Antero-lateral ST-segment elevation myocardial infarction in a patient with stent in the left anterior descending implanted ten years earlier and angiographic control two weeks earlier

Zawał ściany przednio-bocznej mięśnia sercowego u chorej po wszczepieniu stentu do gałęzi przedniej zstępującej przed dziesięciu laty, kontrolowanej angiograficznie dwa tygodnie wcześniej

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Abstract

The article refers to a case of a woman who underwent an anterior wall ST-segment elevation myocardial infarction (STEMI) and angioplasty left anterior descending (LAD) 10 years ago. The angiography that she underwent 2 weeks before the next STEMI did not show any relevant atherosclerotic changes. Nonetheless, she was hospitalized 14 days later due to a new ST elevation in a scar after pre-existing myocardial infarction. After a coronarography we identified an occlusion LAD below the stent. In this publication we discuss the problem of the lack of a simple, cheap and unmistakable method that would enable us to identify an unstable sclerotic plaque.

Key words: infarction in a scar, unstable sclerotic plaque, myocardial infarction in an artery without significant stenosis

Streszczenie

W artykule przedstawiono przypadek kobiety po przebytym przed 10 laty zawale mięśnia sercowego z uniesieniem odcinka ST (ST-*segment elevation myocardial infarction* – STEMI) ściany przedniej i po odroczonej angioplastyce gałęzi przedniej zstępującej (GPZ), u której 2 tygodnie przed kolejnym STEMI wykonano angiografię i nie stwierdzono zmian zwężających światło tętnicy. Czternaście dni później pacjentka trafiła do szpitala ze świeżym uniesieniem odcinka ST w obszarze blizny, a w wykonanej koronarografii rozpoznano okluzję GPZ poniżej stentu. W publikacji poruszono problem braku prostej, taniej i jednoznacznej metody identyfikacji tej blaszki miażdżycowej, która w niedalekiej przyszłości stała się podłożem dla wystąpienia ostrego zespołu wieńcowego.

Słowa kluczowe: zawał w bliźnie, niestabilna blaszka miażdżycowa, zawał w tętnicy bez istotnego zwężania jej światła

Case report

We present a case of a 50-year-old female patient with risk factors of ischemic heart disease – hypercholesterolemia (LDL 157 mg/dl a year earlier despite treatment with statin) and moderate hypertension. She was admitted to the Catheterization Laboratory of the Multidisciplinary Regional Hospital in Ciechanów because of ongoing chest pain lasting 6 h with coexisting ST-segment elevation (Figure 1) in leads I, aVL and V1-6 and ST-segment depression in leads II, III and aVF. Previous electrocardiograms (Figure 2) showed permanent changes in the form of persistent ST-segment elevation after myocardial infarction of the anterior wall 10 years earlier treated with thrombolysis (streptokinase – Sep 1999) and a different percutaneous coronary intervention (PCI) of the left anterior descending (LAD) (Oct 1999) performed after confirmation

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of contractility reserve in the antero-lateral wall with the dobutamine test.

Two weeks earlier the patient was electively admitted to the Department of Cardiology because of atypical chest pain and positive ambulatory exercise test. Elective coronary angiography (Figure 3) performed at that time showed good flow in the previously implanted stent to the LAD with only subtle neointimal proliferation. Other coronary arteries were normal. Both the clinical picture and comparative analysis of the ECGs obtained at that time with

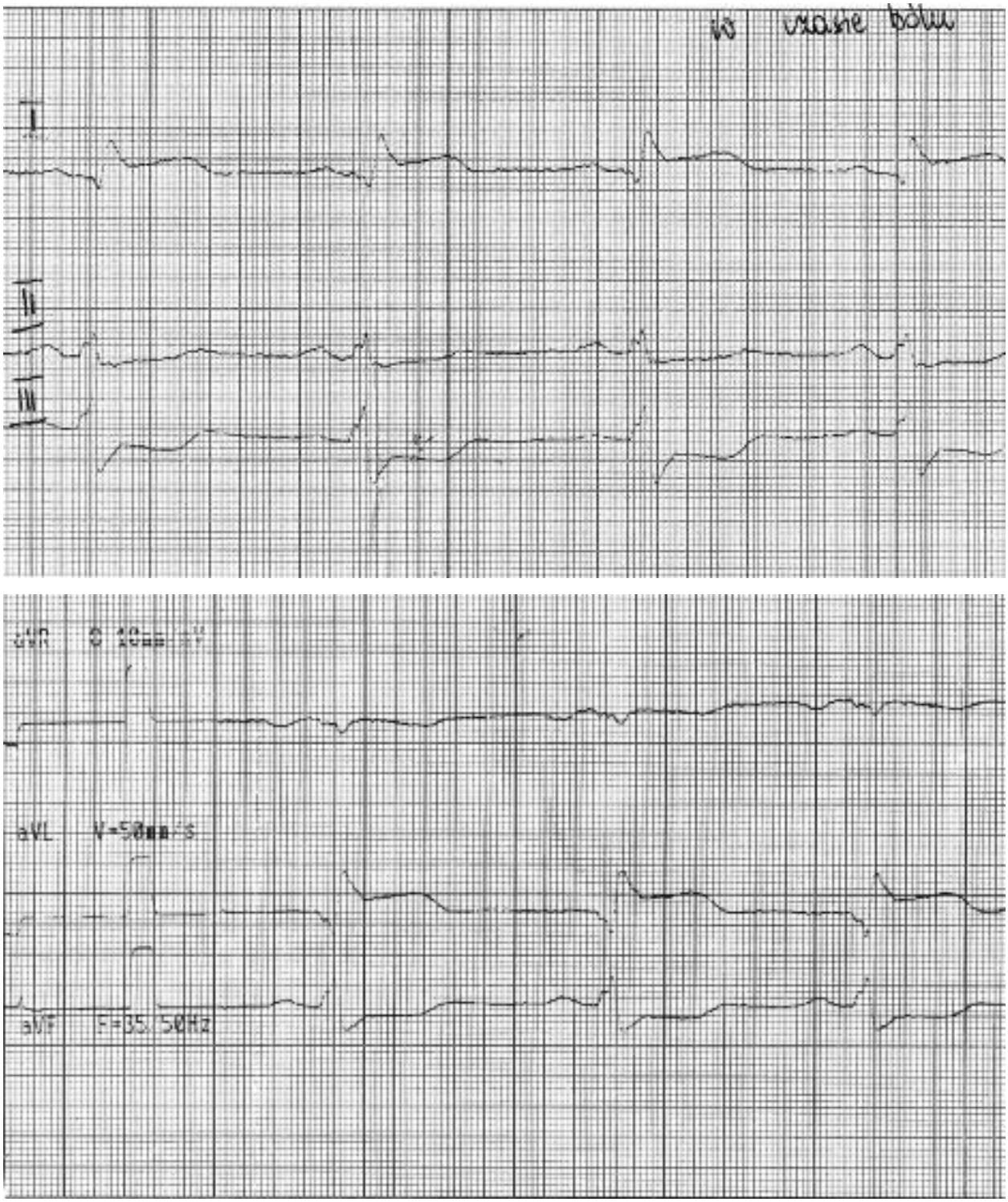

Fig. 1. First ECG after admission to the hospital
Ryc. 1. *Pierwsze EKG wykonane po przyjeździe do szpitala*

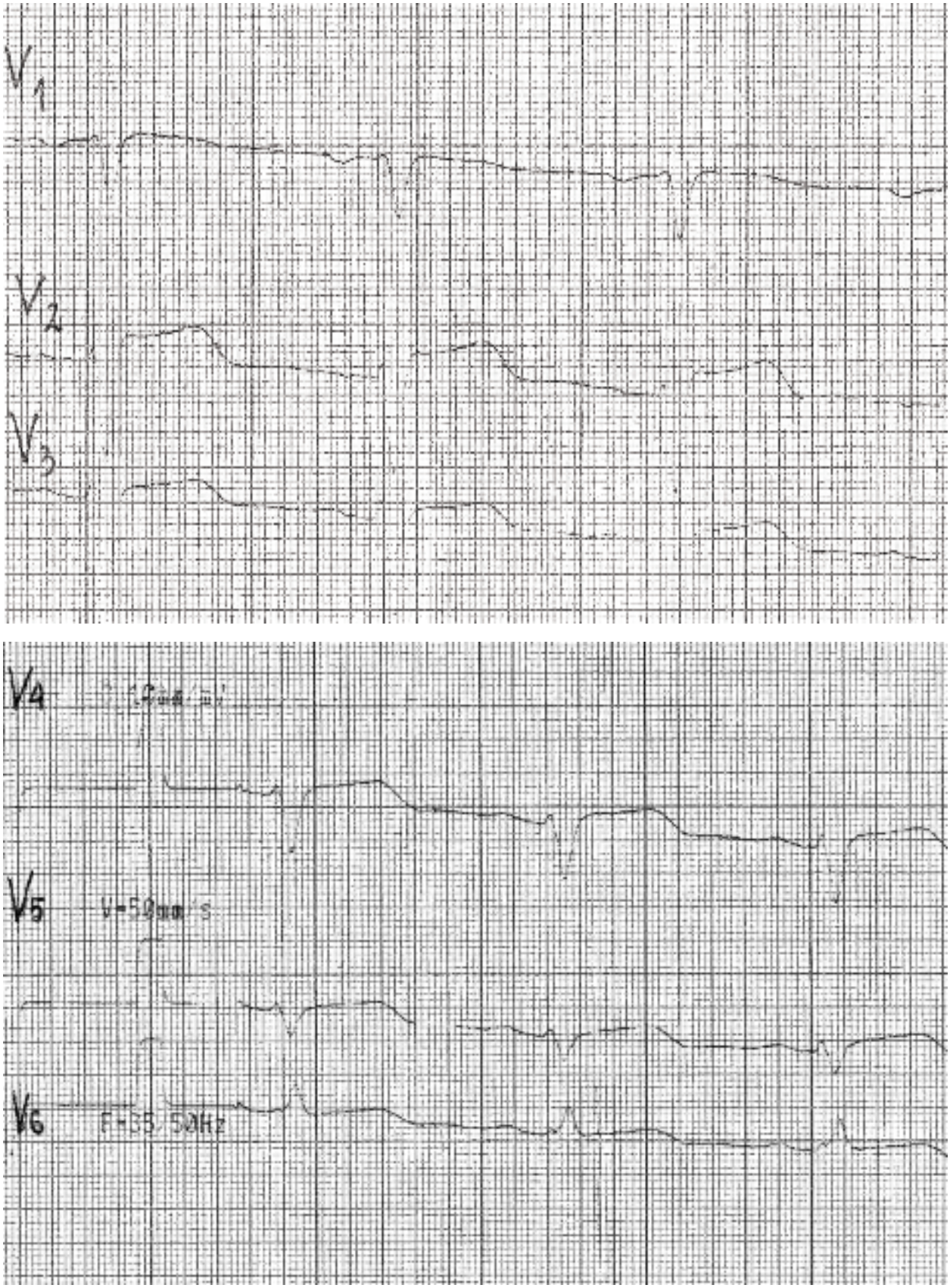

Fig. 1. cont.
Ryc. 1. *cd.*

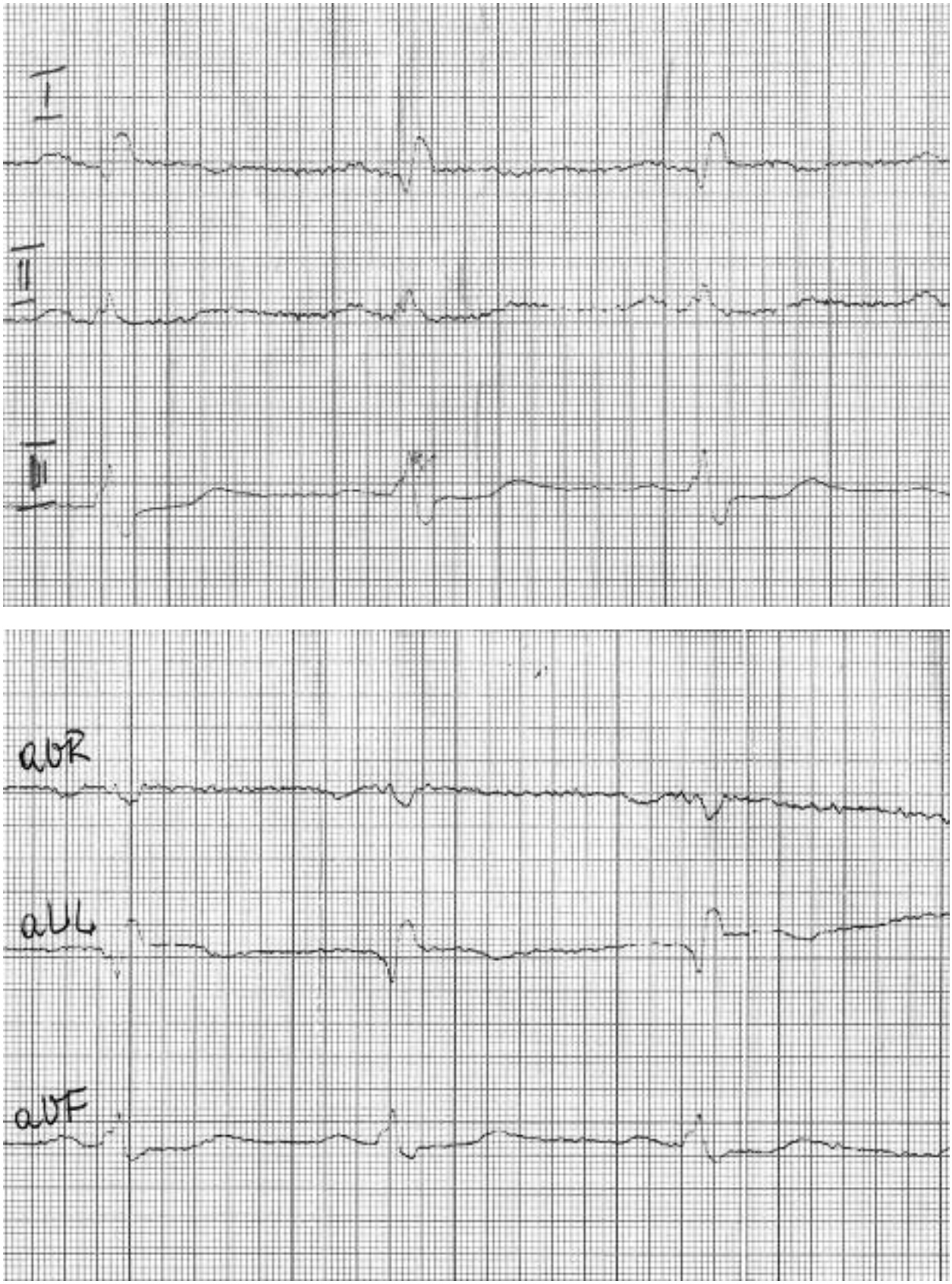

Fig. 2. "Old" ECG
Ryc. 2. *Wcześniejsze EKG*

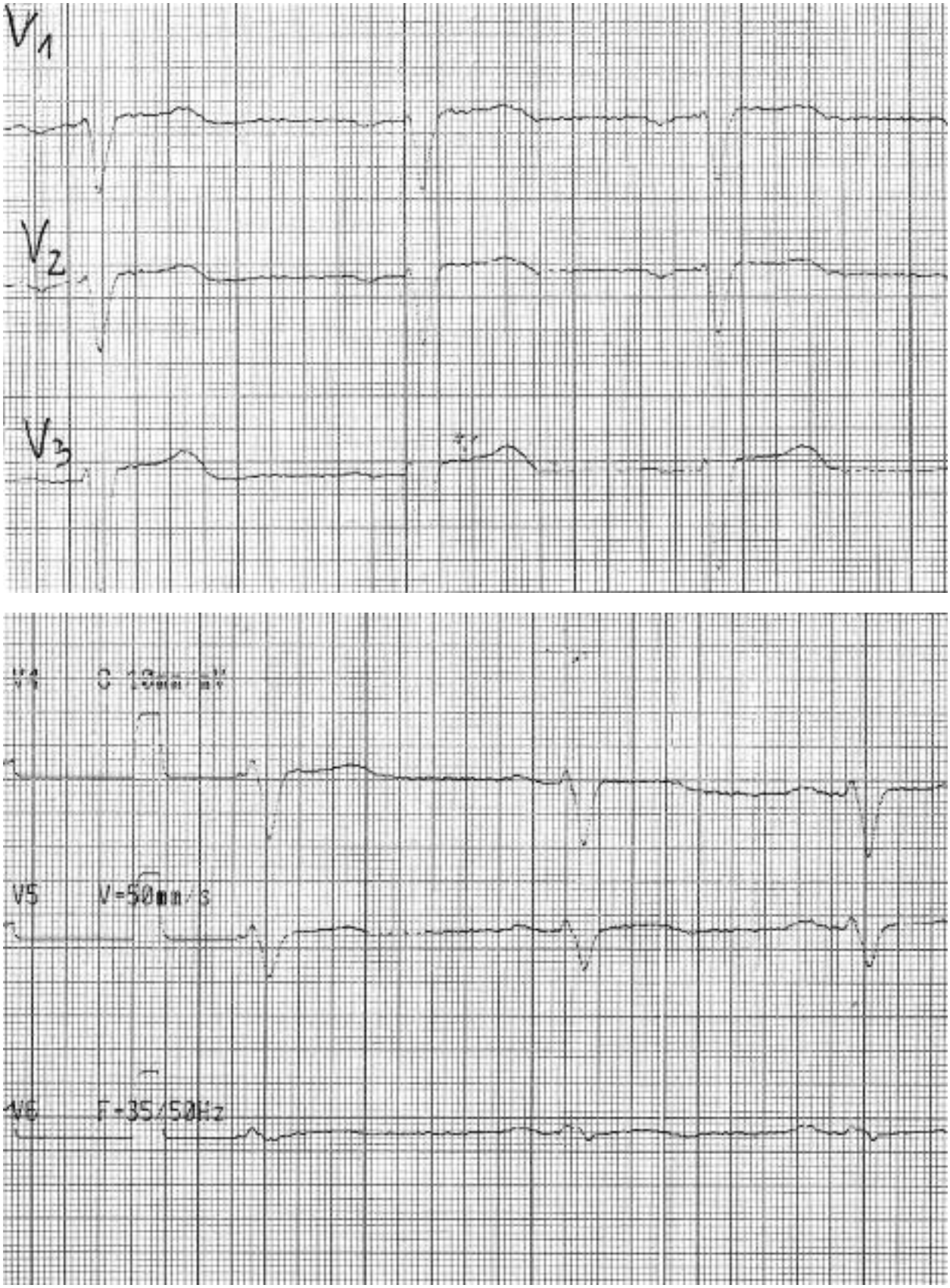

Fig. 2. cont.
Ryc. 2. *cd.*

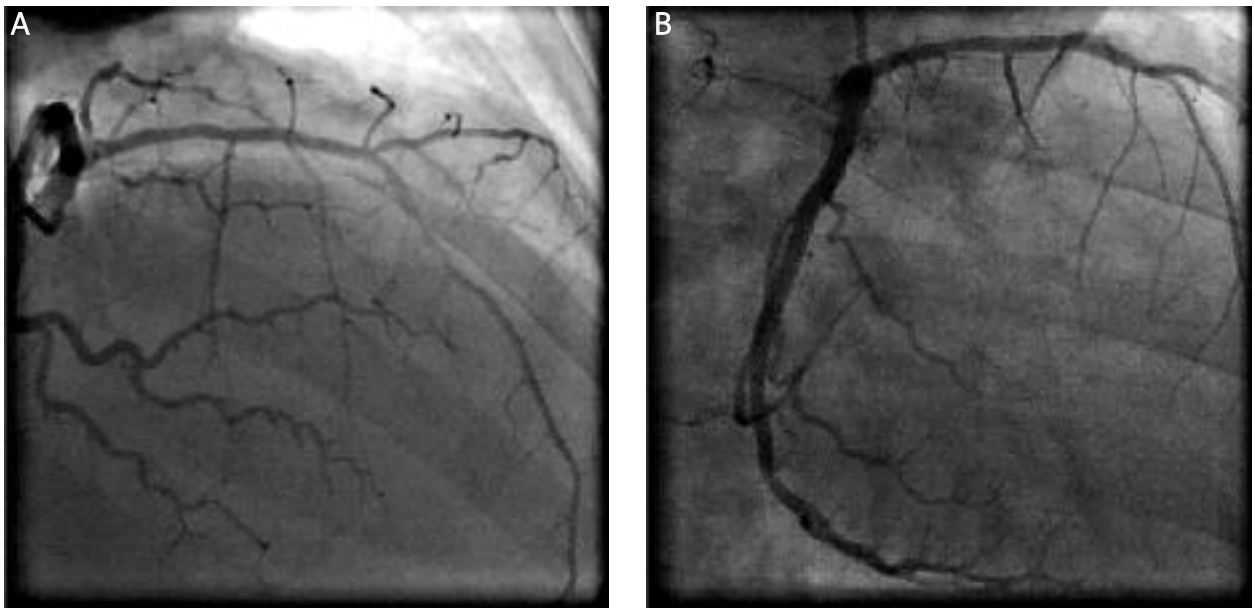

Fig. 3. Coronary angiography 2 weeks before current hospitalization
Ryc. 3. *Koronarografia wykonana 2 tygodnie przed obecną hospitalizacją*

the current one led to the decision to perform another coronary angiography. In comparison to the ECG made 2 weeks earlier there was an increase of the ST-segment elevation in leads I, aVL and V1-6 with a more pronounced ST-segment depression in leads II, III and aVF.

Coronary angiography, was performed via the right radial artery in the 7th h of chest pain and showed occlusion in the proximal part of the LAD, but located below the stent implanted 10 years earlier (Multilink 3.5 mm × 15 mm) [1-4]. After crossing through the occlusion site with a guidewire, a thrombectomy device was introduced and used for thrombus aspiration (Figure 4). This was followed by direct implantation of the DRIVER Sprint RX stent 3.5 mm × 15 mm overlapping the previous stent and expanded at 20 atm with achievement of TIMI 3 flow (Figure 5). Reo-Pro was administered during the procedure and as an intravenous infusion that followed. Further clinical course was uneventful. Subsequent ECGs demonstrated typical early evolution of the anterior myocardial infarction. There were no new left ventricular contraction abnormalities on echocardiography in comparison to previous ambulatory studies, which showed extensive post-infarction injury to the antero-lateral wall and the apex in the form of hypokinesis. The patient left the hospital on the fourth day of treatment.

From the time of the first myocardial infarction in 1999, the patient received standard treatment: acetylsalicylic acid (ASA) 75 mg/day, trandolapril 2 mg/day to control blood pressure and to unload the injured left ventricle (ejection fraction [EF] 30%), metoprolol 12.5 mg 3×. This treatment provided good control of blood pressure. The patient also received 20 mg of atorvastatin, but this treatment was insufficient because it did not provide the target level of LDL cholesterol below 100 mg/dl. The target level of LDL cholesterol introduced 10 years earlier was higher than currently, but in recent years physicians have not modified the previously established dose of statin. After successful angioplasty the dose of the drug was raised to 40 mg/day with the recommendation to perform a control lipid profile in 3 months and spironolactone was added because of marked impairment of the left ventricle. After PCI a standard dual antiplatelet therapy with the recommendation to continue it for a year was also introduced.

Discussion

A surprising and interesting fact in the described situation was the occurrence of acute coronary syndrome in a patient who did not have significant changes in coronary arteries on angiography performed 2 weeks earlier [5, 6].

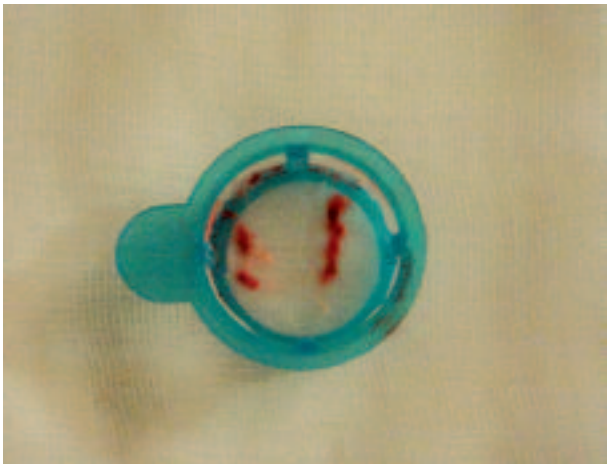

Fig. 4. Thrombus aspired from the LAD
Ryc. 4. Skrzeplina odessana z GPZ

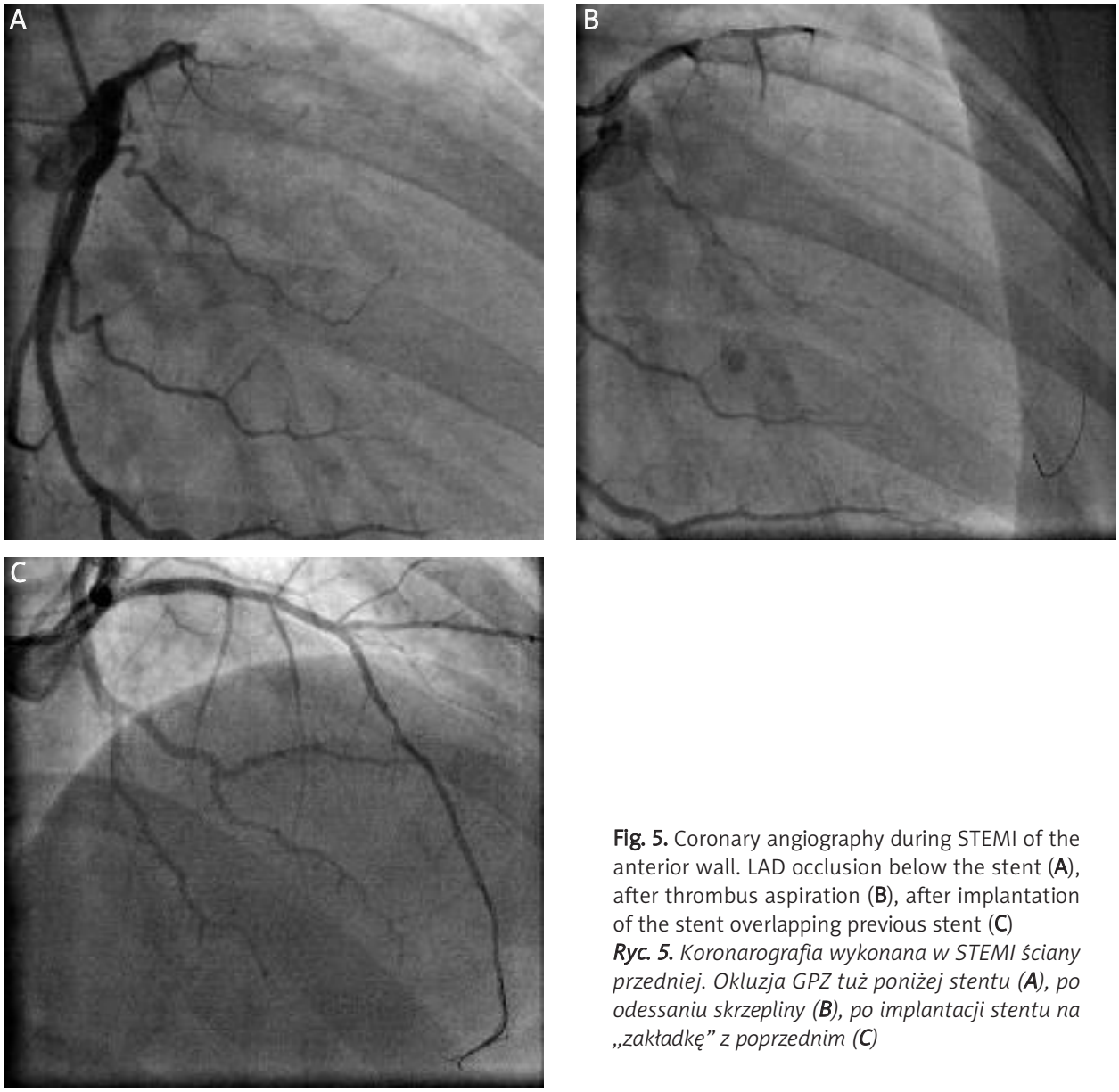

Fig. 5. Coronary angiography during STEMI of the anterior wall. LAD occlusion below the stent (**A**), after thrombus aspiration (**B**), after implantation of the stent overlapping previous stent (**C**)
Ryc. 5. *Koronarografia wykonana w STEMI ściany przedniej. Okluzja GPZ tuż poniżej stentu (**A**), po odessaniu skrzepliny (**B**), po implantacji stentu na „zakładkę" z poprzednim (**C**)*

A post factum detailed analysis of the previous coronary angiography revealed the presence of an insignificant perimural atherosclerotic plaque located below the stent implanted 10 years earlier. Coronary angiography performed in STEMI suggested that this atherosclerotic plaque had ruptured, initiating thrombus formation occluding the LAD [4, 7], which was aspired during the procedure using the Export system. The presented case is further evidence of the possible occurrence of acute coronary events in relation to the presence of an unstable atherosclerotic plaque, which may be practically invisible in coronary angiography and only slightly narrow the arterial lumen without compromise to the blood flow. The described sequence of events highlights the need for in-depth analysis of patients with earlier confirmation of ischemic heart disease, even without the presence of significant lesions in coronary arteries. Such patients represent a high-risk group for re-infarction, even in the absence of significant atherosclerotic lesions in coronary angiography. Certainly, these patients need better control of the lipid profile and routine assessment of indicators of active inflammation. The increase in the level of indicators such as high-sensitivity C-reactive protein (hs-CRP) should lead to intensification of anti-inflammatory therapy with a statin, which may improve the control of inflammatory processes within the vascular wall. Earlier introduction of a second antiplatelet drug may also be considered in such situations. The course of disease in the present patient confirms the need for a continuous search for ways to identify "hot" vulnerable plaques present in so-called angiographically normal coronary arteries, whose rupture may lead to vessel occlusion. A series of questions arises. First of all, how

to identify these patients? Assessment of hs-CRP relates mainly to symptomatic patients and determines the risk of the presence of "hot" atherosclerotic plaques located not only in the coronary arteries. It is also known that even the identification of a specific atherosclerotic plaque (done usually after its rupture and occurrence of acute coronary syndrome) not only does not exclude, but also suggests the existence of other unstable plaques. Currently available methods used to identify vulnerable plaques are complex and not always fully reliable. The question of how to deal with them if they do not cause significant stenosis of the artery remains open. This raises another question: Who should undergo hs-CRP level assessment? Of course, an elevated level of this protein does not identify unstable plaques, but only determines a higher risk of acute coronary syndrome (ACS) or other acute vascular events caused by the presence of inflammatory changes in the arterial wall. Should such patients as the one described above – practically asymptomatic, with only electrocardiographically positive exercise test –undergo hs-CRP assessment in order to clarify the risk of ACS (another one as in this patient)? We can only answer with certainty that this patient should be effectively treated with statin and aspirin regardless of the level of hs-CRP. Perhaps in the case of an elevated hs-CRP it would be reasonable to add a second antiplatelet drug. However, currently there is no evidence that this approach effectively prevents the occurrence of acute vascular events.

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