A case of takotsubo cardiomyopathy precipitated by lumiracoxib, a selective COX-2 inhibitor

J KER, CJ VAN WYK

Summary
Considerable controversy exists regarding the cardiovascular safety of COX-2 inhibitors, both in patients with and without established cardiovascular disease. Currently, the major focus is on thrombotic complications presenting as myocardial infarction. In this report, we present a case of takotsubo cardiomyopathy, also known as the apical ballooning syndrome, precipitated by the use of lumiracoxib. We are concerned that this might be the first case of COX-2 inhibitor-induced apical ballooning syndrome and that more may follow.

Case report
A 65-year-old Caucasian woman had an acute onset of apical ballooning syndrome (takotsubo cardiomyopathy) 48 hours after the initiation of lumiracoxib (Prexige®). The patient underwent a routine cardiac examination 48 hours prior to presentation because of essential hypertension, which had been well controlled with 10 mg per day of ramipril for the past two years.

The clinical examination, echocardiogram, carotid-IMT measurement and pro-BNP level were normal. The patient had lower back pain after mild trauma and lumiracoxib (Prexige®) was administered at a dose of 400 mg mane po for three days. After the second dose (48 hours) the patient presented with chest pain, dyspnea and a troponin-T level of 0.08 ng/ml.

The ECG (Fig. 2) demonstrated striking T-wave inversion, as well as mild ST-segment elevation in leads V2 and V3. The pro-BNP level rose to 10.01 ng/ml from 0.134 ng/ml measured 48 hours earlier. Echocardiography (Fig. 1) demonstrated apical akinesia with the typical appearance of apical ballooning syndrome. Urgent coronary angiography was performed. This demonstrated normal epicardial coronary arteries and the ventriculogram confirmed severe apical akinesia with ballooning and a left ventricular ejection fraction of 35%.

The patient was managed with clexane 80 mg sc twice daily, disprin cv 100 mg once daily and plavix 75 mg once daily. The lumiracoxib (Prexige®) was withdrawn immediately. Ten days later the pro-BNP level decreased to 0.628 ng/ml and the apical akinesia resolved.

Discussion
Transient left ventricular apical ballooning syndrome, also known as takotsubo cardiomyopathy, is a cardiac syndrome characterised by chest pain, electrocardiographic changes, release of myocardial enzymes and apical akinesia in the absence of obstructive coronary artery disease. This condition was first described by Sato in 1990, who chose the term ‘takotsubo’ because the end-systolic left ventriculogram resembled a takotsubo, a Japanese pot with a round bottom and narrow neck, used for trapping octopuses.

The true prevalence of this peculiar syndrome remains uncertain. It was originally reported in Japan. Several cases have since appeared recently in Caucasians in Europe and North America. This condition has a female predominance and physical and/or emotional stressors are recognised as important predisposing factors.

Apical ballooning syndrome has been described in patients with hypertrophic obstructive cardiomyopathy, in the intensive care unit with a variety of systemic illnesses, after subarachnoid haemorrhage and as a post-operative phenomenon. However, this is the first report of this syndrome associated with the use of COX-2 inhibitors, specifically lumiracoxib. Recently, a new midventricular variant of takotsubo cardiomyopathy has been described, however the exact pathophysiology of both variants still remains elusive.

Thrombotic complications, presenting as myocardial infarc-
tion and/or cerebrovascular events, has been observed with rofecoxib, celecoxib, valdecoxib and its intravenous prodrug parecoxib. This is the first report of a possible association between COX-2 inhibitors and apical ballooning syndrome in our practice and a search of the Medline database also failed to reveal any such cases.

References

Fig. 2. ECG of the patient.