

Clinical Episode Suggestive of Left Main Coronary Artery Spasm After Gingival Anesthetic Infiltration

To the Editor:

In 1959, Prinzmetal described the classic symptoms of the variant of angina pectoris that occurs secondary to coronary vasospasm and which is accompanied by electrocardiographically observable ST segment elevation. Subsequently, coronary artery spasm has been reported in association with numerous factors. It may present not only as angina, but also as myocardial infarction. Moreover, it can manifest electrocardiographically as a subendocardial lesion (i.e., with ST segment depression). We present the case of a 23-year-old woman who arrived at the emergency department at our center with severe oppressive central chest pain that had been induced by the local gingival injection of an anesthetic along with a vasoconstrictor (i.e., 36 µg of adrenaline, 36 µg of noradrenaline acid tartrate, and 36 mg of lidocaine hydrochloride) during a stomatology consultation. She had no known cardiovascular risk factors. On her arrival at the emergency department, while still in pain, an electrocardiogram (ECG) was recorded. The ECG showed the presence of a subendocardial lesion, which was characterized by alterations of up to 3 mm in all limb and precordial leads and by an ST segment elevation of 2 mm in lead aVR, thereby suggesting the presence of an arterial trunk lesion (Figure 1). Following sublingual nitroglycerin administration, the pain decreased within 2 hours and the ECG normalized, with resolution of the alterations described above. Echocardiographic investigation showed severe anteroapical hypokinesia and mitral insufficiency in the left ventricle. Emergency coronary angiography showed normal coronary arteries. Echocardiography carried out 6 days after admission demonstrated a normal-sized left ventricle with preserved systolic function, no segmental anomalies, and minor mitral insufficiency. Subsequently, the patient visited her dentist for essential dental treatment on two occasions without the occurrence of a similar incident (a local anesthetic was used

without vasoconstrictors).

Left coronary artery spasm is an infrequent cause of angina. It is characterized by generalized ST segment depression in all leads due to subtotal vessel occlusion.¹ The appearance is different from that of typical vasospastic angina, which is usually associated with a subendocardial lesion caused by total occlusion of the affected area and which results in transmural ischemia. Numerous factors can give rise to coronary spasm. Some are chemical, such as exposure to ergonovine, acetylcholine, serotonin, cocaine, noradrenaline, substances induced by hyperventilation, or anesthetics. And some are physical, such as cold exposure and exercise.

In the present case, it was not possible to demonstrate arterial spasm in any segment of the heart during cardiac investigation as the patient was asymptomatic at that time and the ECG had practically returned to normal. Nevertheless, because clinical symptoms were triggered immediately by local vasoconstrictor administration and because angiographically normal coronary arteries were observed in the presence of an ECG that was characteristic of a left coronary artery lesion, we concluded that the symptoms had been caused by spasm of this artery. Earlier studies have detailed the systemic cardiovascular effects of vasoconstrictors (usually noradrenaline and adrenaline) administered along with the local anesthetics used in stomatology.^{2,3} The use of vasoconstrictors is generally contraindicated in patients with unstable angina, recent infarctions, arterial hypertension, cardiac insufficiency, or poorly controlled arrhythmias. Although ST segment depression has been observed during the administration of local anesthetics, it does not appear to be significant.⁴

Given the nature of dental practice (i.e., generally private), it is very difficult to determine the incidence of any particular clinical complication that occurs during treatment. Nevertheless, data collected from questionnaires completed by 302 British dentists indicate that the annual incidence of chest pain indicative of angina is 0.17 cases per dentist, and that of infarction and cardiac arrest is 0.003 and 0.002 cases per dentist, respectively.⁵ Spanish and international dental journals have published guidelines on how to deal with patients with cardiopathies, especially those with coronary disease.^{6,7} However, the previously mentioned

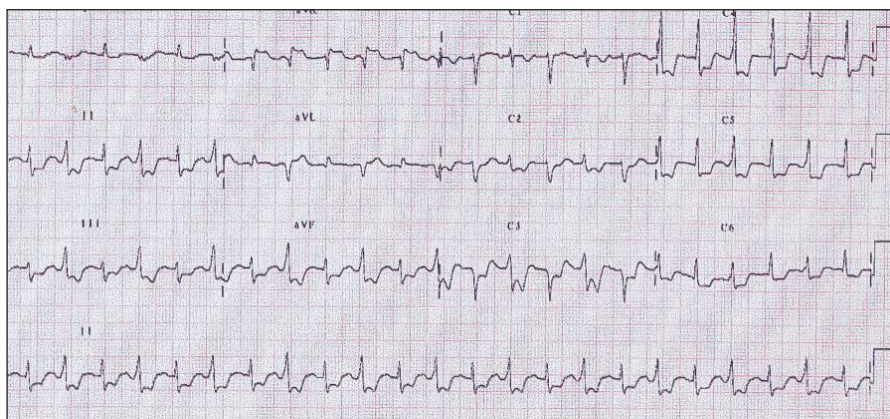


Fig. 1. Electrocardiogram recorded during an episode of chest pain.

study found that 50% of British dentists surveyed do not know how to insert an oral airway or how to perform an intravenous injection.⁵ On the other hand, our case illustrates that it is possible to encounter potentially serious cardiac complications in patients who have no relevant disease history or risk factors.

We conclude that it is important to be aware of the possibility that coronary vasospasm can occur in patients who have undergone the local administration of anesthetic-vasoconstrictor combinations, including young patients with no previous history of disease. It is important that the patient's disease history is known and that the combined use of anesthetics and vasoconstrictors is, as far as possible, reduced or avoided in patients with coronary disease. Clinics using these drugs should have adequate resources available for dealing with such situations (e.g. sublingual nitroglycerin) and professional staff should undergo training in their prevention and management.⁸

Susana González Enríquez^a, José M. de la Torre Hernández,^b and Fermín Sainz Laso^a

^aServicio de Cardiología, Hospital Universitario Marqués de Valdecilla, Santander, ^bUnidad de Hemodinámica, Hospital Universitario Marqués de Valdecilla, Santander, Spain.

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