our patient met all the criteria for successful thrombolysis, we are unable to explain its failure. We note, however, that this is a case of thrombolysis performed immediately after the angioplasty, for which we have no prior perspective based on the current literature.

4. Conclusion

Stent thrombosis remains the "Achilles' heel" of angioplasty. While this complication is infrequent, it has dramatic consequences when it occurs. A comprehensive understanding of its anatomical, biological, humoral, and clinical predictive factors should improve prevention. Most importantly, as illustrated by our case, antiplatelet therapy remains the cornerstone of this prevention.

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Authors' Contributions

All authors contributed to the writing of this work. Consequently, all authors as-

sume responsibility for all aspects of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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