Jurnal
Kardiologi Indonesia
J Kardiol Indones. 2010;31:196-205
ISSN 0126/3773

Laporan Kasus

Percutaneous Intra Arterial Thrombolysis on Peri-coronary Angiography Ischemic Stroke: Initial Experience in National Cardiac Center Harapan Kita

Muhammad Fadil, Dicky Hanafy, Irmalita, Doni Firman, Yoga Yuniadi

Background. Ischemic stroke is a rare but well-known complication of cardiac-catheter interventions attributable mostly to embolism. Peri-coronary angiography stroke, represents a unique opportunity for immediate stroke interventional therapy with intravenous, catheter based intra-arterial thrombolysis (IAT) or combined therapy using recombinant tissue plasminogen activator (rtPA). Intra-arterial thrombolysis is an option for treatment of selected patients who have major stroke of < 6 hours duration due to occlusion of the *Media Cerebral artery* (MCA) and who are not otherwise candidates for intravenous rtPA (Class I, Level of Evidence B, AHA guidelines).
Objective. To review the first experience of *percutaneous intra-arterial thrombolysis* (PIAT) as a management of acute ischemic stroke due to complication of coronary angiography procedure.
Summary. We present 3 cases of acute ischemic stroke which happened during coronary angiography procedures. All of them (NIHSS 30, 20 and 34) were treated immediately with intra-arterial thrombolysis (IAT) recombinant tissue Plasminogen Activator (rtPA) and had a very good outcome with no residual of stroke (NIHSS 0). Although it has been stated clearly in the AHA guidelines for the early management of adult with ischemic stroke, up until now the procedure of IAT or (intra-venous thrombolysis) IVT upon acute ischemic stroke is not established yet in standard operational procedure of National Cardiovascular Center Harapan Kita (NCCHK) Jakarta, which perhaps makes the physicians hesitate on performing these procedures due to legal aspects, while in the other hand the risks of acute ischemic stroke remain the potential threats on every single coronary angiography or any intensive catheter based procedures.

Department of Cardiology and Vascular Medicine, Faculty of Medicine,
University of Indonesia, and
National Cardiovascular Center Harapan Kita, Jakarta

(J Kardiol Indones. 2010;31:196-205)

Keywords: Percutaneous Intra-Arterial Thrombolysis, Peri-coronary Angiography Ischemic Stroke

Jurnal
Kardiologi Indonesia
J Kardiol Indones. 2010;31:196-205
ISSN 0126/3773

Case Report

Trombolisis Intra-arterial Perkutan pada Stroke Iskemik Peri-angiografi Koroner: Pengalaman Awal di Pusat Jantung Nasional Harapan Kita

Muhammad Fadil, Dicky Hanafy, Irmalita, Doni Firman, Yoga Yuniadi

Latarbelakang: Sekalipun jarang terjadi stroke iskemik merupakan komplikasi intervensi koroner yang sangat dikenal yang terutama terjadi akibat emboli. Stoke iskemik peri-angiografi ini memberikan kesempatan untuk dilakukanya trombolisis intravena, intraarterial memakai kateter atau kombinasi keduanya dengan menggunakan recombinant tissue plasminogen activator (rtPA). Trombolisis intraarterial (TIA) merupakan pilihan terapi stroke iskemik mayor dengan awitan < 6 jam akibat sumbatan pada arteri serebri media (ASM) yang bukan merupakan kandidat rtPA intravena (Kelas I, level bukti B pada panduan AHA). Tujuan: Ulasan terhadap TIA yang dilakukan pada stroke iskemik peri-angiografi. Ringkasan: TIA rtPA dilakukan pada 3 kasus stroke iskemik peri-angiografi (NIHSS 30, 20 dan 34) dengan hasil yang sangat memuaskan tanpa gejala sisa (NIHSS 0). Sekalipun sudah merupakan pilihan terapi yang dianjurkan pada panduan AHA tetapi TIA ataupun trombolisis intravena belum merupakan standar terapi di PJNHK. Laporan kasus ini diharapkan memberikan wawasan baru bagi tatalaksana stroke iskemik khususnya akibat komplikasi tindakan intervensi koroner.

(J Kardiol Indones. 2010;31:196-205)

Kata kunci: Trombolisis intraarterial perkutan, stroke iskemik peri-angiografi

Coronary angiography is associated with a peri-procedural stroke rate of 0.3%[1]. This risk is higher with interventional versus diagnostic coronary angiography. Peri-coronary angiography stroke may be related to air embolism, thrombus formation at the catheter tip, or thrombo-embolic material dislodged by the catheters or from the aortic arc. Fluoroscopic time and severity of coronary artery disease (CAD) may increase the risk of peri-coronary angiography stroke.[2]

Peri-coronary angiography stroke represents a unique opportunity for immediate stroke interventional therapy with intra-venous, catheter based intra-arterial thrombolysis (IAT) or combined therapy using recombinant tissue plasminogen activator (rtPA), prourokinase, or urokinase because of very short time to-treatment and readily available resources.[3] Intra-arterial thrombolysis is an option for treatment of selected patients who have major stroke of < 6 hours

Alamat Korespondensi:
dr. Muhammad Fadil, Departemen Kardiologi dan Kedokteran Vaskular, Fakultas Kedokteran Universitas Indonesia. E-mail: mfadil98@yahoo.com

duration due to occlusions of the MCA and who are not otherwise candidates for intravenous rtPA (Class I, Level of Evidence B). Treatment requires the patient to be at an experienced stroke center with immediate access to cerebral angiography and qualified interventionalists. Facilities are encouraged to define criteria to credential individuals who can perform intra-arterial thrombolysis (Class I, Level of Evidence C).[8]

In National Cardiac Center Harapan Kita (NCCHK) Jakarta Indonesia, last year we had 4 cases of acute ischemic stroke due to coronary angiography procedure, and this year we already have had 4 cases[4] and the last 3 cases were treated by intra-arterial thrombolysis with the good outcome results. Intra-arterial therapy is increasingly used in the treatment of acute stroke either as the primary modality for patients presenting 3 to 8 hours of symptoms onset or as an adjuvant measure in patients treated with intravenous tissue plasminogen activator (IV tPA) who do not improve in a timely fashion. Primary IAT has been shown to be improved clinical outcome when administered 3 to 6 hours from symptom onset using intra-arterial thrombolytics[5] and is approved up to 8 hours using mechanical clot retrieval (Merci Retriever) or suction thrombectomy (Penumbra System). [6]

According to SYNTHESIS trial, rapid initiation of IAT is a safe and feasible alternative to IVT in acute ischemic stroke.[7] AHA Guidelines 2007 for the early management of Adults with ischemic stroke stated that intravenous rtPA is recommended for selected patients who may be treated within 3 hours of onset of ischemic stroke (Class I, Level of Evidence A) and intra-arterial thrombolysis is an option for treatment of selected patients who have major stroke of < 6 hours duration due to occlusions of the Middle Cerebral Artery(MCA) and who are not otherwise candidates for intravenous rtPA (Class I, Level of Evidence B).[8] Furthermore, because recent advances in endovascular treatment techniques for stroke patients, nowadays it is possible to perform percutaneous transluminal angioplasty (PTA) of the intracranial artery as an adjuvant to intra-arterial thrombolysis.[9]

Case Illustration 1

Mr. DR, 74 years old came to National Cardiovascular Center Harapan Kita Jakarta (NCCHK) to have elective coronary angiography due to stable angina pectoris CCS II. He was also diagnosed as post CABG 1996 and 2004 with hypertension stage II. He presented chest pain with moderate activity and diminish with rest or sublingual nitrate, with duration about 5-10 minutes, the pain did not radiate and there were no diaphoretic, nausea nor vomiting. He had been suffering these symptoms for last two months, with risk factors of hypertension and dyslipidemia. The treadmill test was performed with only 02 minutes 11 second achieved.

Physical examination revealed mild ill, blood pressure was 150/80 mmHg, heart rate was 52 bpm, respiratory rate 20 x/minutes, and body temperature was 36,5^{o} C. Auscultation revealed normal heart sound, normal vesicular sound with no rales and wheezing from lung examination, and other examinations was within normal limit.

The electrocardiogram showed sinus rhythm, QRS rate 50 bpm, QRS axis (-15), normal P wave, PR interval 20 msec, QRS duration 6 msec, Q pathologic at III, aVF and slight T inverted at I, aVL, V5 and V6. The laboratory finding revealed hemoglobin was 12.6 g/dl, Hematokrit 39 vol%, WBC 8620/ul, ureum 65 mg/dl and creatinin level was 1.8 mg/dl.

Chest X-ray revealed cardiac enlargement with cardio-thoracic ratio 55% with the prominent aortic segment and normal pulmonal segment, flattening of cardiac waist, downward apex and no congestion nor infiltrate. Echocardiography showed EDD 56mm, ESD 42mm, with the ejection fraction 48%, hypo-kinetic at posterior segment, normal valves and diastolic dysfunction with abnormal relaxation. His last medications were ISDN 2 x 20 mg, bisoprolol 1 x 2.5 mg, cardace 1 x 5 mg, amiodarone 1 x 200 mg, ascardia 1 x 80 mg, HCT 1 x 12.5 mg, simvastatin 1 x 10 mg, micardis 1 x 80 mg.

The coronary angiography was performed upon this patient with the result, LAD proximal stenosis 95%, LCX proximal and RCA proximal, both were total occlusion, LRA-LAD graft was patent. The catheter was failed to advance to SVG-RCA and SVG-LCx grafts. After coronary angiography procedure carried out, the patient felt fatique and unable to speak clearly. The right extremities were unable to move, the National Institute of Health Stroke Scale (NIHSS) was 30 (severe stroke). Then the patient was diagnosed as CAD 3 VD with graft failure at SVG-RCA and SVG-LCx with the complication of cerebrovascular disease stroke non hemorraghe (CVD SNH).

Patient was planned to perform cerebral percutaneous intra-arterial thrombolysis (PIAT) with r-TPA. MP Catheter was advanced to Left Internal Carotid Artery and cerebral angiography was carried out, showed total occlusion on Media Cerebral Artery (MCA). Then, the rTPA (actyles) was injected bolus 6 mg through the Microcatheter which placed upon MCA, angiography evaluation showed the occlusion begin to be opened, followed with actyles drip 0.9 mg/kgBW within an hour intra-catheter. Within 30 minutes rTPA drip, there was bleeding on the gum, angiography evaluation showed the occlusion was opened with good flow, the drip was stopped. Neurological examination showed patient was in good orientation, both extremities moved equally, the NIHSS was 3 (mild stroke) and moved to CVC unit.

In CVC unit patient was fully alert, BP was 208/88 mmHg, HR 51 bpm, with mild neurological deficit. BP was stable under intravenous perdipin 0.3 mg/kgBW/minutes, MAP target was 100. Brain CT scan was performed with no detection of intracranial hemorraghe. Neurological expertise showed minimal neurological deficit (mild stroke) and patient moved to adult ward on the next 3 days. Brain CT scan after 24 hours of PIAT procedures showed no detection of intracranial hemorraghe. Now patient is stable at adult ward with NIHSS 0.

Case Illustration 2

Mr. MTZ, 72 years old came to National Cardiovascular Center Harapan Kita Jakarta (NCCHK) ambulatory to have elective coronary angiography standby PTCA due to stable angina pectoris CCS II. He was also diagnosed as post CABG 2001 with hypertension stage I and type 2 DM. He presented chest pain with moderate activity and diminish with rest or sublingual nitrate, with duration about 5-10 minutes, the pain did not radiate and there were no diaphoretic, nausea nor vomiting. He had been suffering these symptoms for one last month, with risk factors of hypertension, diabetes and dyslipidemia. Cardiac perfusion scanning was performed upon this patient and the result showed

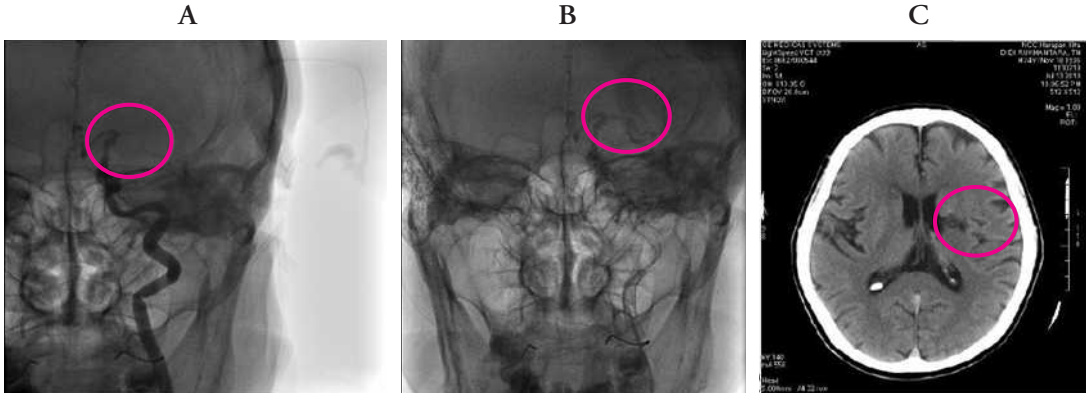

Figure1. Case illustration 1: **A.** Before PIAT **B.** After PIAT **C.** Brain CT showed ischemic stroke

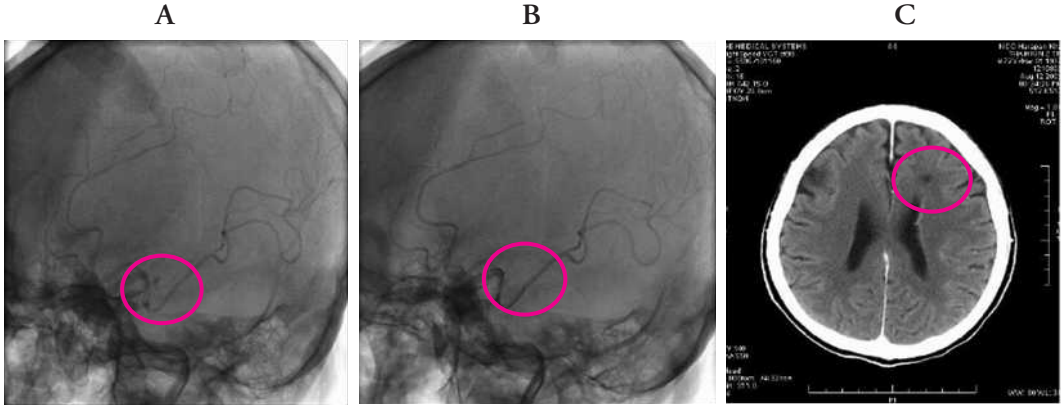

Figure 2. Case illustration 2: A. Before PIAT B. After PIAT C. Brain CT showed ischemic stroke

parsial defect at lateral wall as inducible ischemia.

Physical examination revealed mild ill, blood pressure was 134/70 mmHg, heart rate was 60 bpm, respiratory rate 18 x/minutes, and body temperature was 36,4° C. Auscultation revealed normal heart sound, normal vesicular sound with no rales and wheezing from lung examination, and other examinations was within normal limit.

The electrocardiogram showed sinus rhythm, QRS rate 50 bpm, QRS axis (-15), normal P wave, PR interval 12 msec, QRS duration 4 msec, Q pathologic at III and aVF, slight T inverted at I and aVL. The laboratory finding revealed hemoglobin was 15.3 g/dl, Hematokrit 45 vol%, WBC 12900/ul, ureum 43 mg/dl and creatinin level was 0.9 mg/dl.

Chest X-ray revealed cardiac enlargement with cardio-thoracic ratio 60% with the prominent aortic segment and normal pulmonal segment, flattening of cardiac waist, downward apex and no congestion nor infiltrate. Echocardiography showed EDD 41mm, ESD 27mm, with the ejection fraction 65%, hypokinetic at posterior segment, normal valves and diastolic dysfunction with abnormal relaxation. His last medications were tanapress 1 x 10 mg, maintate 1 x 5 mg, vascardin 3 x 10 mg, thromboaspilet 1 x 80 mg, glucophage 2 x 500 mg, alganax 1 x 0.25 mg.

The coronary angiography was performed upon this patient with the result, LAD proximal stenosis 90%, diffuse stenosis proximal-mid 70-90%. LCX subtotal occlusion at distal part and OM 2, OM1 stenosis 80% at proximal-mid, RCA collateral to distal part LCx. RCA total occlusion at proximal with bridging collateral to distal part. Both SVG-D1 graft and SVG-PDA graft were patents, LIMA-LAD graft was total occlusion on distal incersion. The catheter was failed to advance to SVG-OM graft. After coronary angiography procedure carried out, the patient complaint of tiredness and unable to speak clearly. The right extremities were unable to move and the communication was inadequate, the National Institute of Health Stroke Scale (NIHSS) was 28 (severe stroke).

MSCT brain was performed and showed ischemic stroke, no detection of intracranial hemorraghe. Then the patient was diagnosed as CAD 3 VD with graft failure at LIMA-LAD, SVG-OM with the complication of CVD SNH.

Patient was planned to perform cerebral percutaneous intra-arterial thrombolysis (PIAT) with r-TPA. JR Catheter was advanced to Left Internal Carotid Artery and cerebral angiography was carried out, showed thrombus with stenosis Left Media Cerebral Artery (MCA). Then, the rTPA (actyles) was injected bolus 5 mg through the Microcatheter which placed upon MCA, angiography evaluation showed the occlusion begin to be opened, followed with actyles drip 10 mg within 30 minutes intra-catheter. Angiography evaluation showed thrombus was reduced. The rTPA drip was continued 10 mg within 30 minutes. Angiography evaluation showed the occlusion was opened with good flow, the drip was stopped. Integrillin was given followed with 24 hours integrillin drip. Neurological examination showed patient was alert, in good orientation, mild dysarthria, partial facial weakness, both extremities moved equally, the NIHSS was 6 (mild stroke) and moved to CVC unit.

In CVC unit patient was fully alert, BP was 162/72 mmHg, HR 86 bpm, with mild neurological deficit. BP was stable under oral anti-hypertension, MAP target was 100. Neurological expertise showed minimal neurological deficit (mild stroke) and patient moved to adult ward on the next 3 days. Patient had discharged and controlled regularly to policlinic with NIHSS 0

Case Illustration 3

Mrs.EJ, 70 years old came to National Cardiovascular Center Harapan Kita Jakarta (NCCHK) to have elective coronary angiography due to stable angina pectoris CCS II. He was also diagnosed as CHF Fc. II ec Old Inferior MCI. She presented chest pain with moderate activity and diminish with rest or sublingual nitrate, with duration about 5-10 minutes, the pain did not radiate and there were no diaphoretic, nausea nor vomiting. He had been suffering these symptoms for last 2 months, with risk factors of hypertension and menopause. She had heart attack 4 months ago and hospitalized at Kuningan Hospital.

Physical examination revealed mild ill, blood pressure was 140/70 mmHg, heart rate was 85 bpm, respiratory rate 20 x/minutes, and body temperature was 36,4° C. Auscultation revealed normal heart sound, normal vesicular sound with no rales and wheezing from lung examination, and other examinations was within normal limit.

The electrocardiogram showed sinus rhythm, QRS rate 75 bpm, QRS axis (-30), normal P wave, PR interval 20 msec, QRS duration 4 msec, Q pathologic at II, III and aVF, T inverted at I, aVL, V3-V6. The

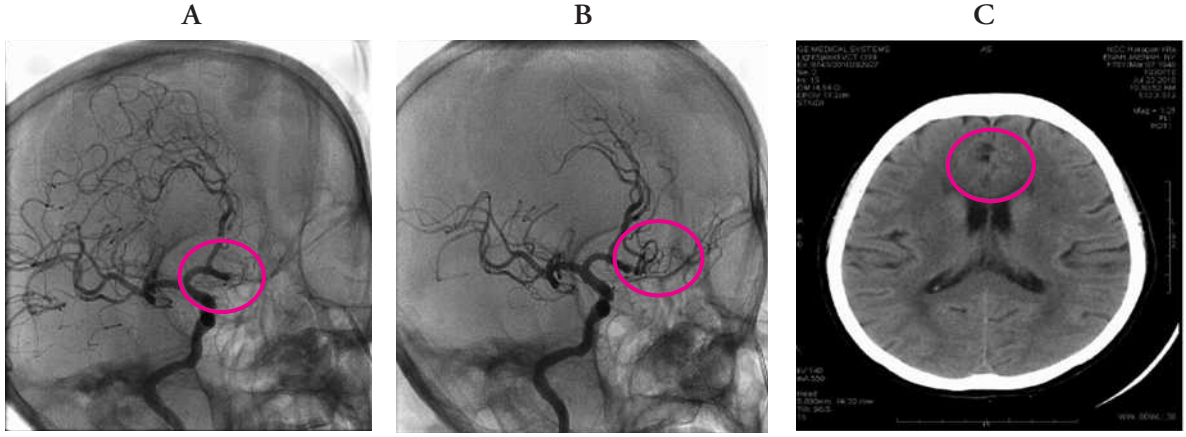

Figure 3. Case illustration 3: **A**. Before PIAT **B**. After PIAT **C**. Brain CT showed ischemic stroke

laboratory finding revealed hemoglobin was 11.2 g/dl, Hematokrit 34 vol%, WBC 7000/ul, ureum 14 mg/dl and creatinin level was 0,9 mg/dl.

Chest X-ray revealed cardiac enlargement with cardio-thoracic ratio 50%, aortic segment and pulmonal segment were normal, flattening of cardiac waist, downward apex and no congestion nor infiltrate. Echocardiography showed EDD 54mm, ESD 41mm, with the ejection fraction 45%, global normokinetic, normal valves and diastolic dysfunction with abnormal relaxation. His last medications were vaclo 1 x 75 mg, ISDN 3 x 10 mg, Hyperil 1 x 5 mg, cardioaspirin 1 x 100 mg, maintate 1 x 2.5 mg.

The coronary angiography was performed upon this patient with the result, LM non significant stenosis at distal, LAD distal stenosis 80% after D2, stenosis 70% at osteal D1 and stenosis 60-70% at osteal D2, distal goes to RCA. LCX was normal. RCA mid long-stenosis 95% with severe calsification.

After coronary angiography procedure carried out, the patient complaint of tiredness unable to open both eyes. The consciousness level was decreasing and started to snore. The National Institute of Health Stroke Scale (NIHSS) was 34 (severe stroke). MSCT brain was performed and showed ischemic stroke, no detection of intracranial hemorraghe. Then the patient was diagnosed as CHF Fc II CAD2 VD with the complication of CVD SNH.

Patient was planned to perform cerebral percutaneous intra-arterial thrombolysis (PIAT) with r-TPA. JR Catheter was advanced to Right Internal Carotid Artery and cerebral angiography was carried out, showed stenosis with slow flow on Right Media Cerebral Artery (MCA) and no flow to Right Anterior Cerebral Artery. Then, the rTPA (actyles) was injected bolus 3.5 mg through the Microcatheter which placed upon MCA, angiography evaluation showed the occlusion begin to be opened, followed with actyles drip 31.5 mg within 60 minutes intra-catheter. Angiography evaluation showed stenosis was opened with good flow to Right Anterior Cerebral Artery. Neurological examination showed patient was somnolen, dysarthria, partial facial weakness, the strength of left extremities were decreased, the NIHSS was 20 (severe stroke) and moved to CVC unit.

In CVC unit patient on the next day patient was fully alert, BP was 113/38 mmHg, HR 56 bpm, with mild neurological deficit. BP was stable under oral anti-hypertension, MAP target was 100. Neurological expertise showed minimal neurological deficit NIHSS 6 (mild stroke) and patient moved to adult ward on the next 3 days. Patient had discharged and controlled regularly to policlinic with NIHSS 0.

Discussion

Ischemic stroke is a rare but well-known complication of cardiac or neurovascular catheter interventions attributable mostly to embolism. Possible patho-mechanisms include direct arterial injury, air embolism, thrombus formation on the catheter or guide wire, and dislocation of pre-existing atherosclerotic plaques or thrombotic material, especially from the aortic arch.[10] Those 3 cases are the real example of how ischemic stroke possibly happen during procedure of coronary angiography. The similarities of 3 cases above are; above 65 years old and post CABG except the case 3. The

complication happened while the operators were about to advance the catheter through the grafts. It gives us a very good lesson on how to be more careful and extra safe to perform any invasive procedure, and knowing exactly how to manage the complication immediately with correct standard procedure, so we do not put the patient in more harmful conditions.

Brain Imaging: Hemorraghe or Not Hemorraghe

Brain imaging remains a required component of the emergency assessment of patients with suspected stroke. Both CT and MRI are options for imaging the brain, but for most cases and at most institutions, CT remains the most practical initial brain imaging test. A physician skilled in assessing CT or MRI studies should be available to examine the initial scan. In particular, the scan should be evaluated for evidence of early signs of infarction.[11] Baseline CT findings, including the presence of ischemic changes involving more than one third of a hemisphere, have not been predictors of responses to treatment with rtPA when the agent is administered within the 3-hour treatment window. Information about multimodal CT and MRI of the brain suggests that these diagnostic studies may help in the diagnosis and treatment of patients with acute stroke.[8]

Imaging of the brain is recommended before initiating any specific therapy to treat acute ischemic stroke (Class I, Level of Evidence A).Nevertheless, data are insufficient to state that, with the exception of hemorrhage, any specific CT finding (including evidence of ischemia affecting more than one third of a cerebral hemisphere) should preclude treatment with rtPA within 3 hours of onset of stroke (Class IIb, Level of Evidence A). Emergency treatment of stroke should not be delayed in order to obtain multimodal imaging studies (Class III, Level of Evidence C).[8]

It is clear enough stated at the AHA guidelines for the early management of adults with ischemic stroke that brain imaging is mandatory before initiating any specific therapy, otherwise it will do harmful to the patient. Contrary to this statement on the case illustration 1, brain imaging was performed after the therapy instead due to limitation of time. Fortunately the patient was in good condition and was proof with no detection of intracranial hemorraghe afterward. According to the AHA guidelines, it could have been harmful to the patient.

Neurological Examination and Stroke Scale Scores

The evaluation and initial treatment of patients with stroke should be performed as a priority in the hospital. The development of an organized protocol and stroke team should speed the clinical assessment, the performance of diagnostic studies, and decisions for early management. The clinical assessment (history, general examination, and neurological examination) remains the cornerstone of the evaluation. The goals are to determine whether the patient has had a stroke and to establish potential contraindications for emergency treatment with agents such as rTPA. [8]

A stroke rating scale, such as the National Institute of Health Stroke Scale (NIHSS) (table 1), provides important information about the severity of stroke. It provides prognostic information, and the score may influence decisions about acute treatment. Some of the recommendations included in the present statement are influenced by the NIHSS. This scale can be performed with a reasonable degree of accuracy by practitioners in a broad range of specialties. Education in the nuances of NIHSS can improve the accuracy of this scale. Based on the NIHSS, all of case illustrations were presented as severe stroke with 30, 28 and 34 score respectively.

Because time is critical, a limited number of diagnostic tests are recommended (table 2). These tests should be available on a 24-hours-per-day, 7-days-per-week basis. These tests are used to screen for ischemic stroke, to exclude important alternative diagnoses (especially intra-cerebral hemorrhage), to assess for serious comorbid diseases, and to search for acute medical or neurological complications of the stroke. Although it is desirable to know the results of these tests (table 2) before giving rtPA, thrombolytic therapy should not be delayed while awaiting the results unless there is clinical suspicion of a bleeding abnormality or thrombocytopenia, or the patient has received heparin or wafarin, or use of anticoagulants is not known.[8] Regarding to these cases, all of the patients were received heparin due to catheter procedures, and by that time all the case illustrations, were only tested for blood glucose, ECG and oxygen saturation. The other tests were performed when patients had already transferred to the CVC unit.

Intra-arterial or intra-venous thrombolytic?

Based on SYNTHESIS trial, the first randomized controlled trial comparing IAT and IVT with alteplase

Table 1. National Institutes of Health Stroke Scales[8]

Tested Item	Title	Responses and Scores
1A	Level of consciousness	0—alert
		1—drowsy
		2—obtunded
		3—coma/unresponsive
1B	Orientation questions (2)	0—answers both correctly
		1—answers one correctly
		2—answers neither correctly
1C	Response to commands (2)	0—performs both tasks correctly
		1—performs one task correctly
		2—performs neither
2	Gaze	0—normal horizontal movements
		1—partial gaze palsy
		2—complete gaze palsy
3	Visual fields	0—no visual field defect
		1—partial hemianopia
		2—complete hemianopia
		3—bilateral hemianopia
4	Facial movement	0—normal
		1—minor facial weakness
		2—partial facial weakness
		3—complete unilateral palsy
5	Motor function (arm)	0—no drift
	a. Left	1—drift before 5 seconds
	b. Right	2—falls before 10 seconds
		3—no effort against gravity
		4—no movement
6	Motor function (leg)	0—no drift
	a. Left	1—drift before 5 seconds
	b. Right	2—falls before 5 seconds
		3—no effort against gravity
		4—no movement
7	Limb ataxia	0—no ataxia
		1—ataxia in 1 limb
		2—ataxia in 2 limbs
8	Sensory	0—no sensory loss
		1—mild sensory loss
		2—severe sensory loss
9	Language	0—normal
		1—mild aphasia
		2—severe aphasia
		3—mute or global aphasia
10	Articulation	0—normal
		1—mild dysarthria
		2—severe dysarthria
11	Extinction or inattention	0—absent
		1—mild (loss 1 sensory modality)
		2—severe (loss 2 modalities)

≤14 mild stroke; 15-19 moderate stroke; ≥ 20 severe stroke[12]

Table 2. Immediate Diagnostic Studies : Evaluation of aPatient with Suspected Acute Ischemic Stroke[8]

All patients
Noncontrast brain CT or brain MRI
Blood glucose
Serum electrolytes/renal function tests
ECG
Markers of cardiac ischemia
Complete blood count, including platelet count*
Prothrombin time/international normalized ratio (INR)*
Activated partial thromboplastin time*
Oxygen saturation
Selected patients
Hepatic function tests
Toxicology screen
Blood alcohol level
Pregnancy test
Arterial blood gas tests (if hypoxia is suspected)
Chest radiography (if lung disease is suspected)
Lumbar puncture (if subarachnoid hemorrhage is suspected and CT scan is negative for blood)
Electroencephalogram (if seizures are suspected)

MRI indicates magnetic resonance imaging.

*Although it is desirable to know the results of these tests before giving rtPA, thrombolytic therapy should not be delayed while awaiting the results unless (1) there is clinical suspicion of a bleeding abnormality or thrombocytopenia, (2) the patient has received heparin or warfarin, or (3) use of anticoagulants is not known.

in acute ischemic stroke, rapid initiation of IAT is a safe and feasible alternative to IVT in acute ischemic stroke.[7] The PROACT II trial showed that intra-arterial thrombolysis (IAT) is effective for treatment of acute ischaemic stroke attributable to M1 and M2 segment occlusions. Incidence of symptomatic intracranial haemorrhage (sICH) was 10%.[13] Based on Brekenfeld et.al study of 294 patients, showed that incidence of sICH after IAT is distinctly low. This result underlines the important role of IAT in the treatment of acute stroke.[14]

According to AHA guidelines for the early management of adults with acute ischemic stroke, intravenous rtPA (0.9 mg/kg, maximum dose 90 mg) is recommended for selected patients who may be treated within 3 hours of onset of ischemic stroke (Class I, Level of Evidence A). Intra-arterial thrombolysis is an option for treatment of selected patients who have major stroke of < 6 hours duration due to occlusions of the MCA and who are not otherwise candidates for intravenous rtPA (Class I, Level of Evidence B). Treatment requires the patient to be at an experienced stroke

Table 3. Characteristics of Patients With Ischemic Stroke Who Could Be Treated With rtPA8

Diagnosis of ischemic stroke causing measurable neurological deficit
The neurological signs should not be clearing spontaneously.
The neurological signs should not be minor and isolated.
Caution should be exercised in treating a patient with major deficits.
The symptoms of stroke should not be suggestive of subarachnoid hemorrhage.
Onset of symptoms <3 hours before beginning treatment
No head trauma or prior stroke in previous 3 months
No myocardial infarction in the previous 3 months
No gastrointestinal or urinary tract hemorrhage in previous 21 days
No major surgery in the previous 14 days
No arterial puncture at a noncompressible site in the previous 7 days
No history of previous intracranial hemorrhage
Blood pressure not elevated (systolic <185 mm Hg and diastolic <110 mm Hg)
No evidence of active bleeding or acute trauma (fracture) on examination
Not taking an oral anticoagulant or, if anticoagulant being taken, INR ≤1.7
If receiving heparin in previous 48 hours, aPTT must be in normal range.
Platelet count ≥100 000 mm^3
Blood glucose concentration ≥50 mg/dL (2.7 mmol/L)
No seizure with postictal residual neurological impairments
CT does not show a multilobar infarction (hypodensity >1/3 cerebral hemisphere).
The patient or family members understand the potential risks and benefits from treatment.

INR indicates international normalized ratio; aPTT, activated partial thromboplastin time.

center with immediate access to cerebral angiography and qualified interventionalists. Facilities are encouraged to define criteria to credential individuals who can perform intra-arterial thrombolysis (Class I, Level of Evidence C). Intra-arterial thrombolysis is reasonable in patients who have contraindications to use of intravenous thrombolysis, such as recent surgery (Class IIa, Level of Evidence C). The availability of intra-arterial thrombolysis should generally not preclude the intravenous administration of rtPA in otherwise eligible patients (Class III, Level of Evidence C).[8]

Three trials that mentioned above, state that IAT is preferable. Meanwhile the AHA guideline suggests us to perform IVT at the first place unless in some other criteria which make IVT become out of option or the onset more than 3 hours. But on these cases, IAT is the better option due to very short time to-treatment and readily available resources. The potential problem that had happened during that time was the absence of rtPA itself, and had to wait for another 1-2 hours to receive the drug. Fortunately, the drug could have been administered within less than 3 hours of the onset. According to AHA guideline about the characteristics of patients who could be treated with rTPA, those 3 cases are eligible candidates for rtPA therapy, but the only pitfall was not assessing the brain CT on the first place for the case 1. The rest of criteria were fulfilled.

The monitoring and follow up at the cath-lab and CVC ward upon these cases before and after procedures, were correct, according to the AHA guidelines. But there was a controversial issue which was raised at the field about the need to obtain a follow up CT scan at 24 hours after procedures before starting anticoagulants or antiplatelet agents. The neurologist thought no need for another CT scan 24 hours after procedure. To detect any changes could be done by clinical signs and symptoms, while in the other hand the cardiologist thought it was necessary to obtain clearly evidence or signs of hemorraghe. According to the AHA guideline, CT scan at 24 hours after procedure is necessary to see any evidence of intracranial hemorraghe before starting anticoagulants or antiplatelet agents. Meanwhile in our cases, the antipltelet agents and anticoagulants were given to the patient after procedure before follow up CT scan. On the follow up at the ward and by phone after discharge, both patient case 1 and 2 have a very good outcome regarding the ischemic stroke, no residual at all (NIHSS 0), unfortunately for the patient on the illustration case 3, whom medicated without IAT nor IVT he has residual neurological deficit, which impacts his quality of life.

Although it has been stated clearly on the AHA guidelines for early management of Adults with sacute ischemic stroke, up until now the procedure of IAT or IVT upon acute ischemic stroke is not established yet in standard operational procedure of National Cardiovascular Center Harapan Kita Jakarta, which perhaps makes the physicians hesitate on performing these procedures due to legal aspects, while in the other hand the risk of acute ischemic stroke remain the potential threats on every single coronary angiography or any intensive catheter based procedures.

Summary

Ischemic stroke is a rare but well-known complication of cardiac-catheter interventions attributable mostly to embolism. We presented 3 cases of acute ischemic stroke which happened during coronary angiography

procedures. All of them (NIHSS 30, 20 and 34) were treated immediately with intra-arterial thrombolysis rtPA. All of the cases had a very good outcome with no residual of stroke (NIHSS 0). Although it has been stated clearly in the AHA guidelines for the early management of adults with ischemic stroke, up until now the procedure of IAT or IVT upon acute ischemic stroke is not established yet in standard operational procedure of National Cardiovascular Center Harapan Kita (NCCHK) Jakarta, which perhaps makes the physicians hesitate on performing these procedures due to legal aspects, while in the other hand the risks of acute ischemic stroke remain the potential threats on every single coronary angiography or any intensive catheter based procedures.

References

1. Brown DL, Topol EJ. Stroke complicating percutaneous coronary revascularization. Am J Cardiol. 1993;72:1207-1209
2. Zaidat OO, Slivka AP, Mohammad Y, Graffagnino C, Smith TP, Enterline DS, Christoforidis GA, Alexander MJ, Landis DM, Suarez JI. Intra-arterial thrombolytic therapy in peri-coronary angiography ischemic stroke. Stroke. 2005;36:1083-1084.
3. Zaidat OO, Suarez JI, Santillan C, Sunshine JL, Tarr RW, Paras VH, Selman WR, Landis DM. Intra-arterial and combined intravenous and intra-arterial thrombolytic therapy for distal carotid artery occlusion. Stroke. 2002;33:1828-1833.
4. Data of medical record of the National Cardiovascular Center Harapan Kita Jakarta (unpublished)
5. Hallevi H, Barreto AD, Liebeskind DS, Morales MM, Martin-Schild SB, Abraham AT, Gadia J, Saver JL; UCLA Intra-Arterial Therapy Investigators, Grotta JC, Savitz SI. Identifying patients at high risk for poor outcome after intra-arterial therapy for acute ischemic stroke. Stroke. 2009;40:1780-1785.
6. Bose A, Henkes H, Alfke K, Reith W, Mayer TE, Berlis A, Branca V, Sit SP; Penumbra Phase 1 Stroke Trial Investigators. The penumbra system: a mechanical device for the treatment of acute stroke due to thromboembolism. AJNR Am J Neuroradiol. 2008;29: 1409-1413.
7. Ciccone A, Valvassori L, Ponzio M, Ballabio E, Gasparotti R, Sessa M. Intra-arterial or intravenous thrombolysis for acute ischemic stroke? The SYNTHESIS pilot trial. J NeuroIntervent Surg 2010;2:74-79.
8. Guidelines for the early management of adults with ischemic stroke. Circulation. 2007;115:e478-e534.
9. Ueda T, Sakaki S, Nochide I, Kumon Y, Kohno K, Ohta S. Angioplasty after intra-arterial thrombolysis for acute occlusion of intracranial artery. Stroke. 1998;29:2568-2574.
10. Arnold M, Fischer U, Schroth G, Nedeltchev K, Isenegger J, Remonda L, Windecker S, Brekenfeld C, Mattle HP. Intra-arterial thrombolysis of acute iatrogenic intracranial arterial occlusion attributable to neuroendovascular procedures or coronary angiography. Stroke. 2008;39:1491-1495.
11. Roberts HC, Dillon WP, Furlan AJ, Wechsler LR, Rowley HA, Fischbein NJ, Higashida RT, Kase C, Schulz GA, Lu Y, Firszt CM. Computed tomographic findings in patients undergoing intra-arterial thrombolysis for acute ischemic stroke due to middle cerebral artery occlusion. Stroke. 2002;33:1557-1567
12. Gönner F, Remonda L, Mattle H, Sturzenegger M, Ozdoba C, Lövblad KO, Baumgartner R, Bassetti C, Schroth G. Local intra-arterial thrombolysis in acute ischemic stroke. Stroke. 1998;29:1894-1900.
13. Furlan A, Higashida R, Wechsler L, Gent M, Rowley H, Kase C, Pessin M, Ahuja A, Callahan F, Clark WM, Silver F, Rivera F. Intra-arterial prourokinase for acute ischemic stroke. The PROACT II study: a randomized controlled trial. JAMA. 1999;282:2003-2011.
14. Brekenfeld C, Remonda L, Nedeltchev K, Arnold M, Mattle HP, Fischer U, Kappeler L, Schroth G. Symptomatic intracranial haemorrhage after intra-arterial thrombolysis in acute ischaemic stroke: assessment of 294 patients treated with urokinase. J Neurol Neurosurg Psychiatry. 2007;78:280-285.