Acta Clin Croat (Suppl. 1) 2024; 63:90-95
doi: 10.20471/acc.2024.63.s1.17

Case Report

# TWO ACUTE STEMIS AND TWO ACUTE ISCHEMIC STROKES IN THE SAME PATIENT WITHIN FIVE DAYS – WHAT WENT WRONG?

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ABSTRACT – It is not uncommon for patients to suffer from both acute myocardial infarction and acute stroke during the hospitalization. According to some studies, about 12% of the elderly population initially hospitalized for acute ischemic stroke also develop type-1 acute myocardial infarction during the same hospitalization. On the other hand, about 0.9% of patients hospitalized for acute coronary syndrome develop acute stroke during the same hospitalization1. The therapeutic approach to such "overlapping" patients is challenging, especially if we also take into account a high risk of bleeding and/or active bleeding. Therefore, interdisciplinary collaboration between cardiology, neurology and interventional neuroradiology is of key importance. Timely intervention and adequate concomitant drug therapy (primarily antiplatelet and anticoagulant therapy) determine treatment outcomes and long-term results. In our recent clinical work, we treated a patient with a series of acute cardiac and cerebral incidents presenting multiple therapeutic dilemmas, who ultimately had an unfavourable neurological outcome.

Key words: *STEMI, anaemia, antiplatelet drugs, ischemic stroke, thrombectomy*

## Introduction

Although protocols for the treatment of acute infarction are well defined in the guidelines, as are combinations and doses of antiplatelets/anticoagulants and protocols for bleeding risk assessment, in our everyday clinical practice we often face therapeutic dilemmas. This confirms the importance of an individual approach to each patient and case. In the era of interventional cardiology, ST elevation myocardial infarction (STEMI) is primarily treated with intervention. Intervention is followed by dual antiplatelet therapy (acetylsalicylic acid (ASA) + P2Y12 receptor antagonists), which increases the risk of bleeding. Moreover, it significantly increases the risk of bleeding if the patient is already at high risk even when they are not on antiplatelet therapy. Ischemic cerebrovascular incidents are known and statistically clearly defined as

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complications of coronary angiography and percutaneous coronary intervention (PCI) procedures. They occur in 0.27–0.5% of these procedures[2]. Since these complications can be fatal or result in the development of significant disability, much research has been devoted to finding the best therapeutic approach [4]. Based on the available results and its well-developed network over time, as a therapeutic approach[5,6], mechanical thrombectomy has an advantage over systemic intravenous thrombolysis and direct intra-arterial thrombolysis because these iatrogenic cases are frequently embolic and are the result of scraped aortic debris or scraped atherosclerotic plaque obstructing cerebral circulation, and such material does not respond well to lithic therapy[2,3,4]. Another issue is parenteral anticoagulant therapy and eptifibatid administered during diagnostics and PCI procedures, which contraindicate, at least relatively, systemic intravenous thrombolysis. After successful mechanical thrombectomy, dual antiplatelet therapy is indicated. Its duration depends on the cerebrovascular intervention itself, i.e., balloon angioplasty alone or in combination with stent implantation[5,6]. This highlights the importance of interdisciplinary collaboration between cardiology, neurology and interventional neuroradiology, which must result in timely procedure for optimal treatment outcome of a patient[7]. The paper presents the case of a 68-year-old patient (as well as our therapeutic dilemmas) who suffered two acute STEMIs and two acute ischemic strokes, ultimately ending with an unfavourable neurological outcome.

## Case report

A 68-year-old patient with no previous medical history was hospitalized for chest pain. Upon admission, the patient was in extremely poor health: cachectic, weight 60 kg, height 170 cm, with severe microcytic iron-deficiency anaemia (Hb 68, MCV 61, Fe 3) and reactive thrombocytosis (trc 518). The first electrocardiogram (ECG) showed lateral STEMI (slight ST elevation in D1, aVL, slight ST depression in inferior leads, hyperacute T-waves in the anterior wall) (Figure 1). Approximately three hours after admission, the pain subsided completely, and ECG returned to normal. Due to severe anaemia, complete regression of pain and ECG changes, emergency coronary angiography was not performed. Anaemia was corrected with erythrocyte concentrate with control level of Hb up to 95, patient received pantoprazol 40 mg IV, ASA 100 mg orally and no low molecular weight heparin. On the third day after admission, the patient developed respiratory arrest with bradycardia. Cardiopulmonary resuscitation (only chest compression) was immediately initiated. After a short time, the patient restored a sinus rhythm up to 88/min, systemic blood pressure up to 110/60 mmHg and spontaneous breathing. Then we noticed short runs of nonsustained ventricular tachycardia, one paroxysm of atrial fibrillation on telemetry, apicolateral hypokinesia on echocardiography, total left ventricular ejection fraction preserved and no significant valvular pathology, so we decided to perform coronary angiography. It was performed via right transfemoral access without periprocedural heparin. We found the first diagonal branch (D1) occlusion, left anterior descending (LAD) and trifurcation branch (RI) irregularities, normal circumflex (ACx) and normal right coronary artery (RCA) (Figure 2). Based on the findings (smaller blood vessel and small supply area), stabilized clinical condition of the patient and due to anaemia of unknown aetiology and the fact that the continuation of dual antiplatelet therapy if we perform PCI would be required, we opted for a conservative treatment. Only ASA was continued, without heparin. Forty-eight hours after the first coronary angiography, the patient experienced severe chest pain. ECG showed lateral STEMI with diffuse ST depression in most other leads (Figure 3). Emergency coronary angiography was re-initiated through the right transfemoral access. In addition to the previously known D1 occlusion, thrombotic occlusion of the RI and thrombotic lesion in the left main coronary artery (LMCA) ostium were found (Figure 4). PCI of the RI and afterwards LMCA were performed with periprocedural heparin (7000 units intra - arterial). One drug eluting stent (DES) was implanted in the RI (Supraflex Star 2.5 x 24 mm) and one DES in the LMCA ostium (Supraflex Star 4.5 x 8 mm) (Figure 5). Towards the end of the procedure, the patient became restless, showed signs of motor dysphasia and developed left hemiplegia. Emergency MSCT of the head excluded haemorrhage. Computed tomography angiography revealed occlusion of the right internal carotid artery (ICA) above the bifurcation (Figure 6). ECG still showed a slight elevation in D1, aVL, while diffuse ST depression was mostly resolved (Figure 7). An attempt of mechanical thrombectomy was indicated in consultation with a neurologist and an interventional neuroradiologist from a partner institution.

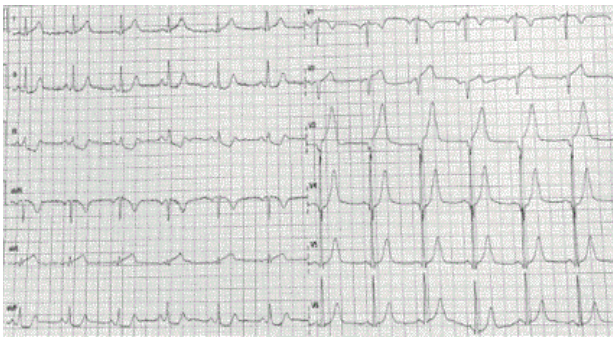

*Figure 1: ECG at admission with pain present – first STEMI*

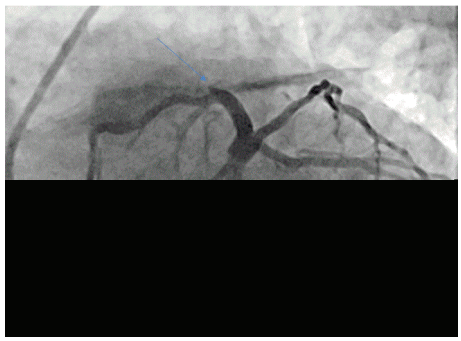

*Figure 2: First coronary angiography – D1 occlusion*

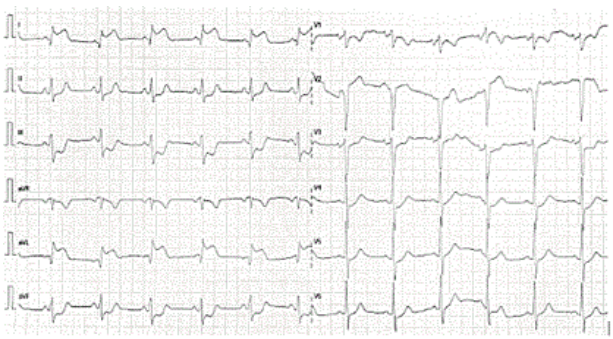

*Figure 3: Recurrence of pain – second STEMI*

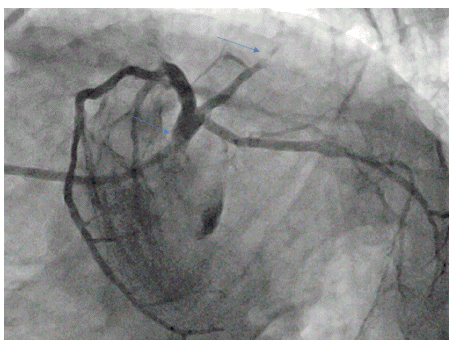

*Figure 4: Second coronary angiography – thrombotic occlusion of the RI, thrombotic lesion at the LMCA ostium*

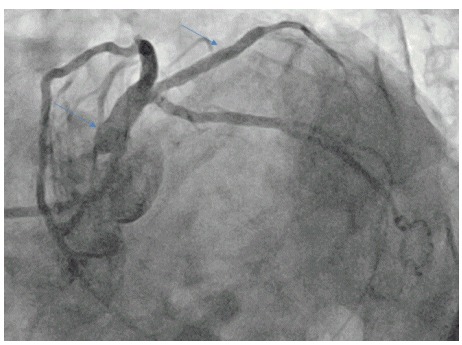

*Figure 5: After the PCI of the RI and LMCA*

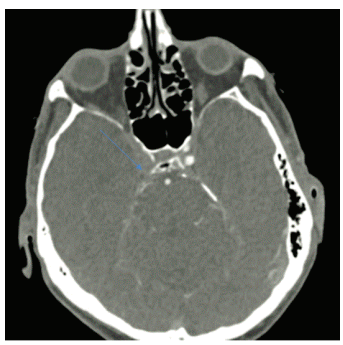

*Figure 6: MSCT angiography – right ICA occlusion*

The patient was transported to the partner institution. Within three hours from the onset of neurological deficits, cerebral angiography was performed and an occlusion of the terminal part of the right ICA was found (Figure 8). After multiple manipulations, a solid embolus (Figure 9) was aspirated using a wide aspiration catheter. A follow-up angiogram showed TICI 2c flow and stenosis of the M1 segment of the right middle cerebral artery (MCA), which persisted even after having performed balloon angioplasty (Figure 10). Atlas (4 x 24 mm) stent was implanted into the lesion. A follow-up angiogram with a 5-minute time delay showed TICI 2c flow distal to the implanted stent (Figure 11). CT of the brain parenchyma did not show marked ischemic lesions (Figure 12). Immediately after the stenting of the MCA, the patient periprocedurally received IV 400 mg ASA and IA bolus 20 mL of eptifibatide. The treatment was continued in the neurological intensive care unit. In addition to ASA 100 mg daily orally, another antiplatelet drug - ticagrelor

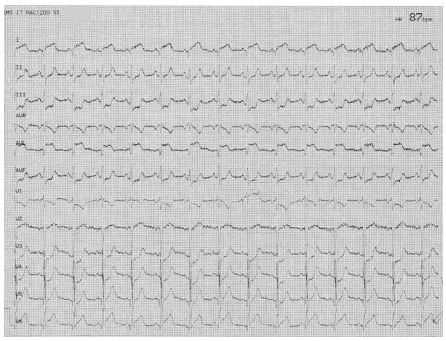

*Figure 7: Partial recovery of ECG changes after PCI*

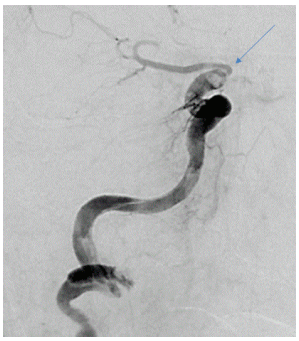

*Figure 8: DSA – right MCA occlusion*

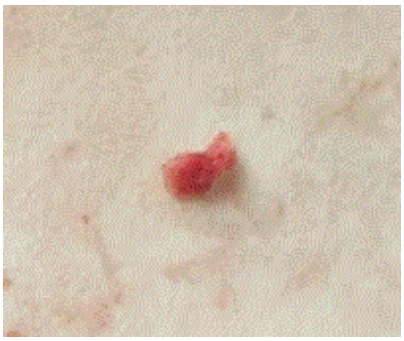

*Figure 9: Aspirated solid thrombus*

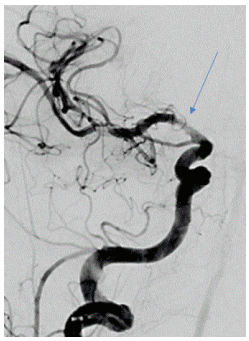

*Figure 10: MCA after balloon angioplasty*

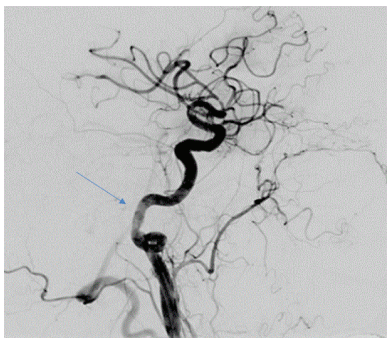

*Figure 11: MCA after stent implantation*

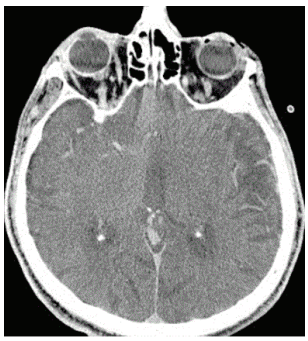

*Figure 12: After thrombectomy – no signs of marked ischemic lesion*

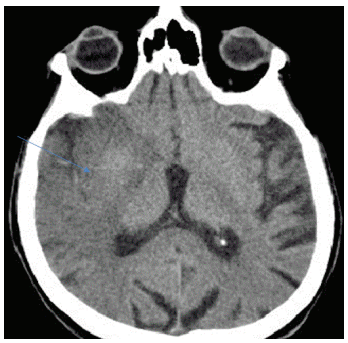

*Figure 13: Stent thrombosis and demarcated ischemic area*

180 mg daily orally, was added after the admission of the patient in the neurological intensive care unit. Eptifibatide infusion was than discontinued. The initial neurological deficit started to recover. The patient was cardiologically completely stable. Sixteen hours after the stenting of the MCA, the patient became somnolent, unable to verbally communicate, while the deterioration of the motor function of his left extremities led to hemiplegia again. Follow-up CT of the brain showed acute ischemia in the supply area of the right MCA with indirect signs of thrombotic stent occlusion in the MCA (Figure 13).

## Discussion

We presented a case of a non-smoking patient with severe microcytic, iron-deficiency anaemia of unknown aetiology, initially presented as lateral wall STEMI without other present and proven cardiovascular risk factors. Due to anaemia, fast regression of chest pain, and ECG normalization, coronary angiography was not preformed initially, and the patient was not aggressively treated with dual antiplatelet therapy and parenteral anticoagulant therapy. Anaemia was partially corrected. First coronary angiography was performed on the third day after admission, when the patient developed hemodynamic instability. Based on small vessel occlusion, stable clinical condition of the patient after coronary angiography, and anaemia of unknown aetiology, we decided for conservative treatment only with ASA 100 mg daily orally. The patient then developed a new STEMI. Repeated coronary angiography showed progression of the thrombotic events in epicardial coronary arteries – thrombotic occlusion of the RI and significant thrombotic lesion of the LMCA ostium. At this point, we would like to emphasize the role of the iron-deficiency anaemia of unknown aetiology (given the age and the condition of the patient probably as part of a paraneoplastic syndrome)[8], which triggered the procoagulable state plus reactive thrombocytosis as the second mechanism of platelet dysfunction and thrombotic events[9,10] occurring to the patient. A high bleeding risk was determined. Therefore, we took a more sparing approach to antiplatelet and anticoagulant medication. The second STEMI was the result of the same, previously mentioned procoagulable state plus a non-aggressive treatment with antiplatelet and anticoagulant therapy – only ASA without heparin. During the second coronary angiography and PCI procedure, the patient developed an ischemic stroke, probably as a mechanical complication of the procedure[4]. We presume that it was an embolic event with scraped atherosclerotic plaque off the aortic arch which then migrated as a solid atherothrombotic embolus and occluded the terminal part of the right ICA. In consultation with neurology and interventional neuroradiology, emergency cerebral mechanical thrombectomy was organized[3,5,6]. Due to the newly developed neurological deficit, the patient did not receive a loading dose of another antiplatelet drug during and at the end of the PCI procedure, or before cerebral mechanical thrombectomy with stent implantation, he received ticagrelor loading dose after the admission in the neurological intensive care unit, which, in addition to procoagulable state caused by anaemia and thrombocytosis, probably also led to early stent thrombosis in the MCA and the development of a new ischemic stroke. From the beginning, we faced dilemmas concerning an adequate approach and risk weighing when treating this patient. We often opt for a sparing approach when it comes to antiplatelet and anticoagulant drugs because we are afraid of bleeding complications, even more so in this case, given the unknown cause of anaemia. If we compare the first and the second coronary angiography and the first and the second ECG, the size of the lesion was definitely larger during the second time, which supports the idea of progression of the thrombotic event with an inadequate/sparing treatment approach (only one antithrombotic drug without heparin in patient with procoagulable state). Ischemic cerebrovascular incidents are known and statistically defined mechanical complications of coronary angiography and PCI procedures. According to the literature, they occur in up to 0.5% of these procedures[2]. When it comes to our patient, the complication was initially successfully handled thanks to an adequate and timely choice of therapeutic procedure (mechanical thrombectomy versus systemic thrombolysis) and collaboration between various intervention disciplines, which is extremely important in such "overlapping" patients in whom timely intervention saves lives but also prevents disability[7]. Unfortunately, the development of disability was not prevented in our patient in the end because of the development of early thrombosis in the ICA stent as a result of the interaction between the untimely dual antiplatelet therapy and still undefined and untreated cause of severe iron-deficiency anaemia[8,10]. The question is whether the outcome would be much different if we had treated the first STEMI more aggressively.

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Sažetak

## DVA AKUTNA STEMI I DVA AKUTNA ISHEMIJSKA MOŽDANA INFARKTA U PET DANA KOD ISTOG BOLESNIKA - ŠTO JE POŠLO KRIVO?

*V. Slivnjak, I. Šesto, A. Jelić, V. Kalousek, D. Cerovec, A. Škopljanac Mačina* i *K. Štambuk*

Akutni srčani infarkt i akutni moždani infarkt nerijetko susrećemo kod istog bolesnika tijekom jedne hospitalizacije. Prema nekim istraživanjima oko 12 % starije populacije koja je inicijalno hospitalizirana radi akutnog ishemijskog moždanog infarkta tijekom iste hospitalizacije razvije i akutni srčani infarkt tip 1, a oko 0.9 % hospitaliziranih radi akutnog koronarnog sindroma u istoj hospitalizaciji razvije akutni moždani infarkt1. Terapijski pristup takvim „preklapajućim" bolesnicima je izazovan, naročito ako tome svemu dodamo još visoki rizik od krvarenja i/ili aktivno krvarenje. Suradnja struka – kardiologije, neurologije i intervencijske neuroradiologije od velike je važnosti. Intervencija u vremenskom prozoru te adekvatna popratna medikamentozna (prvenstveno antiagregacijska i antikoagulantna) terapija definiraju ishod liječenja i dugoročni rezultat. Nedavno smo u kliničkom radu imali bolesnika s nizom akutnih srčanih i moždanih incidenata, nizom terapijskih dilema i naposljetku nepovoljnim neurološkim ishodom.

Ključne riječi: *STEMI, anemija, antiagregacijska terapija, ishemijski moždani infarkt, mehanička trombektomija*