DOI 10.4149/BLL_2024_43

Bratisl Med J 2024; 125 (5):
289–298

CLINICAL STUDY

Rare instances of concomitant acute myocardial infarction and stroke

Damian MACIEJEWSKI[1], Karol NOWAK[2,3], Magdalena WAWAK[1], Aleksandra KARCINSKA[4], Lukasz TEKIELI[1], Mariusz TRYSTULA[5], Robert MUSIAL[6], Jakub PODOLEC[1], Piotr PIENIAZEK[1,4], Jaroslaw ZALEWSKI[2]

Department of Coronary Disease and Heart Failure, Institute of Cardiology, Jagiellonian University Medical College, Saint John Paul II Hospital, Krakow, Poland. **k.nowak.uj@gmail.com**

ABSTRACT

Cardio-cerebral infarction (CCI) is a term coined to describe concomitant myocardial infarction and acute ischemic stroke. Acute myocardial infarction and stroke, as separate events, constitute some of the most important causes for disability and mortality in aging societies. Stroke can either occur simultaneously with myocardial infarction or become a serious complication of myocardial infarction and/or its treatment. The frequency of CCI has been reported at a 0.009% incidence rate in stroke patients and is associated with an extremely high mortality. Because of the rare occurrence of CCI, there are currently no guidelines for assessing its diagnosis and optimal treatment. Therefore, currently, the management of CCI cases needs to be individualized. Hopefully, in the future, the results of large clinical trials or prospective registries are expected to enhance our understanding of managing concomitant acute MI and stroke. In this review we have focused on the current literacy in the diagnosis and treatment of CCIs. The paper illustrates potential distinct scenarios of CCI through the analysis of three patient cases *(Fig. 5, Ref. 65)*. Text in PDF *www.elis.sk*

KEY WORDS: myocardial infarction, stroke, cardio-cerebral infarction, carotid artery stenting, cardiac surgery.

Introduction

Myocardial infarction (MI) and stroke remain important clinical and public health problems in aging societies. They are the major causes of mortality worldwide and both have a significant global impact on patients' survival (1).

Coronary heart disease and some subtypes of ischemic stroke share similar risk factors and pathophysiology, including inflammation and the progression of atherosclerotic plaques (2, 3). Additionally, MI itself can be a stroke risk factor, either during revascularization or from atrial fibrillation in association with acute MI or from blood stasis in dysfunctional left ventricle (4–7). While stroke and MI share some common vascular risk factors, acute vascular damage to specific brain areas has also been implicated in cardiac dysfunction and myocardial damage (8).

MI complicated by a stroke, two possible scenarios

Stroke can either cooccur with MI simultaneously or become a serious complication after MI or its treatment. Cardio-cerebral infarction (CCI) is a term coined to describe concomitant MI and acute ischemic stroke. CCI has been reported at an incidence rate of 0.009% in patients who suffered from a stroke (9). Given the rarity, there are currently no guidelines concerning the diagnosis and treatment of CCI. Therefore, CCI requires careful evaluation as it is a serious clinical situation with high mortality (10).

Spontaneous co-occurrence of MI and stroke 2,4,

The overlapping pathogenesis of stroke and MI contributes to the spontaneous co-occurrence of those two conditions while only a few possible mechanisms have been described so far. Firstly, in the case of akinesis or dyskinesis of the myocardium, an intracardiac thrombus can form and embolize the coronary and cerebral arteries. Likewise, a thrombus resulting from peri-myocardial atrial fibrillation can cause CCI. Deep vein thrombosis is also a risk factor for CCI, as emboli can move through patent *foramen ovale* and occlude coronary and cerebral arteries concurrently. Other mechanisms of simultaneous MI and stroke unrelated to thrombi include ascending aorta dissection involving ostia that extend to

[1]Department of Interventional Cardiology, Institute of Cardiology, Jagiellonian University Medical College, Saint John Paul II Hospital, Krakow, Poland, [2]Department of Coronary Artery Disease and Heart Failure, Institute of Cardiology, Jagiellonian University Medical College, Saint John Paul II Hospital, Krakow, Poland, [3]Department of Thromboembolic Disorders, Intitute of Cardiology, Jagiellonian University Medical College, Krakow, Poland, [4]Students' Scientific Group at the Department of Coronary Artery Disease and Heart Failure, Institute of Cardiology, Jagiellonian University Medical College, Krakow, Poland, [5]Department of Vascular Surgery and Endovascular Interventions, Saint John Paul II Hospital, Krakow, Poland, and [6]Department of Anesthesiology and Intensive Care, Saint John Paul II Hospital, Krakow, Poland

Address for correspondence: Karol NOWAK, Department of Coronary Disease and Heart Failure, Institute of Cardiology, Jagiellonian University Medical College, Saint John Paul II Hospital, 80 Pradnicka Street, 31-202 Krakow, Poland.
Phone: 12 614 22 18, Fax: 12 614 22 19

the coronary and carotid arteries. Moreover, primary myocardial infarction that results in hypotension and cardiogenic shock can subsequently cause a brain ischemic injury in the mechanism of hypoperfusion (9). The safe and effective treatment of these patients is complicated. The proper order of interventions is difficult to determine because there are no clear indications as to whether MI or stroke should be treated first. However, every decision can impact the patient's health, including disability or heart failure in future (11–13).

MI therapy complicated by a stroke

Among various complications of MI, stroke is one of the most life-threatening conditions, with significantly higher in-hospital and long-term mortality rates as compared to stroke occurring alone. Both ischemic and hemorrhagic strokes can complicate acute MI. The short-term and 1-year mortality rates of MI-related ischemic stroke are 30.1% and 36.5%, respectively (14). The hemorrhagic stroke is less frequently observed within 30 days after MI as compared to ischemic stroke (2.1 vs 0.22%) (15, 16), but its mortality rate is higher (65.4% at 30 days) (17). Risk factors for ischemic stroke after MI include a history of prior stroke, older age, female sex, diabetes mellitus and atrial fibrillation (16, 18). Moreover, any new onset of atrial fibrillation complicating the course of MI is associated with a significantly increased risk of ischemic stroke (19). The main and most important risk factor of stroke of any type after discharge from the hospital is the history of prior stroke (20).

Among patients from the GRACE registry admitted to hospital with an acute coronary syndrome, in-hospital stroke was a relatively uncommon event, occurring in 0.88% of cases. In the available literature, stroke complicated the course of ST segment elevation MI (STEMI) more frequently compared to non-ST segment elevation MI (NSTEMI) (1.3 vs 0.9% respectively) (20). Although most strokes were non-hemorrhagic, the proportion of hemorrhagic strokes was higher in the STEMI population compared to the NSTEMI population. Almost one-third of strokes were fatal, which emphasizes the severity of this condition (14, 20). In the population of the APEX-AMI trial, stroke events occurred in 1.3%, with most of them occurring later than 48 hours after percutaneous coronary interventions (PCI), indicating that they might not be directly related to the invasive procedure (21).

Merkler et al (22) have shown that the risk of ischemic stroke is elevated for 12 weeks after MI, maintaining its peak within four weeks after the incident. For almost 25 years, the frequency of stroke occurrence after acute coronary syndromes has been reduced due to the routine use of acetylsalicylic acid, P2Y12 inhibitors, statins, and advances in PCI (16, 18). Albeit, the effects of fibrinolytic treatment, the second potential option in acute MI therapy, are inconclusive. Fibrinolytic therapy in acute MI is associated with a decreased risk of ischemic stroke (18). Simultaneously, fibrinolysis in MI increases the risk of intracranial bleedings leading to hemorrhagic strokes (23, 24) and the risk of cardiac walls rupture (25). The benefits of early fibrinolytic reperfusion in patients who cannot undergo PCI outweigh the slight risk of bleeding, but careful monitoring of the patient is essential.

Routine thrombectomy is currently not recommended in the invasive management of MI patients (26) since in the large trials, it did not improve clinical outcomes (27, 28) despite promising results in the TAPAS trial (29). The TOTAL trial showed that thrombectomy did not improve the primary outcomes including death from cardiovascular causes, recurrent myocardial infarction, cardiogenic shock, or new or progressing up to New York Heart Association (NYHA) class IV heart failure within 180 days (hazard ratio [HR] 0.99, 95% confidence interval [CI] 0.85–1.15, p=0.86). Moreover, in this trial, thrombectomy with PCI was associated with an increased risk of ischemic and hemorrhagic strokes (HR 2.08, 95% CI 1.29–3.35, p=0.002) (30) which resulted in a higher mortality rate.

The role of anticoagulation and antiplatelet drugs is a key point in stroke prevention. Although new P2Y12 inhibitors such as ticagrelor and prasugrel are more effective in reducing cardiovascular mortality than clopidogrel, especially in the acute phase of MI, their advantage has not been proven in the case of strokes (31, 32). Moreover, compared with unfractionated heparin, enoxaparin in the treatment of acute coronary syndromes did not significantly increase the risk of both hemorrhagic and non-hemorrhagic stroke (incidence rates of stroke among patients on enoxaparin and unfractionated heparin were 1% and 0.9%, respectively) (33, 34). In terms of post-MI pharmacological treatment, the PEGASUS-TIMI 54 trial showed that the addition of ticagrelor to the aspirin in the secondary prevention after MI significantly reduced the risk of stroke without excessive of hemorrhagic stroke but with a higher prevalence of major bleedings (35).

The left ventricular (LV) thrombus is a well-known complication in acute MI patients. Its most feared complication is cardioembolism causing an acute ischemic stroke (36). Before the advent of primary PCI for the treatment of STEMI, LV thrombus used to be a serious complication reported in up to 46% of patients following anterior wall STEMI (37). Since the introduction of primary PCI, the incidence of LV thrombus after STEMI has decreased, ranging from 2% to 4% in large studies. The steady decline in the incidence of LV thrombus can be related to the improvement in PCI practice and increasing accessibility of catheterization labs over the years (38, 39).

Stroke as a complication of percutaneous coronary intervention

Although a rare event, stroke after a percutaneous coronary intervention is a life-threatening complication. The prevalence of peri-PCI stroke is reported from 0.38 (40) to 0.96% of procedures in the latest publication (41), and is correlated with an unfavorable prognosis associated with a significant increase in long-term mortality rate (hazard ratio 1.71 [95% confidence interval 1.25–2.33]) (41). The independent predictors of stroke occurrence during percutaneous procedures are acute coronary syndrome during hospitalization, older age, heart failure, carotid artery disease, atrial fibrillation, chronic obstructive pulmonary disease, and neoplasm (41). Interestingly, the increase in the prevalence of a transradial approach compared to transfemoral intervention was not associated with an increase in the incidence of stroke (42), but in some studies it was linked to the reduction in cerebral incidents (odds

ratio; 0.33 [95% confidence interval, 0.16–0.71], p=0.004) (43). Despite that, the incidence of post-PCI strokes is increasing over time. This is partially caused by the higher complexity of patients undergoing PCI in the current era (44).

A series of three cases

The concomitant occurrence of acute MI and stroke is infrequently encountered among patients suffering from life-threating conditions. There are no evidence-based guidelines for the management of such patients, nor are there any published studies addressing the decision-making process in such cases. In the third case we would like to underscore the risk of cerebral events during percutaneous vascular procedures. Each of these cases was diagnosed or treated in our tertiary center.

First case

A 78-year-old male, ex-smoker, was admitted to the district hospital emergency department with focal neurologic deficits, which had started 48 hours earlier. Neurological examination revealed mixed transcortical aphasia, right-sided central facial paresis, and right hemiparesis (more expressed in the upper extremity). The patient's electrocardiogram showed biphasic T-waves in III, aVF, V2-V4 and inverted T-waves in I, aVL, V5-V6 leads. Cardiac troponin I level was elevated at 489.50 ng/l (the upper limit of normal <34.2 ng/l) with a downward trend on repeat every 3 hours. Both before admission to the hospital and during the hospitalization, the patient did not complain about chest pain.

Emergent bedside transthoracic echocardiography showed akinesis of the basal segments of the inferior wall, basal interventricular septum, and hypokinesis of the lateral wall with mildly reduced LV systolic function up to 45%. A computed tomography (CT) brain scan showed subacute ischemic stroke lesions of the frontal and parietal lobes without intracerebral hemorrhage. Based on clinical symptoms and laboratory results a diagnosis of NSTEMI with concomitant brain ischemia was made. As the time window for thrombolytic treatment had elapsed, the patient was disqualified from thrombolytic therapy.

After an interventional cardiologist consultation, owing to his stable condition without chest pains, the patient was qualified for conservative pharmacological treatment with clopidogrel and acetylsalicylic acid. Both duplex ultrasound and CT angiography revealed 70–75% stenosis of the left carotid artery however carotid revascularization was delayed due to recent MI. During hospitalization the patient's neurological condition had improved and after 13 days, the patient was discharged on dual-antiplatelet therapy and referred to a tertiary cardiovascular center.

Four weeks later, the patient was admitted to the tertiary cardiological unit for further diagnostic procedures and treatment optimalization. Coronary angiography and selective left carotid artery angiography revealed multi-vessel disease (Fig. 1AB) with

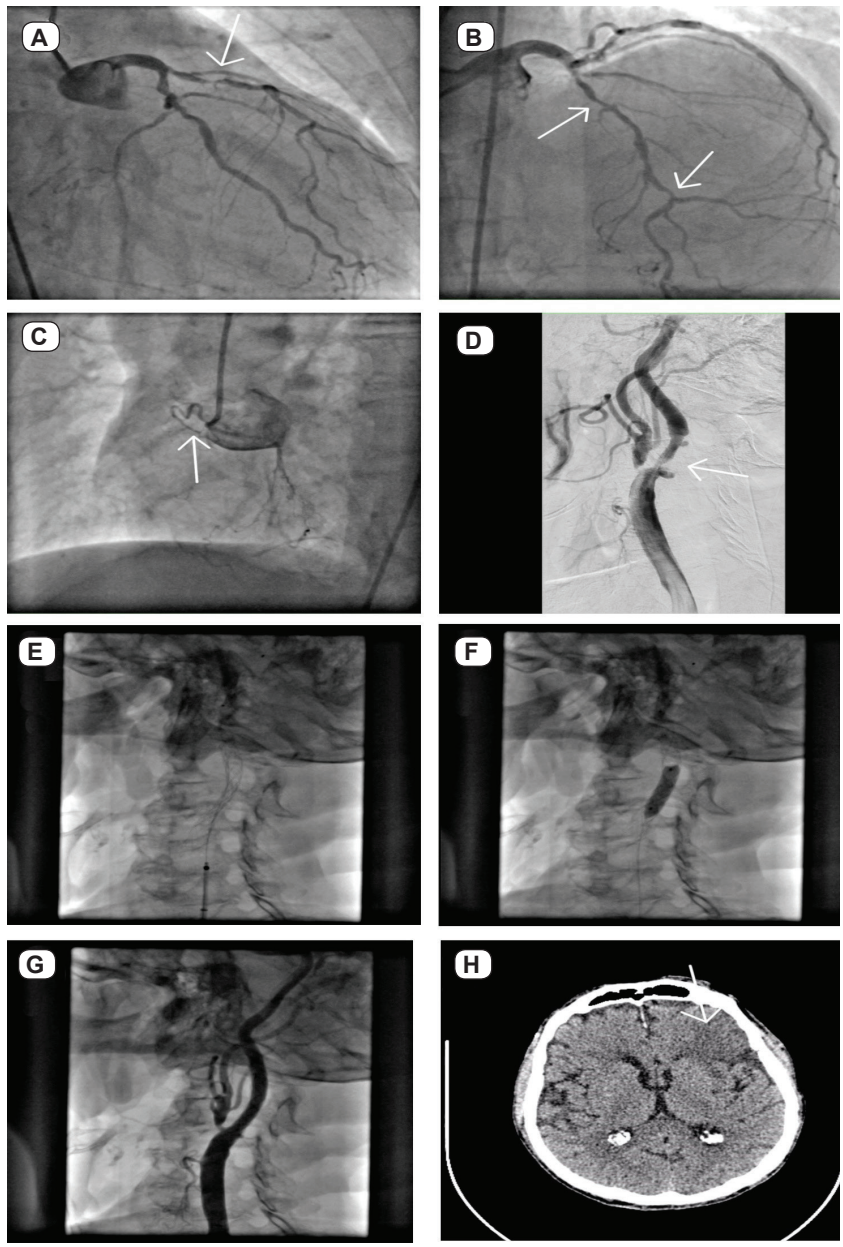

Fig. 1. The results of coronary angiography with multivessel disease (A–C), left internal carotid artery angiography with 80% stenosis (D) CAS procedure (E–G) and ischemic lesion in left hemisphere (H).

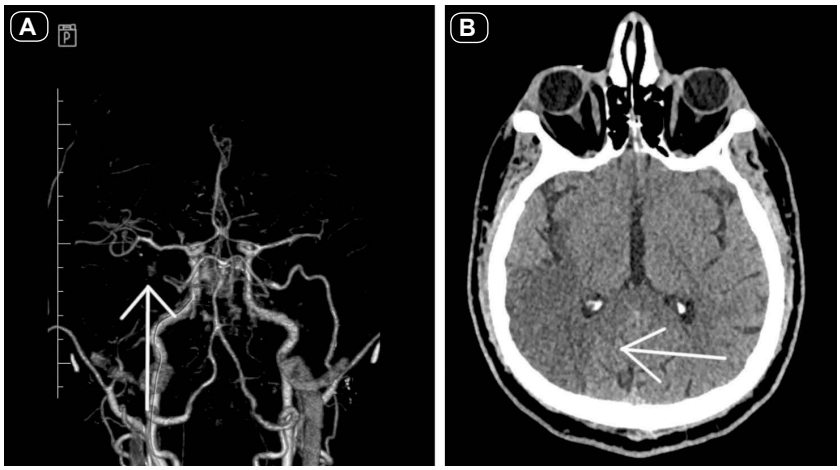

Fig. 2. Occlusion of the right middle cerebral artery in 3D reconstruction of angio-CT (A) and hypodense area between parietal, temporal and occipital lobes in the right hemisphere, in the posterior insula and posterolateral part of the frontal lobe.

total occlusion of the right coronary artery (Fig. 1C) and 80% stenosis of the left internal carotid artery (Fig. 1D). Next day, the patient was assessed by a multidisciplinary, joint Vascular and Heart Teams including a cardiac surgeon, invasive and noninvasive cardiologists, neurologist, vascular surgeon, and an anesthesiologist to plan the best treatment modality. The patient was qualified for hybrid one-day carotid artery stenting combined with coronary artery bypass grafting (CABG).

Five days before the procedures, clopidogrel was discontinued. Carotid artery stenting (CAS) was performed under local anesthesia through percutaneous transfemoral access using a distal embolic protection device and mesh-covered stent (Fig. 1E-G). Next, without any neurological deficits, the patient was immediately transferred to the cardiac operating room, where the CABG surgery was performed. Clopidogrel (loading dose of 300 mg) was administered in the sixth postoperative hour when major surgical bleeding was excluded. After waking up from sedation, following completion of a combined procedure, aphasia and weakness of the right limbs were observed. Computed tomography scan of the brain revealed an acute stroke of the left hemisphere without intracerebral hemorrhage (Fig. 1H). After neurological consultation dual-antiplatelet therapy was continued. Nine days after the procedure, the patient without aphasia and with mild left-sided hemiparesis was discharged to the rehabilitation department.

Second case

A 51-year-old male with a history of smoking and insulin-dependent diabetes mellitus type 2 was admitted to the hospital with left-arm weakness and left-sided central facial paresis. The symptoms appeared on the day of admission at 5 p.m. after waking up from a nap. The patient was admitted to the hospital around 7 p.m., 6 hours after he had been seen without symptoms. Two days before the admission, the patient experienced severe retrosternal pain which he neglected. Neurological examination revealed central-face palsy on the left side and paresis with sensory disturbances in his left hand. Furthermore, he had no reflexes in the lower limbs. He scored 4 points according to the National Institute of Health Stroke Scale (NIHSS). CT showed completed focal brain ischemia in the right cerebellar hemisphere. The occlusion of the right middle cerebral artery was visible on the angio-CT scan (Fig. 2A). As the time window for pharmacological thrombolysis had elapsed, the thrombolytic treatment was not possible. The patient's condition was consulted with the mechanical thrombectomy team, and consequently to the completed brain ischemic lesion, the patient was referred to conservative therapy.

Simultaneously, in the emergency room, the patient had dyspnea and oxygen saturation decrease, was pale and sweaty. The patient's electrocardiogram revealed ST-elevation in the precordial leads. On laboratory tests, the levels of high-sensitive troponin (hsT) and creatine-kinase MB (CK-MB) were above the upper limits (hsT 0.486 ng/mL, the upper limit of normal 0.014 and CKMB 33 U/L, the upper limit of normal 24 U/L). Emergent bedside transthoracic echocardiography showed reduced left ventricular ejection fraction with 35%, segmental akinesis of the apex, intraventricular septum, anterior and lateral wall. Moreover, a large narrow-pedunculated, movable structure that looked like a clot was observed in the left ventricle (Fig. 3A).

The patient was disqualified from surgical removal of the ventricular thrombus and opening of infarct-elated artery by the Heart Team due to his recent stroke and very high risk of complications during late coronary procedure. Conservative treatment with two subcutaneous administrations of 80 mg low-mass weight heparin was applied daily after multispecialty consultation including a cardiologist, interventional cardiologist, cardiac surgeon, neurologist, anesthetist, and vascular surgeon.

On the second day of admission, the patient's clinical status deteriorated. He was drowsy and developed new symptoms that included peripheral face paresis on the left side, left hemiamblyopia, sensory loss on the left side of the body and paralysis in the left upper limb with decreased muscle tone and brisk reflexes. Paresis occurred in the left lower limb with decreased muscle tone and absent reflexes. He scored 16 points according to the NIHSS scale. A CT scan revealed a hypodense area between the parietal, temporal and occipital lobes in the right hemisphere, in the posterior insula and posterolateral part of the frontal lobe (Fig. 2B). On echocardiography, the structure in the left ventricle was smoother and had fewer movable elements probably due to resorption or small partial separation (Fig. 3B). Again, two subcutaneous administrations of 80 mg low-mass weight heparin daily were advised. A diuretic, small doses of ACE-inhibitor, spironolactone and statin were added to 75 mg acetylic acid and anticoagulation. The P2Y12 inhibitor was excluded due to the high risk of hemorrhage transformation of the ischemic brain

lesions. On subsequent transthoracic echocardiography, the diameters of the structure in the left ventricle were gradually decreasing (Fig. 3A-C). Five days after admission, the patient developed pneumonia (Fig. 4A) which was successfully treated with ceftriaxone.

Subsequently he developed *Clostridium difficile* infection which was treated with an oral dosage of vancomycin. After initial rehabilitation and treatment, the patient's neurological status slightly improved. He was conscious and scored 8 points on the NIHSS scale.

In the next days of hospitalization, the patient was diagnosed with lower limbs' embolism probably due to further detachment from the intraventricular clot. The thrombectomy was delayed, because of the patient's serious condition. To prepare the patient for vascular procedure, planned coronary angiography was performed. It revealed a three-vessel coronary artery disease involving the left descending artery, left circumflex artery and right coronary artery (Fig. 3D-F). The cardiac percutaneous revascularization was abandoned according to scheduled vascular surgery and sepsis. The patient's condition was still serious with worsening ischemia symptoms, especially in the right lower extremity. One month after admission, the patient was transferred to the Intensive Care Unit due to exacerbating respiratory failure and was intubated and put on a ventilator. The patient underwent mechanical thrombectomy and angioplasty of the right popliteal artery (Fig. 4B) and arteries in his right leg localized below the knee. Unfortunately, the patient developed septic shock. Consequently, the right lower extremity was amputated. In subsequent days, the inflammatory and septic parameters increased. After seventy-one days of hospitalization, including forty-two days in the Intensive Care Unit the patient died due to cardiac arrest.

Third case

A 59-year-old female with a history of hypertension, hypercholesterolemia, intermittent claudication, right kidney agenesis, osteoarthritis and right-sided inguinal hernia was admitted to the hospital for coronary angiography. During hospitalization six months earlier, she underwent non-invasive tests due to symptoms including dizziness and dyspnea occurring mainly in the evening or after reaching the first floor by staircase. Stress echocardi-

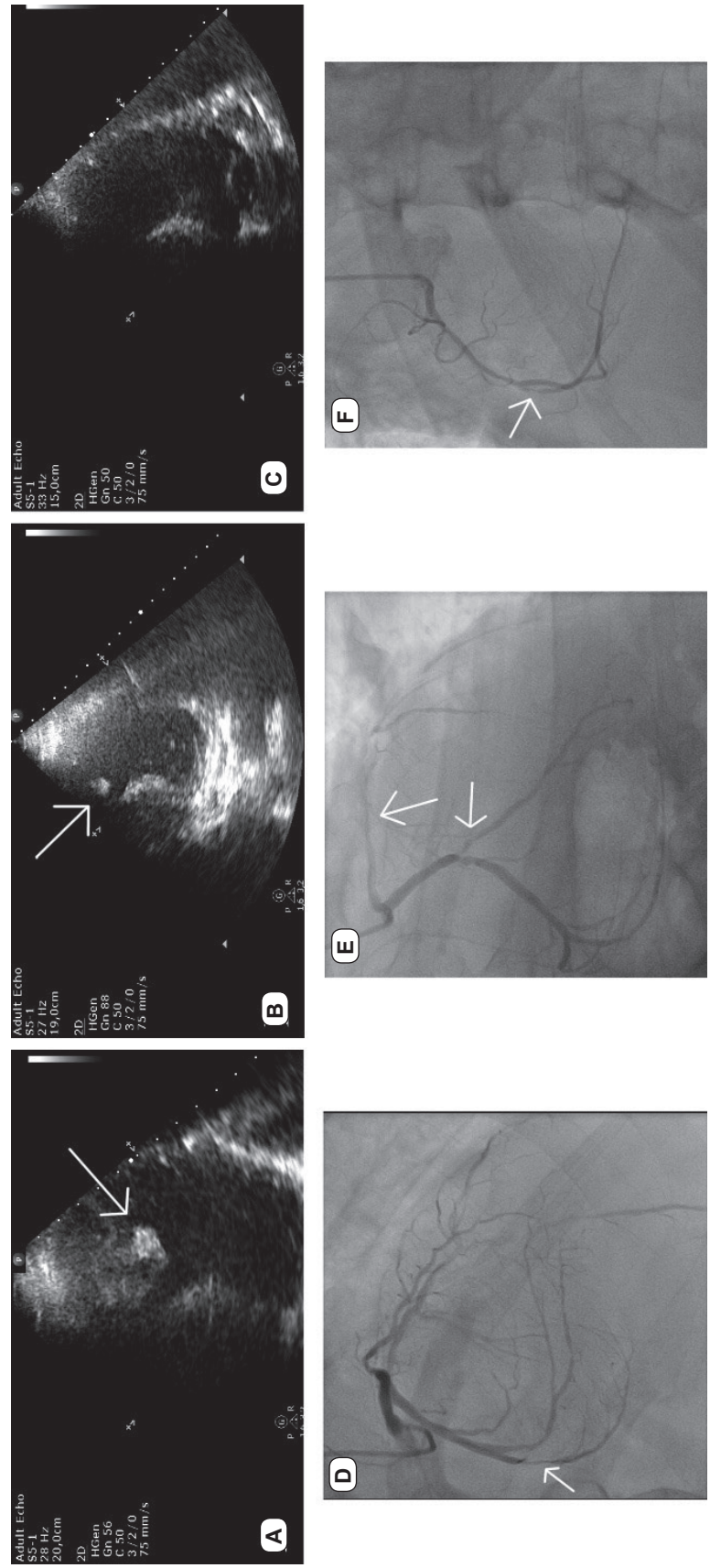

Fig. 3. The sequence of echocardiography of left ventricular clot, diminishing in successive examinations (A–C) and coronary angiogram with multivessel disease (D–F).

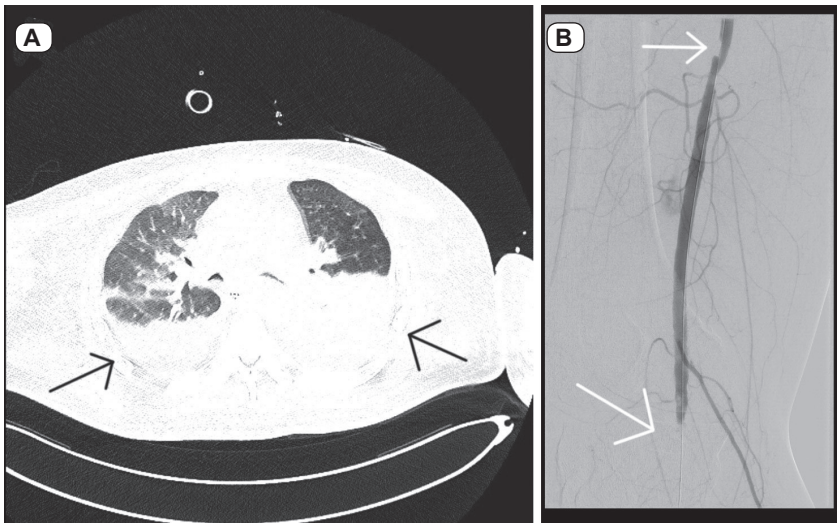

Fig. 4. The pneumonic lesions in both lungs (A). Total occlusion of the right popliteal artery and stenosis of the femoral artery (B).

ography with dobutamine revealed no abnormalities. The patient was referred to the CT angiography of coronary arteries which revealed 60% stenosis in the proximal segment of the left anterior descending artery, 70% stenosis of the left circumflex artery and 50% stenosis in the middle segment of the right coronary artery.

During the current admission, the patient reported newly developed symptoms since the previous hospitalization, including recurrent, excruciating pain in the cervical spine, moving to the retrosternal area, and radiating to the mandible. The symptoms were likely related to coronary artery disease. Moreover, this pain appeared independently of physical activity, sometimes it woke the patient up at night and was sporadically accompanied by sweating and palpitations.

Finally, the patient was qualified for coronary angiography based on her clinical symptoms and the results of non-invasive imaging tests. Angiography performed from the right radial artery revealed an unexpected tortuous course of the right subclavian artery (Fig. 5). Therefore, the procedure had to be re-initiated by shifting the approach to the left radial artery. The coronary angiography showed that the left circumflex artery was stenosed up to 99% while the stenosis of the dominant right coronary artery was 80% in the mid segment and 99% in the distal segment with collateral flow to the right posterior descending artery and left ventricular branch. Furthermore, a borderline lesion in the mid part of the left descending artery was noted (Fig. 5B–D). After angiography, the patient was qualified for two-staged percutaneous coronary intervention, beginning with treating the circumflex artery and then proceeding to the right coronary artery.

After administration of a full dose of unfractionated heparin and ticagrelor, shortly after the intubation of the left main coronary artery with guiding EBU 4.0/6F catheter, the patient developed a new neurological deficit indicating right-hemisphere ischemia. The therapeutic procedure was interrupted before introducing guidewires and the patient was consulted with a neurologist and transferred to the imaging department. The immediately performed CT scan of the brain with the angiography of carotid and intracranial arteries did not show any ischemic or hemorrhagic lesions or obstructed arteries (Fig. 5E). After imaging, the patient was transferred to the neurological ward with a left-sided central facial palsy, paresis in the left arm, left sided dysesthesia, hemianopsia and dysarthria. These symptoms were rated as 14 points on the NIHSS scale. Pharmacological thrombolysis was not applied due to the administration of unfractionated heparin and ticagrelor immediately before the planned percutaneous coronary intervention. Moreover, mechanical thrombectomy could not be applied due to the lack of mechanical stenosis or occlusion in large arteries. On the next day, the control CT scan revealed a focal brain ischemic lesion (11 x 15 mm) localized near the thalamus and visible in the right hemisphere (Fig. 5F). The patient underwent neurological rehabilitation while her pharmacotherapy was optimized. Her neurological status improved significantly during hospitalization. She scored 4 points on the NIHSS scale after a few days, and on discharge, her neurological deficits were diminished. The only remaining neuromuscular symptom was the tandem walking disturbance. During cardiologic consultation, the patient neglected resting stenocardial symptoms, palpitations, and dyspnea. Due to the high risk of hemorrhagic transformation of the brain ischemic lesion, the percutaneous coronary interventions with any potential stent implantations into the left circumflex artery and right coronary artery were postponed and scheduled within the next hospitalization after one month.

Management with MI cardio-cerebral infarction

The intricacy of pathogenesis, the potential for severe complications, and the constraints of limited time available for decision-making are the cornerstones that must be addressed in the management of CCI. Even though studies present various treatment strategies for CCI, the use of an intravenous tissue-plasminogen activator could be the most beneficial for those patients because of its guideline indication (1, 2, 26, 45, 46). However, administration of intravenous alteplase for the treatment of concurrent MI and stroke is not feasible because the doses used to treat MI are higher than those recommended for an acute ischemic stroke. Administration of higher than recommended doses of alteplase in the case of an acute ischemic stroke can result in cerebral hemorrhagic transformation. For the treatment of MI, lower than recommended doses have an unknown efficacy. Furthermore, percutaneous coronary intervention and angioplasty constitute the first-line treatment for MI (40). The American Heart Association and American Stroke Association indicate that a reasonable approach to patients with

CCI includes treatment with intravenous alteplase at a dose appropriate for cerebral ischemia, which is then to be followed by percutaneous coronary angioplasty and stenting if necessary (40). It is reasonable to treat patients with acute ischemic stroke and recent three-month history of MI with intravenous alteplase provided that MI was NSTEMI or STEMI and involved the right or inferior myocardium or the left anterior myocardium (47).

Fibrinolytic treatment for acute myocardial ischemia can result in disintegration of intracardiac thrombi and cause thromboembolic complications such as stroke. If the cause of the stroke is cardioembolism, the acute ischemic stroke is more often major in extent and likely to produce severe disability (48–50). In such a case, according to the guidelines, the treatment with intravenous alteplase may be reasonable. The use of intravenous alteplase is of uncertain benefit in case of a moderate ischemic stroke which is likely to produce mild disability (47).

The optimal management of patients with concomitant severe carotid stenosis and cardiac disease remains an unresolved issue. Both significant internal carotid artery stenosis and unstable carotid plaque causing cerebral hypoperfusion are risk factors for stroke associated with cardiac surgery (51, 52). The risk of a perioperative stroke is approximately 8.5% in patients with symptomatic internal carotid artery stenosis and is a serious complication of cardiac surgery with an in-hospital mortality rate reaching 22% (53). There are several possible treatment strategies for significant concurrent carotid and cardiac diseases. For stable patients, staged revascularization seems to be an adequate strategy. CAS and delayed cardiac surgery have already been reported to have a slightly lower major adverse event rate than staged carotid endarterectomy and cardiac surgery (54). However, unstable patients with aggressive atherosclerotic disease manifestation represent a subgroup of extremely high-risk cases where the combination with a less invasive strategy, e.g., carotid artery stenting with a simultaneous cardiovascular surgery may reduce the global risk of both procedures (55). The procedure of this hybrid treatment was established in several studies. The inclusion of the antithrombotic regimen balances the risk of bleeding during surgery and thrombosis after stenting (55–60). Based on available data, this procedure is safe and effective in such patients. Dzierwa et al (61) have analyzed one-day sequential carotid artery stenting and a cardiac surgery approach. They have shown that all complications occurred among patients at high surgical risk, whereas NSTEMI seemed to be a risk for major adverse cardiac and cerebrovascular events. This report indicates that CAS immediately followed by cardiac surgery could be considered a viable treatment option for patients requiring urgent cardiac surgery.

In turn, stroke management after planned or urgent percutaneous coronary intervention remains an unresolved issue. Although thrombolysis can be applied when a stroke occurs, there are some

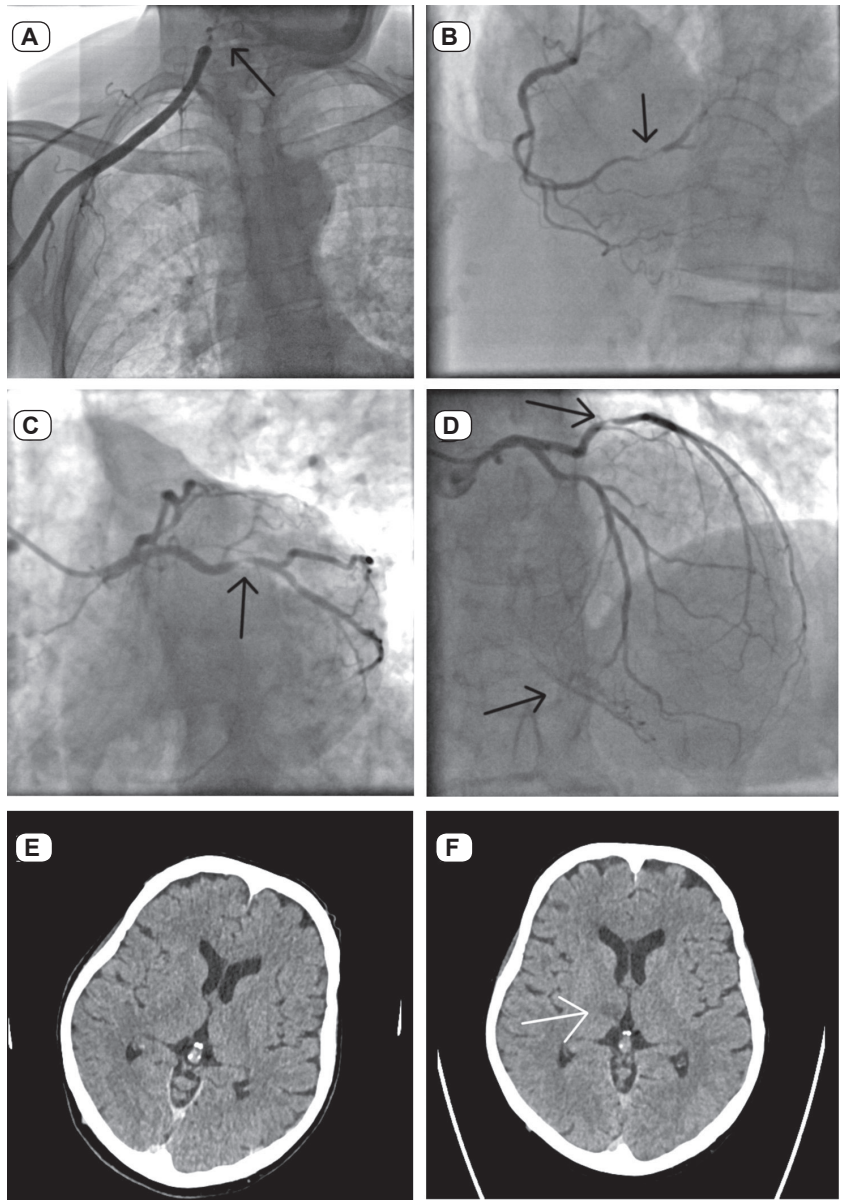

Fig. 5. Angiography of an extremely tortuous right subclavian artery (A) shows a significant stenosis of the right coronary artery (B), left circumflex artery (C) and borderline lesion in the left anterior descending artery with retrograde circulation to right coronary artery (D). The comparison of baseline (E) and next-day results after neurological symptoms onset (F) brain scan shows an ischemic lesion in the right thalamus.

limiting factors that should be considered while the decision about further therapy is taken. Firstly, when the clogging material consists mainly of particles of an atheromatous plaque, the intravenous thrombolysis has a very limited effect. Secondly, anticoagulants and antiplatelet drugs used during percutaneous coronary intervention increase the risk of hemorrhagic complications after intravenous thrombolysis, particularly including the hemorrhagic transformation of ischemic stroke with a subsequent worsening of patient condition, permanent disability, or even death. Also, the risk of access site bleeding is increased and generates problems particularly in the case of the femoral approach. Hence, the potential risks and benefits of thrombolysis should be discussed with the patient while the decisions regarding the treatment should be individualized according to patient's comorbidities, clinical status, risk of transformation, and patient's preferences (62). Nowadays, there is another option of stroke therapy by way of mechanical thrombectomy (63). Thrombectomy is safe and effective also in patients with chronic treatment with oral anticoagulants (64). Moreover, the retrospective data prove that antiplatelet drugs might improve patients' outcomes with a slight increase in hemorrhage transformation (65). Unfortunately, mechanical thrombectomy is the preferred treatment option only in patients with large vessel occlusion, not for lacunar ischemia (63).

Nowadays, selecting a proper and most suitable therapy for CCI is a demanding challenge for physicians. Distinct aspects of patients' comorbidities and dynamically changing clinical symptoms should be considered. The optimal treatment path for every patient with CCI is not described in existing guidelines and the detailed analysis of every patient's status, based on the experience of a multidisciplinary team, is required for achieving satisfactory long-term outcomes.

Perspectives

Large clinical trials are needed for enhancing our understanding of concomitant acute myocardial infarction and stroke. The lack of guidelines impedes the diagnosis and management of CCI. Although the American Heart Association and American Stroke Association recommendations for the treatment of stroke in the case of present or recent myocardial infarction have been published, further research on the benefits of therapy with alteplase for CCI would be desirable. Patients with CCI have different risks including the factor of time elapsed since the first symptoms onset. Therefore, more individualized treatment options should be established. Also, more studies on the optimal management of concurrent carotid and cardiac diseases are needed for both stable and unstable patients to demonstrate the best approach to this condition.

Learning points

The onset of CCI is a life-threatening clinical situation.

The proper treatment of CCS is challenging and requires detailed analysis of patient's comorbidity and clinical status.

Currently, there are no large clinical studies analyzing the outcomes of treatment options in patients with CCI.

The intravenous alteplase administration in cerebral dosage and further percutaneous coronary intervention is probably the most beneficial option for CCI therapy considering available data.

References

1. Roth GA, Abate D, Abate KH, Abay SM, Abbafati C, Abbasi N et al. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet 2018; 392: 1736–1788. //doi.org/10.1016/S0140-6736(18)32203-7.

2. Witt BJ, Ballman K V, Brown RD, Meverden RA, Jacobsen SJ, Roger VL. The incidence of stroke after myocardial infarction: a meta-analysis. Am J Med 2006; 119. //doi.org/10.1016/J.AMJMED.2005.10.058.

3. Stepien K, Nowak K, Wypasek E, Zalewski J, Undas A. High prevalence of inherited thrombophilia and antiphospholipid syndrome in myocardial infarction with non-obstructive coronary arteries: Comparison with cryptogenic stroke. Int J Cardiol 2019; 290: 1–6. //doi.org/10.1016/J.IJCARD.2019.05.037.

4. Crenshaw BS, Ward SR, Granger CB, Stebbins AL, Topol EJ, Califf RM. Atrial fibrillation in the setting of acute myocardial infarction: The GUSTO-I experience. J Am Coll Cardiol 1997; 30: 406–13. //doi.org/10.1016/S0735-1097(97)00194-0.

5. Tsai LM, Lin LJ, Teng JK, Chen JH. Prevalence and clinical significance of left atrial thrombus in nonrheumatic atrial fibrillation. Int J Cardiol 1997; 58: 163–169. //doi.org/10.1016/S0167-5273(96)02862-8.

6. Kaarisalo MM, Immonen-Räihä P, Marttila RJ, Salomaa V, Torppa J, Tuomilehto J. The risk of stroke following coronary revascularization – a population-based long-term follow-up study. Scand Cardiovasc J 2002; 36: 231–236. //doi.org/10.1080/14017430260180391.

7. Zalewski J, Nowak K, Furczynska P, Zalewska M. Complicating Acute Myocardial Infarction. Current Status and Unresolved Targets for Subsequent Research. J Clin Med 2021; 10. //doi.org/10.3390/JCM10245904.

8. Ay H, Koroshetz WJ, Benner T, Vangel MG, Melinosky C, Arsava EM et al. Neuroanatomic correlates of stroke-related myocardial injury. Neurology 2006; 66: 1325–1329. //doi.org/10.1212/01.WNL.0000206077.13705.6D.

9. Yeo LLL, Andersson T, Yee KW, Tan BYQ, Paliwal P, Gopinathan A et al. Synchronous cardiocerebral infarction in the era of endovascular therapy: which to treat first? J Thromb Thrombolysis 2017; 44: 104–111. //doi.org/10.1007/S11239-017-1484-2.

10. Ng TP, Wong C, Leong ELE, Tan BY, Chan MYY, Yeo LL et al. Simultaneous cardio-cerebral infarction: a meta-analysis. QJM 2022; 115: 374–380. //doi.org/10.1093/QJMED/HCAB158.

11. Krawczyk K, Stepien K, Nowak K, Nessler J, Zalewski J. ST-segment re-elevation following primary angioplasty in acute myocardial infarction with patent infarct-related artery: Impact on left ventricular function recovery and remodeling. Postepy w Kardiologii Interwencyjnej 2019; 15. //doi.org/10.5114/aic.2019.90215.

12. Nowak K, Stepien A, Zalewska M, Kanclerz G, Yika ADM, et al. Screening diagnosis of dementia in patients following heart failure decompensation is associated with a higher relative wall thickness. Bratisl Lek Listy 2022; 123: 685–691. //doi.org/10.4149/BLL_2022_109.

13. Stepien K, Furczynska P, Zalewska M, Nowak K, Wlodarczyk A, Owsianka I et al. Dementia screening in elderly high-risk patients following heart failure decompensation may predict unfavorable long-term clinical outcomes. Minerva Cardiology and Angiology 2021; 69. //doi.org/10.23736/S2724-5683.20.05157-9.

14. Brammås A, Jakobsson S, Ulvenstam A, Mooe T. Mortality after ischemic stroke in patients with acute myocardial infarction: predictors and trends over time in Sweden. Stroke 2013; 44: 3050–3055. //doi.org/10.1161/STROKEAHA.113.001434.

15. Binsell-Gerdin E, Graipe A, Ögren J, Jernberg T, Mooe T. Hemorrhagic stroke the first 30 days after an acute myocardial infarction: incidence, time trends and predictors of risk. Int J Cardiol 2014; 176: 133–138. //doi.org/10.1016/J.IJCARD.2014.07.032.

16. Kajermo U, Ulvenstam A, Modica A, Jernberg T, Mooe T. Incidence, trends, and predictors of ischemic stroke 30 days after an acute myocardial infarction. Stroke 2014; 45: 1324–1330. //doi.org/10.1161/STROKEAHA.113.001963.

17. Giugliano RP, Giraldez RR, Morrow DA, Antman EM, Gibson CM, Mohanavelu S et al. Relations between bleeding and outcomes in patients with ST-elevation myocardial infarction in the ExTRACT-TIMI 25 trial. Eur Heart J 2010; 31: 2103–2110. //doi.org/10.1093/EURHEARTJ/EHQ100.

18. Ulvenstam A, Kajermo U, Modica A, Jernberg T, Söderström L, Mooe T. Incidence, trends, and predictors of ischemic stroke 1 year after an acute myocardial infarction. Stroke 2014; 45: 3263–3268. //doi.org/10.1161/STROKEAHA.114.005770.

19. Luo J, Li H, Qin X, Liu B, Zhao J, Maihe G et al. Increased risk of ischemic stroke associated with new-onset atrial fibrillation complicating acute coronary syndrome: A systematic review and meta-analysis. Int J Cardiol 2018; 265: 125–131. //doi.org/10.1016/J.IJCARD.2018.04.096.

20. Budaj A, Flasinska K, Gore JM, Anderson FA, Dabbous OH, Spencer FA et al. Magnitude of and risk factors for in-hospital and postdischarge stroke in patients with acute coronary syndromes: findings from a Global Registry of Acute Coronary Events. Circulation 2005; 111: 3242–3247. //doi.org/10.1161/CIRCULATIONAHA.104.512806.

21. Guptill JT, Mehta RH, Armstrong PW, Horton J, Laskowitz D, James S et al. Stroke after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction: timing, characteristics, and clinical outcomes. Circ Cardiovasc Interv 2013; 6: 176–183. //doi.org/10.1161/CIRCINTERVENTIONS.112.000159.

22. Merkler AE, Diaz I, Wu X, Murthy SB, Gialdini G, Navi BB et al. Duration of Heightened Ischemic Stroke Risk After Acute Myocardial Infarction. Journal of the American Heart Association: Cardiovascular and Cerebrovascular Disease 2018; 7. //doi.org/10.1161/JAHA.118.010782.

23. Van De Werf F. Primary versus tenecteplase-facilitated percutaneous coronary intervention in patients with ST-segment elevation acute myocardial infarction (ASSENT-4 PCI): randomised trial. Lancet 2006; 367: 569–578. //doi.org/10.1016/S0140-6736(06)68147-6.

24. Armstrong PW, Gershlick AH, Goldstein P, Wilcox R, Danays T, Lambert Y et al. Fibrinolysis or primary PCI in ST-segment elevation myocardial infarction. N Engl J Med 2013; 368: 1379–1387. //doi.org/10.1056/NEJMOA1301092.

25. Patel MR, Meine TJ, Lindblad L, Griffin J, Granger CB, Becker RC et al. Cardiac tamponade in the fibrinolytic era: analysis of >100,000 patients with ST-segment elevation myocardial infarction. Am Heart J 2006; 151: 316–322. //doi.org/10.1016/J.AHJ.2005.04.014.

26. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39: 119–177. //doi.org/10.1093/EURHEARTJ/EHX393.

27. Jolly SS, James S, Džavík V, Cairns JA, Mahmoud KD, Zijlstra F et al. Thrombus Aspiration in ST-Segment-Elevation Myocardial Infarction: An Individual Patient Meta-Analysis: Thrombectomy Trialists Collaboration. Circulation 2017; 135: 143–152. //doi.org/10.1161/CIRCULATIONAHA.116.025371.

28. Fröbert O, Lagerqvist B, Olivecrona GK, Omerovic E, Gudnason T, Maeng M et al. Thrombus aspiration during ST-segment elevation myocardial infarction. N Engl J Med 2013; 369: 1587–1597. //doi.org/10.1056/NEJMOA1308789.

29. Vlaar PJ, Svilaas T, van der Horst IC, Diercks GF, Fokkema ML, de Smet BJ et al. Cardiac death and reinfarction after 1 year in the Thrombus Aspiration during Percutaneous coronary intervention in Acute myocardial infarction Study (TAPAS): a 1-year follow-up study. Lancet 2008; 371: 1915–1920. //doi.org/10.1016/S0140-6736(08)60833-8.

30. Jolly SS, Cairns JA, Yusuf S, Meeks B, Gao P, Hart RG et al. Stroke in the TOTAL trial: a randomized trial of routine thrombectomy vs. percutaneous coronary intervention alone in ST elevation myocardial infarction. Eur Heart J 2015; 36: 2364–2372. //doi.org/10.1093/EURHEARTJ/EHV296.

31. Aradi D, Komócsi A, Vorobcsuk A, Serebruany VL. Impact of clopidogrel and potent P2Y 12 -inhibitors on mortality and stroke in patients with acute coronary syndrome or undergoing percutaneous coronary intervention: a systematic review and meta-analysis. Thromb Haemost 2013; 109: 93–101. //doi.org/10.1160/TH12-06-0377.

32. Wiviott SD, Braunwald E, McCabe CH, Montalescot G, Ruzyllo W, Gottlieb S et al. Prasugrel versus clopidogrel in patients with acute coronary syndromes. N Engl J Med 2007; 357: 2001–2015. //doi.org/10.1056/NEJMOA0706482.

33. Ferguson JJ, Califf RM, Antman EM, Cohen M, Grines CL, Goodman S, et al. Enoxaparin vs unfractionated heparin in high-risk patients with non-ST-segment elevation acute coronary syndromes managed with an intended early invasive strategy: primary results of the SYNERGY randomized trial. JAMA 2004; 292: 45–54. //doi.org/10.1001/JAMA.292.1.45.

34. Goodman SG, Fitchett D, Armstrong PW, Tan M, Langer A. Randomized evaluation of the safety and efficacy of enoxaparin versus unfractionated heparin in high-risk patients with non-ST-segment elevation acute coronary syndromes receiving the glycoprotein IIb/IIIa inhibitor eptifibatide. Circulation 2003; 107: 238–244. //doi.org/10.1161/01.CIR.0000050144.67910.13.

35. Bonaca MP, Goto S, Bhatt DL, Steg PG, Storey RF, Cohen M et al. Prevention of Stroke with Ticagrelor in Patients with Prior Myocardial Infarction: Insights from PEGASUS-TIMI 54 (Prevention of Cardiovascular Events in Patients With Prior Heart Attack Using Ticagrelor Compared to Placebo on a Background of Aspirin-Thrombolysis in Myocardial Infarction 54). Circulation 2016; 134: 861–871. //doi.org/10.1161/CIRCULATIONAHA.116.024637.

36. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J. Embolic potential of left ventricular thrombus after myocardial infarction: a two-dimensional echocardiographic study of 119 patients. J Am Coll Cardiol 1985; 5: 1276–1280. //doi.org/10.1016/S0735-1097(85)80336-3.

37. Lamas GA, Vaughan DE, Pfeffer MA. Left ventricular thrombus formation after first anterior wall acute myocardial infarction. Am J Cardiol 1988; 62: 31–35. //doi.org/10.1016/0002-9149(88)91360-4.

38. Robinson AA, Jain A, Gentry M, McNamara RL. Left ventricular thrombi after STEMI in the primary PCI era: A systematic review and meta-analysis. Int J Cardiol 2016; 221: 554–549. //doi.org/10.1016/J.IJCARD.2016.07.069.

39. Zalewski J, Zmudka K, Musialek P, Zajdel W, Pieniazek P, Kadzielski A et al. Detection of microvascular injury by evaluating epicardial blood flow in early reperfusion following primary angioplasty. Int J Cardiol 2004; 96: 389–396. //doi.org/10.1016/J.IJCARD.2003.08.009.

40. Fuchs S, Stabile E, Kinnaird TD, Mintz GS, Gruberg L, Canos DA et al. Stroke complicating percutaneous coronary interventions: incidence, predictors, and prognostic implications. Circulation 2002; 106: 86–91. //doi.org/10.1161/01.CIR.0000020678.16325.E0.

41. Yamamoto K, Natsuaki M, Morimoto T, Shiomi H, Matsumura-Nakano Y, Nakatsuma K et al. Periprocedural Stroke After Coronary Revascularization (from the CREDO-Kyoto PCI/CABG Registry Cohort-3). Am J Cardiol 2021; 142: 35–43. //doi.org/10.1016/J.AMJCARD.2020.11.031.

42. Raposo L, Madeira S, Teles RC, Santos M, Gabriel HM, Gonçalves P et al. Neurologic complications after transradial or transfemoral approach for diagnostic and interventional cardiac catheterization: A propensity score analysis of 16,710 cases from a single centre prospective registry. Catheter Cardiovasc Interv 2015; 86: 61–70. //doi.org/10.1002/CCD.25884.

43. Shoji S, Kohsaka S, Kumamaru H, Sawano M, Shiraishi Y, Ueda I et al. Stroke After Percutaneous Coronary Intervention in the Era of Transradial Intervention. Circ Cardiovasc Interv 2018; 11. //doi.org/10.1161/CIRCINTERVENTIONS.118.006761.

44. Alkhouli M, Alqahtani F, Tarabishy A, Sandhu G, Rihal CS. Incidence, Predictors, and Outcomes of Acute Ischemic Stroke Following Percutaneous Coronary Intervention. JACC Cardiovasc Interv 2019; 12: 1497–1506. //doi.org/10.1016/J.JCIN.2019.04.015.

45. Berge E, Whiteley W, Audebert H, Marchis GM De, Fonseca AC, Padiglioni C et al. European Stroke Organisation (ESO) guidelines on intravenous thrombolysis for acute ischaemic stroke. Eur Stroke J 2021; 6: I–LXII. //doi.org/10.1177/2396987321989865.

46. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation 2018; 138: e618–651. //doi.org/10.1161/CIR.0000000000000617.

47. Demaerschalk BM, Kleindorfer DO, Adeoye OM, Demchuk AM, Fugate JE, Grotta JC et al. Scientific Rationale for the Inclusion and Exclusion Criteria for Intravenous Alteplase in Acute Ischemic Stroke: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 2016; 47: 581–641. //doi.org/10.1161/STR.0000000000000086.

48. Timsit SG, Sacco RL, Mohr JP, Foulkes MA, Tatemichi TK, Wolf PA et al. Brain infarction severity differs according to cardiac or arterial embolic source. Neurology 1993; 43: 728–733. //doi.org/10.1212/WNL.43.4.728.

49. Timsit SG, Sacco RL, Mohr JP, Foulkes MA, Tatemichi TK, Wolf PA et al. Early clinical differentiation of cerebral infarction from severe atherosclerotic stenosis and cardioembolism. Stroke 1992; 23: 486–491. //doi.org/10.1161/01.STR.23.4.486.

50. Guglielmi V, LeCouffe NE, Zinkstok SM, Compagne KCJ, Eker R, Treurniet KM et al. Collateral Circulation and Outcome in Atherosclerotic Versus Cardioembolic Cerebral Large Vessel Occlusion. Stroke 2019; 50: 3360–3368. //doi.org/10.1161/STROKEAHA.119.026299.

51. Naylor AR, Mehta Z, Rothwell PM, Bell PRF. Carotid artery disease and stroke during coronary artery bypass: A critical review of the literature. European Journal of Vascular and Endovascular Surgery 2002; 23: 283–294. //doi.org/10.1053/ejvs.2002.1609.

52. Likosky DS, Marrin CAS, Caplan LR, Baribeau YR, Morton JR, Weintraub RM et al. Determination of etiologic mechanisms of strokes secondary to coronary artery bypass graft surgery. Stroke 2003; 34: 2830–2834. //doi.org/10.1161/01.STR.0000098650.12386.B3.

53. Bucerius J, Gummert JF, Borger MA, Walther T, Doll N, Onnasch JF et al. Stroke after cardiac surgery: a risk factor analysis of 16,184 consecutive adult patients. Ann Thorac Surg 2003; 75: 472–478. //doi.org/10.1016/S0003-4975(02)04370-9.

54. Shishehbor MH, Venkatachalam S, Sun Z, Rajeswaran J, Kapadia SR, Bajzer C et al. A direct comparison of early and late outcomes with three approaches to carotid revascularization and open heart surgery. J Am Coll Cardiol 2013; 62: 1948–1956. //doi.org/10.1016/J.JACC.2013.03.094.

55. Versaci F, Reimers B, Del Giudice C, Schofer J, Giacomin A, Saccà S et al. Simultaneous Hybrid Revascularization by Carotid Stenting and Coronary Artery Bypass Grafting: The SHARP Study. JACC Cardiovasc Interv 2009; 2: 393–401. //doi.org/10.1016/J.JCIN.2009.02.010.

56. Mićović S, Bošković S, Sagić D, Radak Ď, Perić M, Milojević P et al. Simultaneous hybrid carotid stenting and coronary bypass surgery versus concomitant open carotid and coronary bypass surgery: a pilot, feasibility study. European Journal of Cardio-Thoracic Surgery 2014; 46: 857–862. //doi.org/10.1093/EJCTS/EZU009.

57. Piątek J, Kędziora A, Dzierwa K, Konstanty-Kalandyk J, Wróżek M, Bryniarski KL et al. Hybrid one-day coronary artery bypass grafting and carotid artery stenting – cardiac surgeons' perspective on the procedure's safety. Postepy Kardiol Interwencyjnej 2018; 14: 99. //doi.org/10.5114/AIC.2018.74362.

58. Zivkovic I, Krasic S, Milacic P, Milicic M, Vukovic P, Tabakovic Z et al. Same-Day Carotid Artery Stenting and Coronary Artery Bypass Surgery. The Texas Heart Institute Journal 2023; 50. //doi.org/10.14503/THIJ-21-7781.

59. Xiang B, Luo X, Yang Y, Qiu J, Zhang J, Li L et al. Midterm results of coronary artery bypass graft surgery after synchronous or staged carotid revascularization. J Vasc Surg 2019; 70: 1942–1849. //doi.org/10.1016/j.jvs.2019.02.057.

60. Castriota F, Tomai F, Secco GG, Reimers B, Piccoli A, De Persio G et al. Early and late clinical outcomes of endovascular, surgical, and hybrid revascularization strategies of combined carotid and coronary artery diseases: the FRIENDS study group (Finalized Research In ENDovascular Strategies). European Heart Journal Supplements 2015: 17. //doi.org/10.1093/eurheartj/suv008.

61. Dzierwa K, Piatek J, Paluszek P, Przewlocki T, Tekieli L, Konstanty-Kalandyk J et al. One-day, sequential carotid artery stenting followed by cardiac surgery in patients with severe carotid and cardiac disease. Vascular Medicine (United Kingdom) 2019; 24: 431–438. //doi.org/10.1177/1358863X19872547/ASSET/IMAGES/LARGE/10.1177_1358863X19872547-FIG2.JPEG.

62. Abdel-Latif A, Misumida N. Ischemic Stroke After Percutaneous Coronary Intervention: Rare, But Devastating*. JACC Cardiovasc Interv 2019; 12: 1507–1509. //doi.org/10.1016/J.JCIN.2019.05.013.

63. Turc G, Bhogal P, Fischer U, Khatri P, Lobotesis K, Mazighi M et al. European Stroke Organisation (ESO) – European Society for Minimally Invasive Neurological Therapy (ESMINT) Guidelines on Mechanical Thrombectomy in Acute Ischemic Stroke. J Neurointerv Surg 2019. //doi.org/10.1136/neurintsurg-2018-014569.

64. Nowak K, Wlodarczyk E, Porębska K, Chatys-Bogacka Z, Jagiełła J, Pułyk R et al. Mechanical thrombectomy for acute ischaemic stroke during therapeutic anticoagulation: long-term outcomes. Neurol Neurochir Pol 2020; 54: 538–543. //doi.org/10.5603/PJNNS.A2020.0088.

65. Pandhi A, Tsivgoulis G, Krishnan R, Ishfaq MF, Singh S, Hoit D et al. Antiplatelet pretreatment and outcomes following mechanical thrombectomy for emergent large vessel occlusion strokes. J Neurointerv Surg 2018; 10: 828–833. //doi.org/10.1136/NEURINTSURG-2017-013532.

Received July 27, 2023.
Accepted December 22, 2023.