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SCIENTIFIC EDITORIAL

Acute coronary syndrome in HIV-infected patients. Does it differ from that in the general population?

Syndrome coronaire aigu chez le patient infecté par le VIH : existent-ils des différences avec la population générale ?

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Received 21 October 2010; accepted 21 October 2010

Available online 26 November 2010

KEYWORDS

Acute coronary syndrome;
 Percutaneous coronary intervention;
 Human immunodeficiency virus;
 Antiretroviral therapy

MOTS CLÉS

Syndrome coronaire aigu ;
 Angioplastie coronaire ;
 Virus de l'immunodéficience humaine ;
 Traitement antirétroviral

Cardiovascular disease, and particularly coronary artery disease-related morbidity, has become the fourth most common cause of death in HIV-infected patients in France [1] and the third most common cause in the United States [2]. Since the advent of a potent antiretroviral therapy in the mid 1990s, HIV infection has become a chronic rather than a fatal disease. The life expectancy of a young HIV-infected patient without hepatitis co-infection is today more than 35 years [3]. In developed countries, the causes of death in HIV-infected patients besides AIDS-related morbidity (still the primary cause of death usually related to late diagnosis of HIV) are similar to those of uninfected patients (e.g., non-AIDS neoplastic disease, cardiovascular disease, hepatitis). However, these complications tend to emerge earlier (around 10 years) than in the general population, suggesting an acceleration of ageing in patients infected with HIV. Premature and acceleration of atherosclerosis are, therefore, an emerging complication in HIV.

Recently, Lang et al. [4] estimated the incidence of myocardial infarction in HIV-infected patients in comparison with the general population. The sex- and age-standardized morbidity ratio was estimated as 1.5 (95% confidence interval [CI] 1.3–1.7) overall, 1.4 (95% CI 1.3–1.6) in men and 2.7 (95% CI 1.8–3.9) in women. Similar results were observed in the North American population [5]. Of note is the young age at which myocardial infarction occurred. In two recent French studies, the median age at which a first episode of acute coronary syndrome (ACS) occurred was around 48 years [6,7]. Besides the increased incidence and premature occurrence of myocardial infarction in the HIV-infected population compared with the general population, are there specific risk factors that can explain these differences?

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When considering cardiovascular risk factors, in the prognosis of acute coronary syndrome in HIV-infected patients (PACS) study [7], HIV-infected patients with ACS were frequently current smokers (60%), dyslipidaemic (50%), hypertensive (20%) and diabetic (10%), but these differences vanished when the population was matched for age with uninfected patients [7]. Only hypertriglyceridaemia and illicit drug use (particularly cocaine) were more frequent in HIV-infected patients with an ACS compared with the general population. The question is, do these factors explain the higher rate and premature occurrence of myocardial infarction in HIV-infected patients? The process is likely to be multifactorial, involving a combination of chronic HIV infection along with low-grade persistent inflammation, immune dysregulation, antiretroviral therapy and related metabolic disturbances such as insulin resistance, dyslipidaemia and lipodystrophy. In the PACS study [7], the angiographic characteristics of ACS in HIV-infected patients were similar to those of uninfected patients, with predominant single-vessel disease and preserved left ventricular ejection fraction, in agreement with the characteristics reported in the young population with ACS.

The results of immediate coronary revascularization, whether by percutaneous coronary intervention and/or coronary artery bypass graft surgery, are excellent in this population, with no difference with the general population [7–13]. In the PACS study [7], the success of PCI was greater than 94%, with no difference in the rates of acute stent thrombosis. One acute stent thrombosis occurred at day 5 in an HIV-infected patient and another at day 1 in an uninfected patient. No additional cases of stent thrombosis occurred in the subsequent 12 months. The HIV-infected patients were well treated for HIV, with 69% of the entire cohort having an HIV viral load less than 200 copies/mL, suggesting that these patients adhered to their therapeutic drug regimens. These data should allay fears about implanting a drug-eluting stent followed by long-term treatment with dual antiplatelet therapy in this population. However, after 1-year of follow-up in the PACS study, recurrent ACS was more frequent in the HIV-infected population compared with the controls (hazard ratio 6.5, 95% CI 1.7–23.9), but of the rates of recurrent revascularization did not differ. This discrepancy was due to the higher rate of silent myocardial ischaemia detected in the controls. In fact, the rate of stress testing after ACS was more frequent in the controls than in patients infected with HIV (80 vs. 63%, $p=0.004$), and this could have led to a bias in the rate of recurrent revascularization, with an overestimation in the controls. Indeed, the rate of repeat urgent revascularization driven by recurrent ACS was higher in HIV-infected patients (odds ratio 3.29, 95% CI 0.94–11.53, $p=0.06$). The rate of clinical restenosis??? Surprisingly, the rate of smoking cessation was lower in HIV-infected patients than in controls (51 vs. 80%, $p=0.002$), along with persistent atherogenic dyslipidaemia in HIV-infected patients (total cholesterol 2.22 ± 2.4 vs. 1.73 ± 4.8 g/L, $p=0.046$; non-high-density lipoprotein cholesterol 1.53 ± 4.7 vs. 1.25 ± 4.4 g/L, $p=0.020$; total cholesterol/high-density lipoprotein cholesterol 5.3 ± 3.3 vs. 3.8 ± 1.3 , $p=0.004$). Whether these two factors will lead to an increased rate of recurrent ACS in the long-term will be evaluated after 3 years of follow-up, the data for which will become available next year.

In conclusion, ACS is more frequent and occurs earlier in the HIV-infected population than in the general population. Traditional risk factors are frequent, particularly tobacco smoking. Screening for illicit drug use, especially cocaine, should be done immediately after the onset of symptoms in this young population. Coronary revascularization, whether percutaneous coronary intervention and/or coronary artery bypass graft, can be performed safely and with excellent initial results, with no reports of an increased rate of stent thrombosis. The mid-term prognosis seems to be worse in the HIV-infected population, with a higher rate of recurrent ACS, suggesting failure to control the atherothrombotic process. This is probably due to the persistently higher rate of current smokers and uncontrolled dyslipidaemia after ACS. However, many factors could influence the prognosis, such as the HIV infection itself, low-grade inflammation or antiretroviral therapy. In secondary prevention, awareness of smoking cessation and achieving target lipid concentrations (two modifiable cardiovascular risk factors) should be made in the HIV-infected population to decrease the higher rate of recurrent ischaemic events.

Conflicts of interest statement

No conflict of interest.

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