IJSRM
INTERNATIONAL JOURNAL OF SCIENCE AND RESEARCH METHODOLOGY
An Official Publication of Human Journals

Human Journals
Research Article
February 2021 Vol.:17, Issue:4

Evaluation of Type I and Type II Neurological Complications Occurring in the Early Period after Cardiac Surgery

IJSRM
INTERNATIONAL JOURNAL OF SCIENCE AND RESEARCH METHODOLOGY
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Submitted: 03 January 2021
Revised: 23 January 2021
Accepted: 12 February 2021

Keywords: Cardiac surgery, coronary artery bypass grafting, mitral valve replacement, neurologic damage

ABSTRACT

Background: Severe or mild cerebrovascular clinical conditions are one of the most serious complications of cardiac surgery. In this article, we aimed to evaluate patients with postoperative neurological damage in terms of diagnosis and treatment in patients undergoing open-heart surgery. **Material and Methods:** In the last 10 years, 5022 open-heart surgery patients who were performed by the same surgical team in 2 hospitals in our city were evaluated. Patients with Type I and Type II neurological damage in the early postoperative period (within the first 48 hours) were identified. Postoperative neurological examination, monitoring and treatment practices in these patients are discussed in this article. **Results:** Only patients who underwent coronary artery surgery, only heart valve surgery, and a combination of the two were included in this study. Hypertension was evident in 51.1% of patients, diabetes mellitus in 43.3%, and hypercholesterolemia in 79.9%. Among the 5022 patients who underwent surgery, 151 (male: 105, female: 46) patients with Type I and Type II neurological damage were detected in the postoperative period. The mean age of these patients was 61.4 ± 8.1 years. 62 of these patients were patients with Type I neurological damage. Accordingly, it was determined that the embolic event causing ischemia in the etiology of cerebrovascular damage or complaint was responsible for the entire etiology. While permanent neurological damage was 1 % among all operated patients, the percentage of permanent damage was 34.4 % among neurological patients. There was mortality in 34 early-term patients (0.7%).**Conclusion:** As a result of the study, the rate of Type I and Type II neurological damage after open-heart surgery in our city was found to be 3.0 %. Neurological damage in our patients was observed to be caused by transient neurological clinical conditions and ischemic damage due to intra-operative embolism.

INTRODUCTION:

Neurological damage after cardiac surgery is a situation that causes disappointment and distress both by the patient, patient relatives, and the surgical team. A simpler clinical picture Type II complaints or the presence of Type I complaints with more severe clinical symptoms, even if the patient's cardiac problems are resolved, the morbidity caused by cerebral events significantly affects the patient's quality of life and causes high costs.

Stroke, ischemic attack, coma, and encephalopathy, which can be more severe clinical conditions (Type I) and usually occur due to embolic causes, are a condition that can affect patients more and later change their lives. These injuries are usually caused by clinically and radiologically visible lesions in the brain parenchyma. The incidence of stroke (permanent damage) after cardiac surgery varies between 1 and 4% in many literatures (1-3). Type II injuries (the complex of clinical conditions that cannot be diagnosed by standard clinical and radiological findings such as confusion, agitation, memory impairment, seizures without signs of focal damage, and neuro-cognitive dysfunction) are usually seen between 40-70%(2-4). In our article, we presented these 2 types of conditions to our patients, discussed and evaluated the treatments that can be given to them.

MATERIALS AND METHODS:

In two hospitals in our city (Atatürk University and Regional Training and Research Hospital) cardiac surgery operations were performed by the same team on 5022 patients for 10 years, using a classical cross-clamp, at the cardiovascular surgery clinics. These patients consisted of only coronary bypass surgery, only aortic and mitral valve surgery, and their combinations. 3755 of these patients were male (74.7%), the rest were female. Any neurological deficits occurring in the early postoperative period were evaluated. More severe Type I neurological damage conditions, including stroke, transient ischemic attack, coma, and encephalopathy, were evaluated with neurology and radiology consultations in the first 48 hours postoperatively, radiological examinations were performed, and were examined for treatment options. Patients with milder Type II clinical conditions with confusion, agitation, memory impairment, and neuro-cognitive dysfunction were also examined by the neurology clinic and the necessary medications were given.

Pediatric patients, off-pump cardiac surgeries, aortic surgery (aneurysm-dissection surgery), patients with a previous history of cerebrovascular events, carotid artery disease requiring intervention, patients with creatinine levels above 1.2 mg/dL and repeat cardiac surgeries were not included in our study. Postoperative neurological examination, monitoring, and treatment practices in these patients are also discussed in this article. Preoperative, intra-operative and postoperative data of these patients were obtained by scanning files from the hospital's electronic database and archive. Preoperative characteristics of our patients are summarized in Table-1.

Surgical procedure

Anesthesia

In the operations performed under general anesthesia remifentanil (0.5-1 g/kg per minute) and propofol (3 mg/kg per hour) were used. The neuromuscular blockage was performed with pancuronium bromide or vecuronium at a dose of 0.1-0.15 mg / kg. All operations were done by cross-clamp with cardiopulmonary bypass and systemic blood pressure was maintained between 50 and 60 mmHg. Dopamine, noradrenaline or nitroglycerin infusions were used to adjust systemic pressure if necessary.

Surgical Techniques

Operations were performed through median sternotomy. Conduits were harvested and prepared. Cardiopulmonary bypass was instituted by using ascending aortic cannulation and two-stage venous cannulation in the right atrium. Double venous cannula was used in mitral valve surgery. Heparin was given at a dose of 300 lU/kg to achieve a target activated clotting time > 450 seconds. The tranexamic agent was not used as an anti-bleeding agent. The extracorporeal circuit was primed with 1000 mL of Hartmann's solution, 500 mL of gelofusine, 0.5 g/kg of mannitol, 7 mL of 10% calcium gluconate, and 60 mg of heparin. The pulsatile flow was preferred for cardiopulmonary pump flow. Systemic temperature was kept between 28°C and 32°C (middle hypothermic). The aorta was cross clamped, and myocardial protection was achieved with Plegisol or Del-Nido solutions antegrade and retrograde blood cardioplegia. For coronary bypass surgery, the distal anastomoses were constructed with running sutures of 7-0 or 8-0 polypropylene and the proximal anastomoses were connected to the ascending aorta with 5-0 or 6-0 polypropylene sutures during a side clamping period. For valve surgery, valve replacements

were performed by performing left atriotomy for the mitral valve and aortotomy for the aortic valve. After the patient was weaned from cardiopulmonary bypass and decannulated, the heparin was reversed with protamine infusion (1/1.5 rate). In all patients, two drainage tubes were inserted into to the space of a 32 F drainage left thorax and a 30 F drainage anterior mediastinal. The blood loss was recorded until the drain removal the following day. The average 20 mm Hg continuous absorbing pressure was applied for drainage. Chest tubes were removed the following day when the drainage was less than 20 ml/h for a consecutive 4 h. The data and findings related to the operation are shown in Table-2. In addition to these routine managements in our clinic, the use of alpha stat blood gas, moderate hypothermia and partial patient blood management practices were standardized in all our cases. Although cerebral monitoring (electroencephalography and near-infrared spectroscopy) is not routine, it is frequently used in carotid and aortic surgery patients besides cardiac surgery in many clinics. Cerebral monitoring devices were not available in the hospitals where our operations were performed. Further, since our patients did not include additional carotid and aortic surgery cases, there was no indication for neurological monitoring.

In our clinic, patients with anemia in the preoperative period in terms of blood management were referred to the hematology clinic and necessary investigations were carried out, and if necessary, blood replacement was performed and hemoglobin levels were increased above 11 g/dL. However, in some patients whose emergency waiting was unsuitable, many anemic patients were operated in the preoperative period without any intervention. In terms of strategies to reduce intra-operative blood loss, procedures such as the use of cell-saver in surgical procedures and in patients with high bleeding risk, and anti-bleeding agents are preferred in clinics. In our patients, cell-saver use or anti-bleeding agents such as tranexamic acid were not used. In many of our patients, blood was taken from the patient for autologous donation at the stage of anesthesia and was re-transfused at the end of the surgery. In the intra-operative period, although individualized according to the patient and conditions, the threshold hemoglobin value for intra-operative transfusion was accepted as 7 g/dL in patients without comorbidity, and 9-10 g/dL in patients with comorbidity. Besides demographic information, preoperative comorbidities, hemoglobin, glucose, leukocyte and creatinine values were recorded. The intra-operative surgical procedure, cross-clamp and cardiopulmonary bypass times, blood and blood product transfusions, medications, ultra-filtration and intra-aortic balloon pump usage, lowest hemoglobin, highest

glucose and lowest temperature values were recorded. In the postoperative period, depending on the length of the cases and the age of the patients, awakening times were evaluated in the first 48 hours, as well as cerebral injury, other complications, laboratory values, and the etiology of neurological damage. After the injury, the presence of permanent or temporary deficit was evaluated in the early period, mortality information was also recorded.

Patients who did not wake up in the cardiac surgery intensive care unit or had signs of stroke were followed up by the neurology clinic with a pre-diagnosis of cerebral embolism. After radiological cerebral tomography and dynamic magnetic resonance imaging (for the distinction of bleeding and embolic events), the patients were either given medication or they were evaluated for thrombolytic and mechanical thrombectomy treatment by consultation with the neurologist + cardiovascular surgeon + invasive radiologist (since the last 2 years). Patients who were found to have permanent damage due to brain injury in type I patients were evaluated by the physical therapy clinic and included in physical therapy programs.

Statistical analysis

Normally distributed continuous variables, when not normally distributed, were specified as the lowest and highest value with mean ± standard deviation (SD) or median value. Categorical variables were expressed as numbers and percentages. All statistical analyzes were performed using SPSS statistical software (SPSS for Windows 15.0, Inc., Chicago, IL, USA).

RESULTS AND DISCUSSION:

RESULTS:

Coronary bypass surgeries (3845 and combined 163) constituted the majority of 5022 operations performed in cardiac surgery. Valve replacements and coronary + valve replacement were also available in this study. Hypertension was evident in 51.1% of the patients, diabetes mellitus in 43.3% and hypercholesterolemia in 79.9%. 74.7% of the operated patients were male. The mean age of our patients was 59.8 ± 6.9. Pre and intra-operative data are summarized in Tables 1 and 2, and postoperative data in Table-3. Postoperative Type I or Type II neurological damage was detected in 151 (male: 105, female: 46) of the 5022 operated patients. The mean age of these patients was 61.4 ± 8.1. Sixty-two of these patients were Type I, neurological damage patients.

In total, the percentage of neurological damage was around 3 % compared to all operated patients. Of the neurological damages divided into types; While the percentage of Type I patients was 1.3 % according to the number of all patients, it was 41.1 % compared to patients with neurological destruction. While permanent neurological damage was 1 % among all operated patients, the percentage of permanent damage was 34.4 % among all neurological patients (Table-4). As a result of the neurological evaluation and tomographic examination, it was determined that the embolic event causing ischemia in the etiology of cerebrovascular damage or complaint was responsible for the entire etiology. Bleeding was not responsible for these neurological clinical pictures in any of our patients. Mortality was present in early term 34 patients (0.7%) (Table-4).Neurological damage could not be fully evaluated in any of these 34 patients. Because the patients were lost before they woke up. Four of 177 patients who died within the first 30 days had permanent neurological damage and ventilator dependence.

Fifty-two patients with permanent neurological damage were evaluated by a neurologist and interventional radiologist, and some patients were decided to undergo thrombolytic or mechanical thrombectomy. Thrombolytic therapy was given to 5 patients within the first 4 hours and hemiplegic complaints turned into hemiparesis in 2 patients who were given. No patients exhibited re-bleeding into the pericardial space or wound bleeding. In addition, it was decided to perform mechanical thrombectomy (with merci retriever system) in 3 patients, but there was no improvement in the patients. The results of this application and medication are shown in Table-4.

Neurological patients with Type II complaints (89 patients) were 1.7 % according to the total number of patients and 58.9 % compared to all neurological damaged patients. Type II injury clinic consisted of confusion, agitation, memory impairment, seizures without signs of focal damage, and often neuro-cognitive dysfunction. They were followed up by the neurology clinic from the postoperative period. No structural and embolic damage was observed in the cerebral tissues of the patients in the radiological examination (computed tomography or magnetic resonance imaging). Medical treatments appropriate for patients were initiated.

DISCUSSION:

Generally, the percentage of Type I neurological damage was 1.3%, and Type II was 1.7% in patients who underwent cardiac surgery. In some series in the literature, this rate has been shown

in ranges such as 0.5% and 1.6%. Our results are consistent with literature studies. However, since aortic and carotid surgery was not performed in our study, our neurological damage rates may are low. In addition, our patients consisted of less complicated patient groups. However, in some studies, these neurological damage rates were found to be much higher (5.4 %). In these presented studies, the variety of cardiac pathology patients had was greater (2,3,4-8) and cardiac surgery consisted of a large patient population of cardiac surgery such as low cardiac output patient surgery or redo cardiac surgery. Some of his patients were high-risk embolic patients with left ventricular assist devices. Therefore, neurological damage results between 1-5 % can be evaluated as normal and is an acceptable rate. Thousands of cardiac surgery cases are performed a day around the world. However, neurological clinical damage that may occur after surgery can turn this costly and tedious surgery into a disaster for patients and physicians. While the patient regains heart health, he may have to spend his next life in a hemiplegic state. Therefore, the goal after cardiac surgery should always be to provide a better quality of life with fewer complications. The causes of cerebrovascular injury, the most important cause of morbidity, should be determined before the procedure and patients should be evaluated in detail. Conditions that contribute to peri-operative neurological damage can be listed as patient-related factors, intra-operative and postoperative factors. Patient-related factors include being over 60 years of age, female gender; preoperative poor left ventricular function, critical preoperative condition, diabetes, hypercholesterolemia, hypertension, previous stroke (3). These predisposing factors, which are preoperatively owned in our patients, seem to be the cause of a cerebral injury that may occur after the operation. Because when we look at our patient population, the high average age of patients, a certain high number of female patients, high hypertension and diabetic patients support this situation. Also, conditions such as low cardiac functions, previous myocardial infarctions, heart valve disease, emergency surgical conditions, and pulmonary parenchyma diseases are effective in the development of neurological damage as risk factors.

The mean preoperative hemoglobin values of the patients were 14.2 g/dL. The presence of preoperative anemia has been suggested as an independent risk factor for the development of stroke after cardiac surgery (9). No significant anemia was found in most of our patient group. Preoperative minimum and maximum values of hemoglobin were 9.7-16.4 g/dL. Hemoglobin value was below 10 g/dL in some of our patients who came from rural areas and had not had a hematological examination before, but these were not seriously anemic values. The lowest

hematocrit values observed during cardiopulmonary bypass, i.e. the degree of hemodilution, are also thought to contribute to postoperative stroke (9,10). Accordingly, while the risk of stroke increases with hematocrit values below 21 and below 18 g/dL, this probability is 3 times higher. The average hematocrit value observed during cardiopulmonary bypass in our patients was found to be 26.4±3.2, and these data were evaluated in accordance with the information in the literature. Thus, we provided enough hemodilution in our patients and reduced the risk of stroke.

Considering intra-operative risk factors, even if it varies according to the type of surgery, the most important factors are the structure of the ascending aorta and the effect of plaques in the carotid system. Especially ascending aortic surgery, additional carotid lesions (ulcerated or occluded plates) and multiple vascular procedures increase the risk of intra-operative stroke. Vascular factors such as cross-clamping the ascending aorta onto an atheroma plate and a carotid stenosis with advanced stenosis in the preoperative period may facilitate embolization or ischemic changes. Also, microembolization, hypoperfusion, temperature and hematocrit variables that will occur from the system during cardiopulmonary bypass are considered among intra-operative risk factors (3,8). Although our patients did not include patient groups with ascending aorta and carotid system disease, it is not uncommon to see emboli associated with millimetric plaques during cross-clamping or in coronary surgery proximal anastomoses (aortic-saphenous vein anastomosis) due to ascending aortic manipulations. Intraoperative monitoring by transesophageal or epiaortic ultrasound is useful for identifying mobile atheromatous plaques and selecting appropriate aortic manipulations (11).In our hospital, routine epiaortic ultrasound is not applied, manual palpation during surgery is the evaluation procedure performed with the ascending aortic angularity. All of our Type I injuries that we encountered in the postoperative period were found to be neurological damage due to embolic origin, so it is clear that embolic events play a role in the etiology. In addition to atheroma plaques, methods such as axillary artery cannulation, single clamp technique, and carbon dioxide insufflation are recommended as a precaution against other microembolisms and air-lipid emboli (11-14). In our hospitals in our city, these methods are not used in routine applications. Since routine neuro-monitoring (electroencephalography and near-infrared spectroscopy) was not performed in terms of detecting embolism in the peri-operative period, it was predicted that the source of the events was in favor of embolism due to the ischemic lesion detected in most cases, based on the results of cerebral tomography in the postoperative period.

Hypoperfusion is another risk factor for stroke that occurs especially during cardiopulmonary bypass. In 10% of the cases, no finding in favor of ischemia or bleeding was found as a result of tomography, and radiological edema and clinical findings suggesting hypoperfusion were observed. Therefore, providing high perfusion pressures during the pump or preferring pulsatile pump flow is also effective in preventing neurological damage (15). We also kept blood pressure arterial values between 50-80 mmHg during cardiopulmonary bypass in all our patients and we used pulsatile pump current, increasing cerebral autoregulation thresholds in patients due to hypertension leads to the risk of insufficient arterial pressure and perfusion values, which are considered normal according to many people. Therefore, cerebral damage due to hypoperfusion may occur after operations that are thought to be maintained at normal values. Therefore, we tried to stay away from high blood pressure arterial values in our patients with hypertension to increase perfusion during cardiopulmonary bypass.

Perioperative glucose values are also known to be associated with stroke (1). In our clinic, patients diagnosed with diabetes are monitored orally or parenterally with antidiabetic (insulin therapy) in the peri-operative period according to their blood glucose values, and blood glucose monitoring of the patients is performed at frequent intervals daily. Accordingly, insulin treatment was required in about one-third of our patients for blood glucose regulation in the preoperative and postoperative evaluation.

Body temperature management is another important issue for neurological consequences. In addition to the claim that hypothermia has protective effects, some studies do not find a difference between hypothermia and normothermia in terms of stroke risk. The main problem is also said to have negative effects of cerebral hyperthermia due to rapid warming after hypothermia application (16,17). We often preferred moderate hypothermia (30-32 C) in our patients. We avoided rapid and sudden cooling and warming.

Thrombolytic therapy, one of the ultimate treatments in selected and appropriate stroke cases, was recommended in the 2018 Early Management Guidelines for Acute Ischemic Stroke (Stroke) Patients, thrombolytic therapy (18,19), and mechanical thrombectomy applications (20). It has been stated that thrombolytic therapy can be performed within the first 3-4.5 hours after the onset of stroke symptoms and mechanical thrombectomy within the first 6-24 hours. Thrombolytic therapy aims to restore blood flow and limit the infarct area. Improvement has been reported in

patients using urokinase, recombinant prourokinase and often rt-PA (21,22). Also, mechanical thrombectomy, which can be performed using some scores, and physical removal of the thrombus via a catheter can be successfully applied in recent years. For this purpose, products such as Photoacoustic recanalization, Ultrasound Angiojet, hydrolyser, Retriver, Merci retriver, Goose neck snare and Trispan are used in the literature (23,24).In the last 2 years, we have started to perform such invasive procedures in the postoperative period under the leadership of neurology and interventional radiology. Although our experience and number of applications were insufficient, we provided these treatments to our patients, albeit in a limited number. We applied thrombolytic therapy to 5 patients and mechanical thrombectomy to 3 patients. Hemiplegic complaints improved in 2 patients who were given thrombolytic therapy. However, we could not achieve success in our mechanical thrombectomy applications.

Cardiac surgery is often cited as a risk factor for postoperative cognitive dysfunction (Type II neurological damage). These Type II injuries can usually present with confusion, agitation, memory impairment, seizures without signs of focal damage, and neuro-cognitive dysfunction, and it has generally been found to be between 40-70% in the literature (1,2,4). Martin et al. risk factors of possible postoperative Type II damage in cardiac surgery, preoperative (age, education level and previous diseases), intra-operative (micro-embolism, inflammation, hyperglycemia, duration of the procedure, use of cardiopulmonary bypass, mean arterial pressure and hyperthermia), and postoperative (hypoxia and body temperature) factors divided into 3 groups (25). It has been reported that up to 30% of the patients who are candidates for coronary artery bypass grafting surgery have preoperative cognitive dysfunction (26). Hudetz et al. in a prospective study compared patients who underwent CABG surgery and those who underwent valve surgery (valve + CABG or only valve surgery) in terms of postoperative delirium and postoperative cognitive dysfunction, resulting in delirium (27), and they found that postoperative cognitive dysfunction was more common in patients who had valve surgery in the early postoperative period.Our patients had signs of Type II neurological damage, except for Type I severe neurological damage. However, none of these patients had radiological signs of brain damage. Also, no differences regarding Type II injury could be determined in terms of coronary or valve surgery. With the medication given by the neurology clinic, clinical improvement or complete recovery was observed in all patients.

CONCLUSION:

Neurological complications after cardiac surgery are an important problem that increases morbidity and mortality. It can create a difficult and traumatic process for both hospitalization and later lives of patients. Type II mild neurological findings may improve within days and months, while Type II injuries may cause permanent neurological findings. We think that intra-operative thrombo-embolic factors rather than preoperative features and comorbidities may have played a role in the etiology of neurological damage in our patients.

Limitations

There are some limitations in this study. In our study in which we evaluated postoperative neurological damage, we did not include ascending aortic surgery cases in cardiac surgery cases. Because ascending aortic surgery is a form of surgery that can often cause comorbid and embolic problems. Since our patients do not include this group, our rates of Type I neurological damage due to embolic damage may be low. Secondly, the lack of neuro-monitoring devices (EEG, NISR) that allow the preoperative monitoring of neurological functions in our hospital may have prevented patients from being diagnosed earlier in neurological terms. This will delay thrombolytic and mechanical thrombectomy procedures in the early postoperative period and increase the percentage of permanent damage. In addition, thrombolytic and mechanical thrombectomy treatments that have been implemented in our hospital in the last 2 years cannot be performed frequently due to inexperience and insufficient equipment, which may have increased the rates of permanent neurological damage.

Declarations

Consent

Written informed consent was obtained from the patient's legal guardians/parents for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil

Conflict of interest

The authors declare no conflict of interest.

Funding

None

Ethical approval

Not applicable

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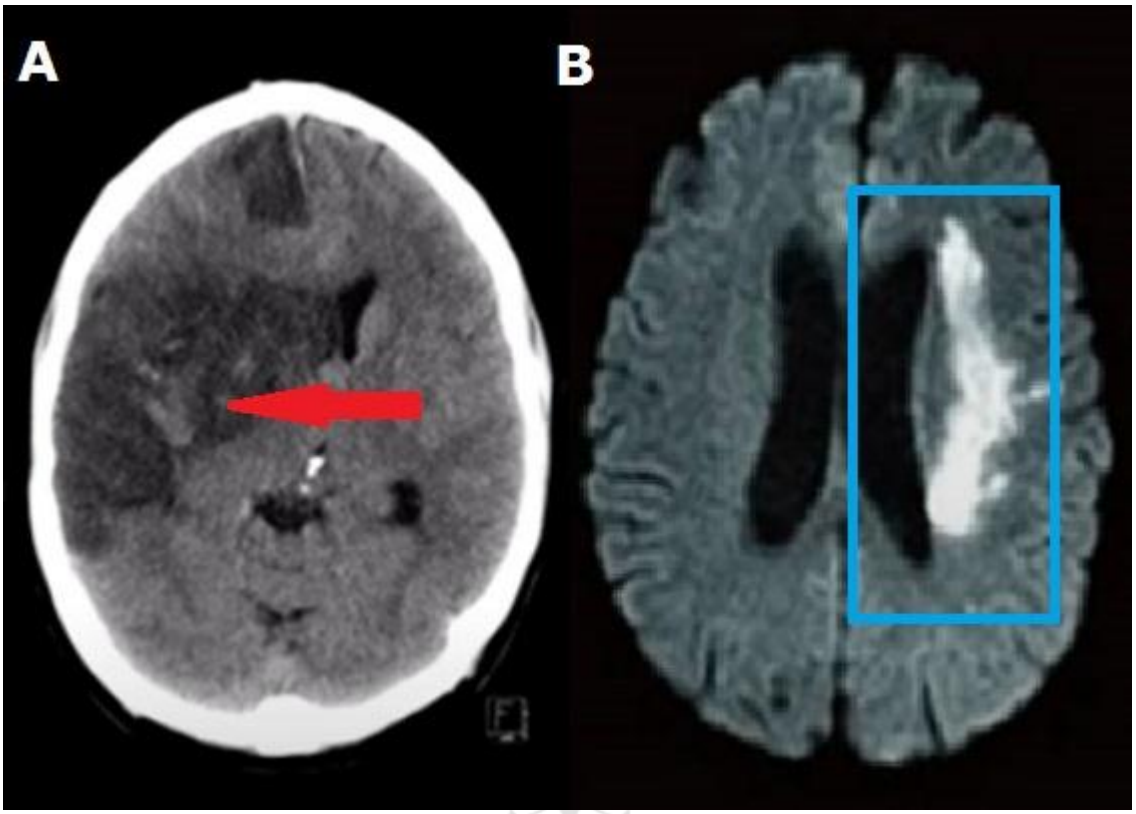

Figure No. 1: Brain computed tomography of one of our patients who underwent cardiac surgery; diffuse embolic infarct view in the right hemisphere (red arrow) **(A)**; Ischemic changes around the left ventricle in the dynamic magnetic resonance image of another operated patient (blue frame) (B).

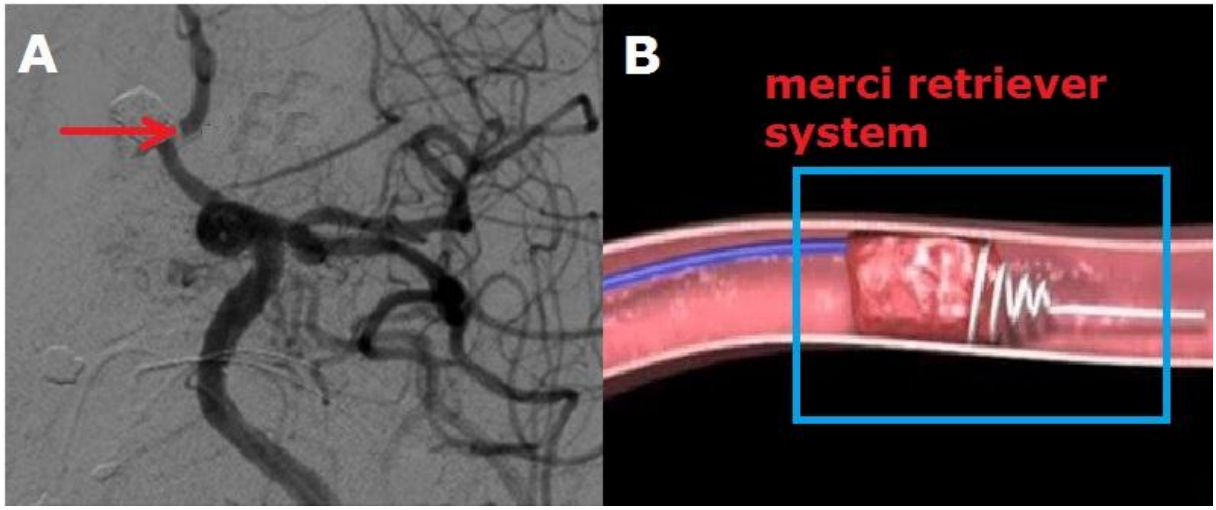

Figure No. 2: The appearance of the occluded cerebral artery on the cerebral DSA image in a patient with mechanical thrombectomy indication (red arrow) **(A);** Figure showing the working principle of the merci retriever system in the picture taken from the literature (blue frame) **(B)**

Table No. 1: Preoperative demographic characteristics

Parameters	Mean ± SD or (N)	Percent (%)
Total number of patients	5022	
Age (years)	59.8 ± 6.9	
Men (within total patients)	3755	74.7
Hypertension	2566	51.1
Diabetes Mellitus	2177	43.3
COPD	712	14.2
PVD	544	10.8
Carotid artery disease that does not require invasive or surgical intervention	1788	35.6
Hypercholesterolemia	4012	79.9
Stable angina	3256	64.8
Unstable angina	589	11.7
Only coronary arterial surgery (CABG)	3845	76.5
Only mitral valve replacement	592	12.2

Only aortic valve replacement	382	8
Double mechanical valve replacement	40	7.9
CABG + valve replacement	163	3.2
EF < 40	54	1.1
LVEF (means)	53.2±5.9	
Atrial fibrillation	1088	21.6
NYHA Class I and II	4045	80.5
Preoperative PTCA	433	8.6
Preoperative IABP	655	13
Angiography time (days)	11.2±9.2	
Previous MI	1209	24
Emergency surgery	302	6
Hemoglobin levels (g/dL)	14.2±1.4	
Hematocrit (%)	26.4±9.2	
Glucose levels (mg/dL)	217.8±28.1	
WBC count ($x10^3/\mu L$)	8.6±5.5	
Creatinine levels (mg/dL)	0.9±0.27	

COPD: Chronic obstructive pulmonary disease; PVD: Peripheral vascular disease; CABG: Coronary arterial bypass grafting; PTCA: Per-cutaneous trans-luminal coronary angioplasty; IABP: Intra-aortic balloon counter-pulsation; MI: Myocardial infarction; LVEF: Left ventricle ejection fraction; NYHA: New York Heart Association; WBC: White blood cell.

Table No. 2: Intra-operative surgical data

Variables	**Mean ± SD or (N)**	**Percent (%)**
CPB time (min)	58 ± 14	
XCL time (min)	41 ± 12	
Operation time (min)	243.8±54.8	
LAD bypass	4008	79.8
Diagonal bypass	3677	73.2
Cx bypass	2890	57.5
RCA bypass	2745	54.6
ITA usage	3711	73.9
Cumulative regional ischemic times (min)	7.1±4.2	
Hemodynamic data (in CPB) Mean arterial pressure (mmHg) Mean heart rate (min)	 45±23 77±16	
Coronary endarterectomy	1087	21.6
Retrograde cardioplegia usage	1877	37.3
Number of distal anastomosis	3.7 ± 0.8	
Only coronary surgery	3845	76.5
Mitral mechanical valve replacement	545	10.8
Mitral bioprosthesis valve replacement	47	1.9
Aortic mechanical valve replacement	357	7.1
Aortic bioprosthesis valve replacement	25	1.5
Total double valve replacement	40	7.9
Coronary surgery + mitral valve replacement	82	16.3
Coronary surgery + aortic valve replacement	81	16.1
Per-operative Ultra-filtration	1788	35.6
Urine output (in CPB)	799.8±329.1	

CPB: Cardiopulmonary bypass; XCL: Aortic cross-clamping; LAD: Left anterior descending coronary artery; Cx: Circumflex coronary artery; RCA: Right coronary artery, ITA: Internal thoracic artery.

Table No. 3: Postoperative parameters

Variables	**Mean ± SD or (N)**	**Percent (%)**
Per-operative AMI	55	1.1
New IABP insertion	239	4.7
Duration of inotropic support (days)	7.4 ± 3.3	
LCOS	177	3.5
New Atrial fibrillation	2199	43.8
Postoperative renal dysfunction (Cr>1,5 mg/dl)	679	13.5
Hemoglobine (g/dL) Blood urea nitrogen (mg/dL) Creatinine levels (mg/dL) Glucose levels (mg/dL)	7.92±2.16 49.5±28.7 1.24±1.1 191.5±48.7	
Post-operative hemodialysis	71	1.4
Pulmonary complications	529	10.5
Gastrointestinal complications	322	6.4
Sternal dehiscence	94	1.8
ICU stay	3.2 ± 2.1	
Hospital stay	8.5 ± 3.7	
Time to extubation (h)	39.2 ± 18	
Infectious complications	73	1.4
DSWI	64	1.2
Surgical revision for bleeding	1456	28.9
Postoperative bleeding > 1000 mL	588	11.7

AMI: Acute myocardial infarction; LCOS: Low cardiac output syndrome; IABP: intra-aortic balloon pump; ICU: Intensive care unit; DSWI: Deep sternal wound infection; LVEF: left ventricle ejection fraction; LVEDD: Left ventricle end-diastolic diameter.

Table-4. Postoperative data related to cerebro-vascular events and mortality

Cerebro-vascular events	**N (m/n)**	**%**
Neurological damage	151 (105/46)	3.0
Age (years)	61.4 ± 8.1	
Type I	62 (48/14)	1.3
Type II	89 (69/20)	1.7
Degree of cerebro-vascular event		
Permanent damage (-), full recovery	89	1.7
Permanent damage (+)	52	1.0
First temporary damage, then healing	10	0.2
Etiology of cerebro-vascular injury		
Ischemic	62	1.2
Bleeding	0	0
Early mortality (48 hours)	34	0.7
Hospital mortality (within 30 days)	177	3.5
Thrombolytic treatment (with t-PA)	5	0.1
partial recovery	2	0.04
un-answered (no improvement)	3	0.05
Mechanical thrombectomy (Merci retriever system)	3	0.05
Un-answered (no improvement)	3	0.05