A 19 year old pregnant woman presented to the coronary care unit with an acute anterior myocardial infarction. She was treated with primary percutaneous transluminal coronary angioplasty of the proximal left anterior descending coronary artery. Ultrasound examination showed patent foramen ovale (PFO) and atrial septal aneurysm. The patient was a heterozygote carrier of factor V Leiden. Despite the lack of a clear clue, it was considered that the pathophysiological cause of this infarction was a paradoxical embolus in the left coronary artery. Pregnancy and factor V Leiden carrierhip are associated with increased risk of venous thromboembolism and the association between PFO and atrial septal aneurysm is a strong risk factor for systemic embolisation.

A 19 year old white woman was admitted to our coronary care unit for an acute myocardial infarction, with two hours' delay from the onset of chest pain. The standard ECG showed ST segment elevation in the anterolateral leads. The only cardiovascular risk factor was cigarette smoking. She reported no drug misuse. She noted she had not had menses for several months, without taking oral contraceptives. A diagnostic test for pregnancy was positive (serum β human chorionic gonadotropin > 10000 UI/l).

The patient was immediately moved to the cardiac catheterisation laboratory for primary percutaneous transluminal coronary angioplasty (PTCA). Shielding was placed on the abdomen to protect the fetus from radiation. Coronary angiography showed complete occlusion of the left anterior descending coronary artery (LAD) in its proximal tract (fig 1A). The other coronary arteries were normal. After crossing the occlusion with a soft guidewire, primary PTCA of the LAD was performed with an optimal result (no residual stenosis and TIMI (thrombolysis in myocardial infarction) grade 3 flow). No “dog bone” effect was noted during the inflation of the balloon and, besides the occlusion, the LAD had no signs of atherosclerosis. However, the control angiogram showed that an important first diagonal branch, originating after the occlusion of the LAD, was occluded; it was treated with PTCA, achieving a good final result (fig 1B). The creatine kinase MB fraction peak was 655 ng/ml in the seventh hour. An echocardiogram, executed in the coronary care unit, showed akinesis of the septum, anterior wall, and apex; ejection fraction was 38%; and an atrial septal aneurysm was present with clear evidence of a patent foramen ovale (PFO) with left to right shunt.

An ultrasound abdominal evaluation showed that the gestational age of the fetus was approximately 22 weeks. A diagnostic investigation for a hypercoagulable state examined antithrombin III, proteins C and S, prothrombin time, partial thromboplastin time, fibrinogen and homocysteine concentrations, cardiolipin antibodies, and genotype analysis for factor II and factor V Leiden. All were within normal limits apart from heterozygosis for factor V Leiden mutation. The patient was discharged in good clinical condition and had an uneventful delivery by caesarean section during the 31st week of pregnancy. She refused any other examination or intervention after the delivery.

DISCUSSION
Myocardial infarction during pregnancy is a very unusual event that occurs in 1 in 20 000–30 000 deliveries. Most commonly, it happens during the third trimester and puerperium of the first and second pregnancy. The average age at occurrence is 32 years. Coronary angiograms have not shown signs of atherosclerosis in more than a half of the cases. The most likely mechanisms underlying this event are

Abbreviations: LAD, left anterior descending coronary artery; PFO, patent foramen ovale; PTCA, percutaneous transluminal coronary angioplasty; TIMI, thrombolysis in myocardial infarction
coronary dissection, secondary to hormonal changes, coronary artery spasm, and thrombosis.\textsuperscript{1, 2}

No previous case report has referred to a possible relation between PFO and myocardial infarction during pregnancy, although several reports have associated acute myocardial infarction with paradoxical embolism.\textsuperscript{3, 4} Our patient was particularly young and she was in her second trimester of pregnancy—both these characteristics are atypical with respect to those previously reported.\textsuperscript{1, 7} There was no evidence of atherosclerotic disease in her coronary arteries; however, the LAD was totally occluded and, after PTCA on this vessel, an occlusion of its branch appeared. Both findings are consistent with embolus of thrombus.\textsuperscript{5} In our opinion, this occlusion was caused by a paradoxical embolus coming from the venous system. Indeed, pregnancy is associated with a fourfold increased risk of venous thrombosis.\textsuperscript{6} Furthermore, the patient was a carrier of factor V Leiden, which is a well known risk factor for venous thromboembolism.\textsuperscript{7} Finally, her ultrasound examination showed an association between PFO and atrial septal aneurysm, which is known to increase fivefold the risk of systemic embolisation.\textsuperscript{8}

Unfortunately the patient refused any other diagnostic examination for a better investigation of the PFO (transoesophageal echocardiography) and to detect possible signs of systemic embolisation (brain magnetic resonance, transcranial echography). Moreover, she declined percutaneous closure of the PFO.\textsuperscript{9}

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