CAROTID ARTERY STENTING IN A PATIENT WITH TRANSIENT ISCHEMIC ATTACK

Suryono
Departement of Cardiology, Faculty of Medicine,
Jember University, Dr. Soebandi Hospital, Jember

ABSTRAK

Stroke adalah penyebab kedua kematian di dunia dan 20-30% disebabkan oleh stenosis arteri karotis. Dewasa ini stenting arteri karotis adalah perawatan darurat pilihan pada stenosis arteri karotis. Teknik dan pengelolaan komplikasi telah dibakukan. Kami melaporkan seorang wanita berusia 80 tahun dengan sinkop (diagnosis neurologi adalah TIA) dan riwayat penyakit sebelumnya adalah diabetes, hipertensi, dan CAD. CT scan normal. USG Doppler menunjukkan stenosis arteri karotis kanan. Angiografi karotis kanan menunjukkan stenosis lebih dari 90%. Kami melakukan pemasangan perangkat perlindungan distal, predilation dan stenting dengan Wall stent pada arteri karotis kanan. Tidak ditemukan stenosis residual. Selama stenting terjadi bradikardia dan hipotensi akibat kompresi stent pada arteri karotis. Tidak ditemuka defisit neurologis. Selama tindak lanjut 2 bulan tidak terdapat keluhan. ***(FMI 2014;50:265-269)***

Kata kunci: *stenting karotid, TIA, penyakit arteri karotis*

ABSTRACT

Stroke is the second cause of death in the world and 20-30% were caused by carotid artery stenosis. Today carotid artery stenting is emergency treatment of choice for carotid artery stenosis. The technique and management of the complication has been standardized. We report a 80-year old woman with syncope (neurology diagnosis was TIA) and history of past illness were diabetes, hypertension, and CAD. CT scan was normal. USG Doppler showed right carotid artery stenosis. Right carotid angiography showed stenosis more than 90%. We performed distal protection device, predilation and stenting with wall stent in right carotid artery. No residual stenosis was found. During stenting there were bradicardy and hypotension due to stent compression in carotid artery. No neurologic deficit was found. In 2-month follow-up there was no complaint. ***(FMI 2014;50:265-269)***

Keywords: *carotid stenting, TIA, carotid artery disease*

Correspondence: Suryono, Departement of Cardiology, Faculty of Medicine, Jember University, Dr. Soebandi Hospital, Jember. Email: suryonocardio@yahoo.com

INTRODUCTION

Carotid artery stenosis remains a public health problem, as it is associated with increased risk of ischemic stroke and embolic stroke. One report states that no incidents of ischemic stroke increased with increasing age, which is 33% before the age of 45 years and 80% after 50 years, while the cause of the whole case is 20% to 30% due to carotid artery (Boosser & Mas 1988). Stenosis of carotid artery can be found by chance, in these patients it is usually asymptomatic and found along with other vascular diseases, such as stenosis at coronary arteries, lower extremities and renal artery. In the patients the symptomatic clinical presentation may include Transient Ischemic Attacks (TIA) or ischemic stroke due to heavy subtotal occlusion/total occlusion in one or both carotid arteries (Ohki et al 1998).

The management of carotid artery stenosis include Medical treatment, surgical carotid endarterectomy (CEA) and Angioplasty. Medical treatment has not been shown to eliminate the carotid artery stenosis, treatment is aimed at treating the risk factors to slow the progression of stenosis and plaque. However, efficacy against Cerebral Ischemic Events are still being debated. CEA cannot be performed in all patients, for example, of more than 79 years of age, patients with heart severe disease/kidney/liver, abnormalities of the valve/ dys-rhythmia is a risk of embolism, patients with angina/ myocardial infarction within 6 months, patients undergoing major operation less from one month and patients who refuse operation action. In this condition carotid angioplasty is preferred as a therapeutic option because it is simpler, has wider patient selection and less invasive (Antiplatelet trialists' collaboration 1994). This article reported cases of PTA-stenting of carotid artery in patients who experience a TIA and coronary heart disease.

CASE REPORT

Ms. S, a woman of 80 years was ushered by her family into a private hospital in Surabaya because of a fall two

days before admission, the patient did not remember the events that happened to her or where it happened. Bruises were found on the right temporo-parital, abrasions on the right elbow and bruises on the right lower chest and obtained disatric which disappeared a few hours after the incident. Past medical history was as follows: diabetes mellitus, hypertension, PTCA twice, double coronary bypass surgery and angioplasty-stenting in the right iliac artery. The patients was on treatment of 1 x 75 mg Clopidogrel, Aspirin 1 x 100 mg, Lercanidipin 1 x 10 mg, 1 x 20 mg Rosuvastatin, irbesartan 1 x 150 mg, 2 x 500 mg Metformin and Ezetimibe 3 x 10 mg.

Physical examination revealed retrograde amnesia, blood pressure 110/60, pulse 68, respiration 18, right and left carotid bruit, stocking hypestesia, decreased physiological reflexes, strength and tone of normal motor and sensory nerves obtained normal brain. Laboratory examination found: Hb 11, leukocytes 16 200, 226 000 platelets, BUN 60, SK 0.9 Na 136, K 3.2 blood glucose during fasting blood glucose 190 and 129. Total cholesterol 163, LDL 81, HDL 54, TG 103 5.1 uric acid SGOT/PT 76/141. ECG revealed sinus 70 times per minute of normal axis. Holter monitoring showed infrequent ectopic supraventicular with the highest degree of couplets and ventricular ectopic with the highest degree trigeminal. Thorax photograph obtained post CABG, the impression of cardiomegaly and long process in both lung fields. Cranial x-ray revealed no fracture. CT scan of the head showing the brain atrophy, especially in frontal lobe, found no signs of hematome. Echocardiography revealed left ventricular hypertrophy, left ventricular function was still good and no abnormalities found either in the valves or the heart cavity.

Using Doppler sonography imaging, calcification plaque was found in common carotid artery with severe narrowing ±90% in bifurcation areas, with compensatory mechanisms of blood flow occurs through the right and left vertebral arteries.

The right carotid artery arteriography showed severe stenosis in internal and external carotid artery and ranging from bifurcation to 2 cm to the cranium, while arteriography of left carotid artery revealed mild stenosis in internal carotid artery in bifurcation area and significant external stenosis carotid.

Based on the above data the patient was diagnosed with severe symptomatic carotid artery stenosis (TIA), coronary heart disease and diabetes mellitus. TIA diagnosis was based on a history of unconsciousness, amnesia and retrograde dysatria, which is improved some time after the attack.

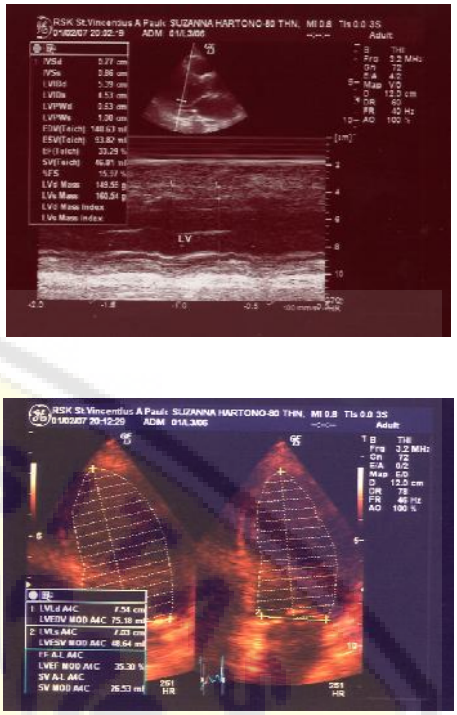

Figure 1. Long axis and 4-chamber apical parasternal echocardiography.

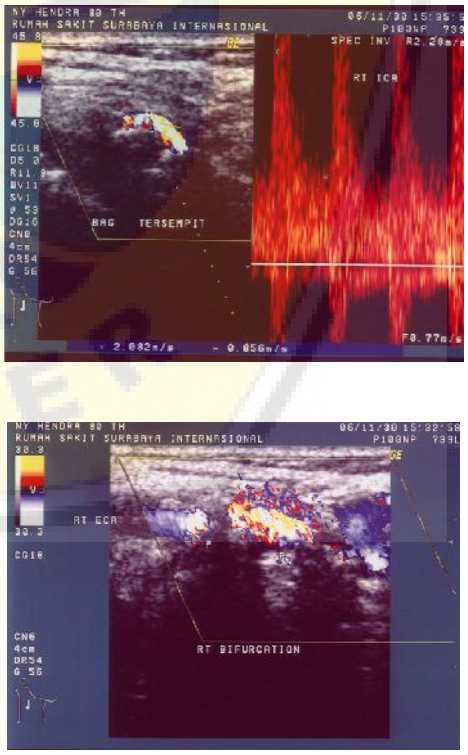

Figure 2. Ultrasound showed severe stenosis in bifurcation area.

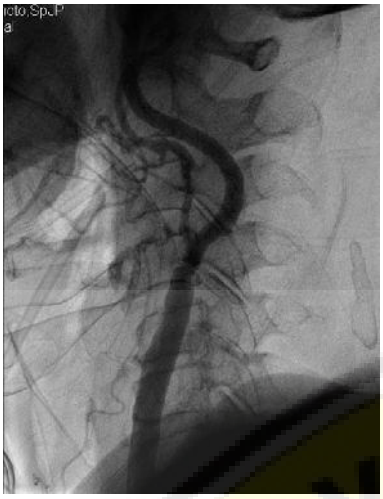

Figure 3. Left carotid Arteriogram

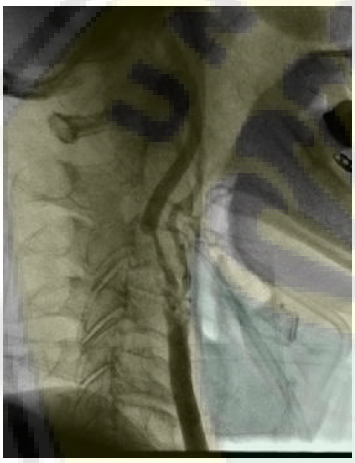

Figure 4. Right carotid Arteriogram

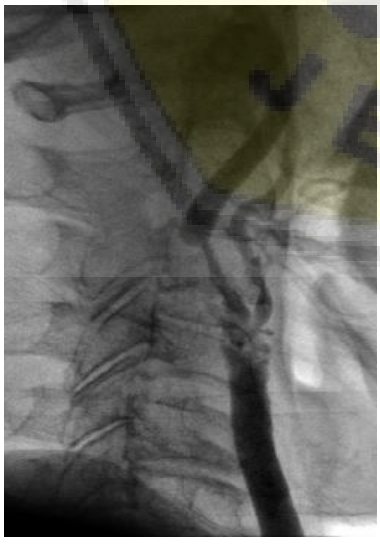

Figure 5. Before PTA-Stenting

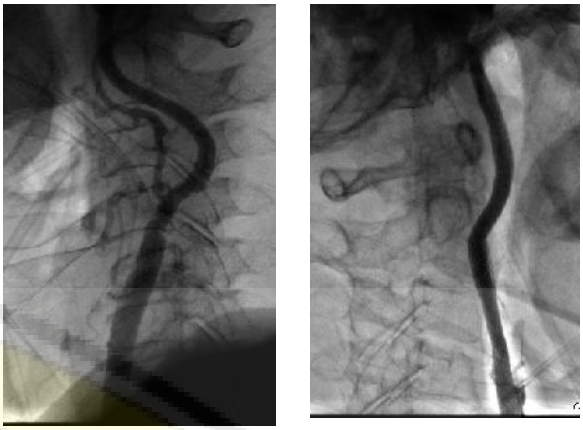

Figure 6. After PTA-Stenting

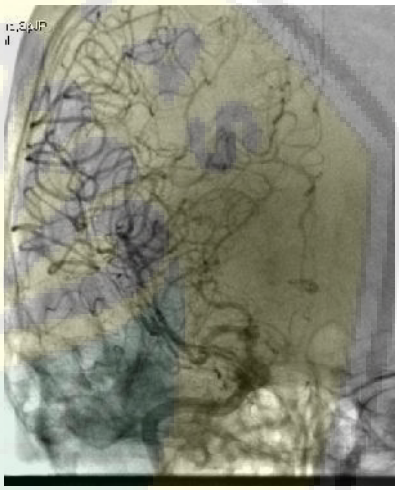

Figure 7. Before PTA-Stenting

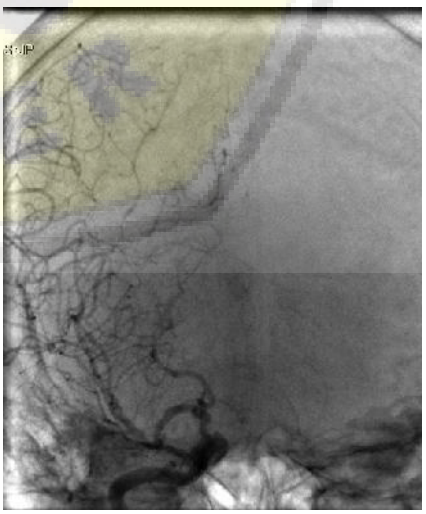

Figure 8. After PTA-Stenting

Once the diagnosis was made, based on clinical judgment and the patient's family consent, we decided to do a carotid stenting using Carotid stent wall 10/24 mm with distal protection filter. Figures 5 and 6 show the differences in the right carotid artery before and after PTA and stenting. Figures 7 and 8 show arteriogram cerebral perfusion after and before the PTA and stenting

After predilatation and stenting in the right carotid artery, we obtained complications such as bradycardia followed by a drop in blood pressure. At that time the patient received therapy of 0.5-1 mg atropine, dopamine Aramin 3 micro and starting from 5 µg, increased based on the response of blood pressure. Patients received dopamine until day 8, the stent was lowered gradually. On day 9 stenting dopamine stand by 1.5 µg and the tenth day the patient was free of dopamine. During and after stenting patients' consciousness remains good, not obtained neurological deficits as other complications of PTA-Carotid Stenting with NIHSS score of 0. On the twelfth day the patient was dicharged from the hospital to go home to continue prior therapy. On the thirtieth day the patient visited the outpatient clinic without any complaint with NIHSS score remained 0.

DISCUSSION

Severe bilateral carotid artery stenosis is a critical condition, and this is a threat to the hemodynamic stability. The compensation mechanism is frequent collateral flow through posterior circulation, but this compensation mechanism does not always work well, leasing to insufficient blood supply to the brain (Rutgers et al 2000). Clinically, signs are easily recognizable for carotid artery stenosis by checking their cervical bruit in the right and left. The existence of cervical bruit in the area need to be followed up with sonography, especially in patients who have vascular abnormalities elsewhere, such as stenosis in coronary, renal artery stenosis or stenosis in extremities area. Research by Cheng and colleagues found carotid stenosis of more than 70% in 11.1% of patients with coronary heart disease and 10.3% of patients with asymptomatic carotid bruit (Cheng et al 1999). Therefore bruit examination in patients with vascular abnormalities in other places is a must (Henry et al 1998).

Currently carotid duplex ultrasonography is the best non-invasive technique in the diagnosis of carotid stenosis. The image obtained from this tool is to determine the degree of stenosis and plaque characteristics such as location, expansion, morphology, calcification and large plaque. Echolucent plaque is easy to rupture so it results in risk of emboli to the brain (Henry et al 1998, Blakeley et al 1995). Clinical features of patients with symptomatic stenosis can be a TIA, syncope or stroke (Henry et al 1998). In this case the patient came with a symptomatic clinical presentation with symptoms of TIA. Clinical signs, in accordance with the literature, were the presence of a carotid bruit, disatri, syncope and vascular abnormalities in other places that are supported by duplex ultrasonography results prove the existence of stenosis in both carotid arteries. The management of carotid stenosis include medicament, CEA and PTA-stenting. PTA-stenting selection in this case was based on the complaint (TIA), performed at high risk for CEA (severe coronary disease), age and previous medical treatment consumed proved not to inhibit the progression of carotid plaque (Antiplatelet trialists' collaboration 1994).

Clinical studies by NASCET (the North American Symptomatic Carotid Endarterectomy Trial) and ECST (the European Carotid Surgery Trial) establishes the CEA as a standard treatment in symptomatic severe carotid stenosis. The study showed that CEA lowers the risk of stroke and death in patients with stenosis exceeding 70% (ECST) or 50% (NASCET). Unfortunately, the data were only obtained from healthy patients who were to undergo surgery and with low risk of complications after surgery (under 6%) 8 Election PTA-stenting in this case was very advantageous because it is free from general anesthesia, avoid the risk of nerve injury and wounds caused by incision in the neck, relatively cheaper cost and shorter hospital stay (Mas et al 2006).

Currently, the only FDA-recommended PTA stenting in the internal carotid artery stenosis exceed 70% and a with high risk of complications after surgery. Research on Stenting and Angioplasty with Protection in Patients at High Risk for endarterectomy (SAPPHIRE) (Yadav et al 2004) which included patients with symptomatic stenosis of more than 50% or asymptomatic stenosis of more than 80% and with high risk operations mainly due to severe coronary artery disease states that carotid stenting is more safe than endarterectomy, the risk of myocardial infarction within 30 days was lower and there was no significant difference in the incidence of stroke or death between carotid stenting and Endarectomy both at 30 days (3.6% vs 3.1%) and 1 year (Furlan 2006). Complication that can occurs in carotid stenting PTA are: (1) Complications in vascular access, such as hematoma and pseudo-aneurysm. (2) embolism, arterial dissection and aneurysm, vasospasm, bradycardia and ekterna occlusion of the carotid artery during recanalization, and (3) with the consequent thrombus formation and thromboembolic occlusion, bradycardia and hypotension that occurs early after the action,

restenosis and occlusion/secondary stent deformation of advanced PTA-Risk (Theron et al 1998). Carotid stenting phase that should be anticipated is stroke, death and myocardial infarction.

In this case the patient experienced bradycardia and hypotension because of stimulation of the vagal reflex during predilatation and stenting. Bradycardia can be treated with atropine sulfate, whereas hypotension is treated with aramin and dopamine. The possibility of embolic stroke complications have been anticipated by the installation of distal protection device before stenting action.

CONCLUSION

Clinical understanding of carotid artery stenosis is very helpful in reducing the risk of stroke and cardiovascular death. Clinical signs easy to detect in all health facilities are a carotid bruit. This sign should be checked regularly, especially in patients with vascular abnormalities elsewhere. Bruit discovery will help clinicians to plan the diagnostic and treatment strategies more quickly and precisely. PTA-stenting is an option for patients with severe symptomatic carotid stenosis or asymptomatic high risk undergoing operation, such as coronary heart disease, old age and severe valvular heart disease.

REFERENCES

Antiplatelet trialists' collaboration (1994). Collaborative overview of randomised trials of antiplatelet therapy--I: prevention of death, myocardial infarction and stroke by prolonged antiplatelet therapy in various categories of patients. BMJ 308, 81-106

Blakeley et al (1995). Carotid artery stenosis: Carotid duplex and carotid Doppler ultrasonography help diagnose. Annal of Internal Medicine 122, 360-367

Boosser MG, Mas JC (1988). Epidemiologie des accidents vascular cerebbaux du sujet jeune. Pressse Mod 17, 143-145

Cheng WK, Wu LH, Lau H, Ting CW, Wong J (1999). Prevalence of significant carotid stenosis in Chinese patients with peripheral and coronary artery disease. Australian and New Zealand Journal of Surgery 69, 44-47

Furlan AJ (2006). Carotid-artery stenting--case open or closed? N Engl J Med 355, 1726-1729

Henry M, Amor M, Masson I (1998). Carotid angioplasty, introduction general data. In: Henry M, Amor M, Theron J, Roubin GS (eds). Carotid Angioplasty and Stenting. International Society of Carotid Artery Therapy.

Mas JL, Chatellier G, Beyssen B, Brancherau A, et al (2006). Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Engl J Med 355, 1660-1671

Ohki T, Marin ML, Lyon RT, Berdejo GL, Soundararajan K, Ohki M, Yuan JG, Faries PL, Wain RA, Sanchez LA, Suggs WD, Veith FJ (1998). Ex vivo human carotid artery bifurcation stenting: correlation of lesion characteristics with embolic potential. J Vasc surgery 27, 463-471

Rutgers DR, Klijn CJ, Kappelle LJ, Van Huffelen AC, Van der GJ (2000). A longitudinal study of collateral flow patterns in the circle of Willis and the ophthalmic artery in patients with a symptomatic internal carotid artery occlusion. Stroke 31, 1913-1920

Théron J, Guimaraeens L, Coskun O, Sola T, Martin J, Rüfenacht DA (1998). Complications of carotid angioplasty and stenting. Journal of Neurosurgery 5, E6

Yadav JS, Wholey MH, Kuntz RE, Fayad P, Katzen BT, Mishkel GJ, Bajwa TK, Whitlow P, Strickman NE, Jaff MR, Popma JJ, Snead DB, Cutlip DE, Firth BG, Ouriel K (2004). Protected carotid-artery stenting versus endarterectomy in high-risk patients. N engl J Med 351, 1493-1501.