SURGICAL MANAGEMENT OF CAROTID ARTERY DISEASE - AN IRISH EXPERIENCE

Pages with reference to book, From 168 To 171

S. Zafar Zaidi, M.P. Colgan, D.J. Moore, D.G. Shanik (Department of Vascular Surgery, St. James`s Hospital, P.O. Box 580, Dublin 8, Ireland.)

Approximately 7,000 patients suffer a stroke in Ireland each year, of these 50% die and of the remainder 44% will require help with eating, washing and dressing with resultant major socioeconomic consequences[1]. Although there has been a slight decline in stroke mortality over the last 25 years it is still responsible for more than 10% of all deaths in developed countries. It is thought that approximately 60% of strokes are secondary to surgically correctable lesions Of the extracranial carotid arteries (C.A.).

Pathophysiology

The majority of strokes are the consequence of arteriosclerosis involving particularly the bifurcation of the common carotid artery (C.C.A.). Other sites less commonly involved are the carotid syphon, the origin of the branches of the aortic arch and the origin of the vertebral arteries. Arteriosclerotic lesions progressively narrow the vessel, eventually resulting in occlusion and thrombosis to the next branch. Of equal importance in the causation of stroke is the production of emboli due to platelet aggregation or thrombus forming on an ulcerated lesion or across a stenosis.

Presentation

Patients with carotid artery disease can present as “Asymptomatic Carotid Bruit”. Symptomatic patients recovering completely in 24 hours have transient ischaemic attacks (TIA’s). The term “transient stroke” is applied to neurological defects recovering within 3 weeks. Acute stroke indicates sudden onset of cerebral infarction. A stroke in evolution or “progressive stroke" is a stepwise deterioration which can span hours or even days while a “completed stroke” indicates stable neurological defect following cerebral infarct.

Investigations

Noninvasive testing of the extracranial carotid arteries is a safe, sensitive and reproducible method of identifying and quantifying atheromatous occlusive disease. Of these the indirect tests such as oculoplethysmography, pulse delay oculoplethysmography and directional periorbital examination depend on their ability to detect alterations of flow or pressure in the vicinity of the eye. Interpretation errors can occur due to collateral pathways or where there is an isolated ophthalmic artery occlusion. The noninvasive direct tests include carotid phohoangiography, phonoangiographic spectral analysis, doppler ultra sonography and B-mode imaging. These require operator skill and errors may occur due to the presence of calcium, tortuosityor misidentification of vessels. Fligh resolution B mode ultrasound provides a means to study not only the anatomy of the vessels but also stenosis and plaque morphology. Attempts at correlating risk of symptoms and risk of embolism from plaque type have been made and plaque morphology has been used for recommending surgery[2]. Transcranial doppler. used to image intracranial vessels around the base of the skull along with dupler scan of the extracranial C.A. ‘s is used routinely in our experience as investigations[3]. Primarily on the audible interpretation and the real time spectral analysis of the doppler signals using criteria outlined by strandness, the vessels are classified as normal, c 50% stenosis, 50-79% stenosis, 80-99% stenosis or occluded. Our value of sensitivity in detecting stenosis of less than 50%; > 50% or occlusion compare favourably with other reported series[4]. However our value of 89% for specificity, is somewhat lower. Bernstein has stated that the most important requirement of a screening test is a high sensitivity even if this is at the expense of specificity[5]. Angiography is considered the gold standard for investigation. But

it is invasive and can damage vessel wall dislodging intimal plaques and throinbus. Digital subtraction angiography using intravenous injection has reduced the invasiveness of this test. Angiography should only be undertaken when surgery is considered. Hill et al have presented a series of 101 cases of carotid surgery without preoperative angiography using noninvasive tests only for evaluation[6]. Angiography is recommended in two groups of patients: a) Symptomatic patients with inconclusive findings on non-invasive testing, b) Patients being considered for surgical treatment.

Indications for surgery

- Those who present with a cervical bruit.
- Patients during investigations found to have disease in their extracranial vessels.
- Those who present with TIA.
- Minor strokes.
- Strokes.

A cervical bruit is present in approximately 5% of the population over the age of 65 and is a poor indicator of underlying pathology and its severity[7]. The Framingham study[8] showed that in a population of 3,709, 4.6% had a bruit and 1.5% neurological events per year while event's appropriate to side of bruit were only 0.4% per year. Surgery for asymptomatic carotid stenosis is a highly controversial topic. Interest was heralded by the report of Thompson et al[9] who noted a significant incidence of TIA's (27%) and stroke (17%) in 138 patients followed for an average of46 months. This was supported by others[10]. However the nonoperated control population used in their study was not randomly chosen and the report included all types and locations of stroke without regard to the site of the bruit. Roederer[11] did a prospective study on 167 asymptomatic patients with cervical bruit followed with duplex scanning. The presence of/or progression to a greater than 80% stenosis was highly correlated (P= 0.00001) with either the development of total occlusion or new symptoms. They suggested surgery to be delayed until appearance of TIA\'s or progression of disease to > 80% stenosis. Major risk factors associated with disease progression were cigarette smoking, diabetes mellitus and age above 65 years. In a prospective study carried out in St. James's Hospital[12] funded by the Medical Research Council of ireland 190 patients with asymptomatic bruits were followed for a 4 year period. 6% suffered a TIA while 2% suffered a stroke. Of the 4 patients suffering stroke one had < 50% stenosis, two had> 50% stenosis while one had > 75% stenosis, illustrating the lack of correlation between degree of stenosis and risk of developing a stroke. Some authors advicate prophylactic surgery if the patient is in good general health[13,14]. The purpose of surgery is to prevent ipsilateral infarcts. The perioperative stroke risk in best referral centres is 2%[15] and in less skilled hands is 5%[16]. If ipsilateral infarct risk is as low as 2% per year in unoperated patients it becomes clear that risk of surgery would outweigh any potential benefit. The only situation in which the benefits of carotid endarterectomy (CEA) might outweigh the risks could be the stage when the asymptomatic patient becomes symptomatic with TIA's. The annual risk of ipsilateral infarct increases from 1.7% to 4.2% after ipsilateral TIA\'s occur[17]. Thompson[18] recommends prophylactic endarterectomy for all patients with asymptomatic carotid stenosis. Busuttl[19] recommends C.E.A. for the haemodynomically significant stenosis. Roederer[11] recommends surgery for > 80% stenosis. Based on our results[12] we recommend that patients with asymptomatic stenosis of the internal carotid artery (ICA) should be carefully followed and considered for surgery only when they develop symptoms of cerebral ischaemia. In USA, C.E.A. is performed 19 times (435 CEA/million/year), more often than in Great Britain and Ireland (23 CEA/million/year) [20]. C.E.A. is now the third most common operation performed in the USA[21]. Asymptomatic bruit as indiction for surgery is considered by only 2% of surgeons in Great Britain and Ireland[20] hence the lower number of operations and restricted range of indications for C.E.A. on this side of the Atlantic. Transient ischaemic attack with ipsilateral carotid artery stenosis is probably the

only surgical indication that is universally agreed upon[22,23]. Callow et al have compared optional surgical results with optional medical results in the therapy of symptomatic patients and have suggested that C. E.A. is beneficial in the prevention of recurrent strokes in patients with mild to moderate neurological deficit[24]. It is difficult however to compare study groups as there is no universally accepted categorization of the post stroke deficit. it has been our policy to consider for surgery only those patients who following a stroke can manage day to day living independently. The second question to be considered in stroke patients who are surgical candidates is the timing of surgery following cerebral insult. Highest complication rate including mortality was found in patients operated on within two weeks of development of an acute neurological deficit[25]. Operation less than 5 weeks after stroke posed an increased risk of worsening an existing deficit or causing a new stroke. A waiting period of 6 weeks before arteriography and surgery in a patient who has suffered a stroke is recommended.

Surgery

The operation which gave the greatest impetus to the development of surgery for carotid disease was that of Eastcott, Pickering and Robb performed at St. Mary's Hospital, London on 19th May 1954[26]. Surgery is carried out under general anaesthesia with endotracheal intubation. Zuccarello et al[27] looked at the morbidity and mortality of 106 CEA\'s under local anaesthesia and have suggested safe operation even in high risk patients under L.A. In their study nonfatal stroke occurred in 2% and TIA in 1% with no perioperative mortality. However G.A. allows control of ventilation and oxygenation and eases the management of blood pressure changes.

Technique

Patient is placed supine with neck hyper extended and turned to the opposite side. Incision is vertical, parallel to the anterior border of sternocleidomastoid muscle. The carotid sheath is incised and the internal jugular vein is retracted laterally to expose the common carotid artery (C.C.A.). The vagus nerve is usually the most posterior occupant of the sheath but occasionally it takes an anterior course and this should be anticipated. The C.C.A. is mobilized circumferentially and encircled with an umbilical tape. The dissection is continued superiorly to a point where the common facial vein crosses the artery. This is a constant landmark for the bifurcation of the C.C.A. The vein is mobilized, ligate4 and divided. Care must be taken to avoid undue handling of the bifurcation as atheromatous lesions are friable and debris may be dislodged with resultant cerebral. cmbolization. The hypoglossal N. is identified and the internal carotid artery (I.C.A.) is mobilized to a point well above the palpable athermatous lesion where the vessel is soft and disease free. Same is done for the external carotid artery (E.C.A.) without damaging its branches. A longitudinal arteriotomy is extended from the carotid bulb into the ICA. A bolus dose of I.V. heparin 2,000-4,000 lU. is given to avoid thrombosis. To reduce the risk of perioperative hypoxia, an intraluminaijavid shunt is used[28]. The shunt is placed in the C.C.A which is then opened with resultant flushing of the shunt, ensuring adequate flow and exclusion of air. The distal end is inserted into the ICA and secured. Decision to shunt is made after assessing collateral hemispheric blood flow. In our experience this is routinely done by assessing the back pressure down the I.C.A. on the occluded side by intra arterial stump pressure. Shunting is performed if stump pressure is less than 40 mmHg. Another technique used for collateral blood flow is BEG monitoring. Selective shunt insertion does not pose any time limitations for surgery. However it has been challenged on grounds of technical difficulty, such as damage and embolization of internal lining, dislodging thrombus or atheromatous debris and air embolism[29]. The most critical technical detail of CEA itself is the selection of the correct plane. The optimum plane lies between the diseased intimar and the circular fibres of the arterial media. Magnification is necessary for ensuring satisfactory end points of the atheroma. The endartcrectomy is continued circumferentially. The atheromatous lesion is sharply divided at its proximal limit, while at the distal end in the ICA it comes free where the intima becomes relatively normal leaving a smooth tapered end point. The endarterectomy area is then meticulously irrigated with hepranized saline and any loose debris or tiny strips of media removed. Any

floating intimal flaps are secured with fine tacking sutures. When complete the arteriotomy is closed with continuous 6/0 proline suture. The ICA is opened first to test for major leaks and then the ECA. The ICA is compressed when CCA clamp is opened allowing any debris to flush into ECA rather than ICA. Gentle pressure to suture line leads to haemostasis since stitch holes bleed freely in endarterectomized artery and in a heparinised patient. Patency is determined by doppler and the wound is drained. The patient receives aspirin for the first 3 postoperative months to theoretically reduce the incidence of myointimal hyperplasia[30].

Complications

Major complications of CEA relate to stroke problems and have been shown nearly always to occur 1/2 to 4 hours after surgery. Important risk factors for post CEA intracerebral haemorrhage are previous cerebral infarction, intraoperativc or postoperative hypertension and use of anti-coagulants[31]. Technical problems like intimal flap and bleeding are rarely a problem. Numbness from division of anterior cutaneous cervical nerve is always present. Hypoglossal N. paresis occurs due to traction. Bilateral hypoglossal N. injury after bilateral CEA is particularly serious[32]. Recurrence of stenosis of the carotid vessel following CEA has been reported[33,34]. We prospectively re-examined our patients using non-invasive techniques and found the incidence of restenosis was 1.25% when based on clinical symptoms, while the overall incidence of restenosis including asymptomatic patients was 12.5%. Stoney and Sting[35] reviewed 1,500 patients having C.E.A. and distinguished between early restenosis occurring within 24 months of surgery characterised by myointimal hyperplasia and late restenosis that occurred up to 15 years postoperatively due to progression of atherosclerotic disease. Risk factors associated with early restenosis following CEA have been identified as diabetes mellitus, hypertension and hyperlipidaemia. Myocardial infarction is the major hazard in patients undergoing CEA who have coronary artery disease[36]. There is a 30% cardiac mortality in 5 years following C.E.A. in patients with coronary heart disease. Zurbruegg et al have done a 10 year literature review comparing mortality and morbidity of C.E.A. with risk of carotid stcnosis managed by best medical treatment[37]. They showed that mortality for all patients undergoing C.E.A. was 2.1%, for asymptomatic patients was 1.1%, for TIA\'s was 1.3% and patients with acute stroke faring the worse with 17.1%. Similarly the incidence of permanent stroke in all patients of CEA was 3.5, for asymptomatic patients 3.1% and for TINs 4.4%.

REFERENCES

1. Radic, A.. Finn. A., Haran, ft. and Dean, 0. Functional recovery in stroke patients. J.Jr.Med.Ass., 1979;72: 129-61.
2. Matalanis. 0. and Lusby, R.J. Is there still a place for carotid endartereetomy? Clin.Exper.Neurol., 1988;25: 17-26.
3. Doorly. T.P., Atkinson. P.I., Kingston, V. and Shanik, DO. carotid ultrasonic arteriographycombincdwilh real time spectrslanab'sis; a comparison with angiography. J.Cardiovase, Surg., 1982;23:243-6.
4. Summer. D.S., Russell, 3.8. and Miles, RD. Are noninvasive tests sufficiently accurate to dent ify patients in need of carotid atieriography? Surgery. 1982;91:700-6.
5. Bernstein. E.F. The current status of noninvasive testing for cerebrovasculsr disease, Edited by R.M. Greenhalgh. hormones and vascular disease. London, Pitman, 1981, pp. 102-12.
6. Hill, J.C.. Carbonneau, K. and Pcabhaker. K. Safe extraersnial vsseusl evsluation and surgetywit soot preoperative arteriograph. Ann.Vasc.Surg., 1990;4:34-8.
7. Hennerici. M.. Aulich, A., Sandmann, W. and Freund, H.J. Incidenee of asymptomstic extraeranial arterial disease, Stroke, 1981;12:750-58
8. Wolf, P.A., Kannel, W.B., Sorlie, P. and MeNamars, P. Asymptomaticcsrotidbruitand risk of stroke.

The Framingham study. JAMA., 1981;245:1442-45.
9. Thompsoon, J.E., Palman, R.D. and Perason, A.V. Managemeni ofasymptomaticcarotid bruits. Am.Surg.. 1978;42:77-80.
10. Dorazio, R.A., Ezzet. F. and Nesbitt, N.J. Long-term follow-up of ssymptomatiecarotid bruits. Am.J.Surg., 1980;140:212-13.
11. Roederer, 0.0.. Langlois, YE., Jager, K.A., Primozich, iF., Beach, K.W., Phillips, Di. and Strandriess, D.E.Jr.The natural history of carotid arterydisease in asympt.omatie patientswith cervical bruits. Stroke, 1984;15:605-13.
12. Colgan, M.P., Kingston, W. and Shanik, DO. Asymptomatie carotid stenosis; is prophylactic endarterectomy justifiable? Br.J.Surg., 1985;72:313-14.
13. Moneta, L.G., Taylor. D.C.. Nicholls, CS., Bergelin, R.O. 'Zierler, RE., Kszmers, A., Cloves, A.W. and Strandriess, DR Jr. Operativeversus nonoperative management of asymptomatic high-grade internal carotid srtety stenosis: Improved results with endarterectomy. St coke, 1987; 18: 1005- 10.
14. Quinonea Baldrieh. W.J. and Moore, W.S. Asymptomaticearotid stenosis. Rationale for management. Arch.Neurol., 1985;42:378-82.
15. Warlow, c. Carotid endsrterectnmy; does itwork? Stroke, 1984;15:1068-76.
16. Brott. T. and Thalinger. K. The practice of carotid endsrtereetomy in a large metropolitan area. Stroke, 1984; 15:950- 55.
17. Boguusslavasky, 3., Despland, PA. and Regli, F Asymptomatie tight stenosis of the internal carotid artery: Long-term prognosis. Neurology. 1986:36:861-63.
18. Thompson, J.E. Don't throw out the baby with bath water. A perspective on carotid endancrectomy. .t.Vasc.Surg.. 1986:4:543- 45.
19. Busut it, R.W., Baker, 3D., Davidson, R.K. and Machleder, H.I. Carotid artery stenosis Haemodynamic significance and clinical course. JAMA., 1981;245:1438-41.
20. Murie, J.A. and Morris, PJ. Carotid endarterectomyin Cireat Britain and Ireland. Br.J. Surg., 1986;73:867-70.
21. Barnett, H.J., Plum, F. and Walton, J.N. Carotid endarterectomy - an expression of concern. Stroke, 1984;15:941.43.
22 Thompson, i.E. Carotid endarterectomy, 1982 - the state of the art. Br.J.Surg., 1983;70:371-76.
23. DeBakey, M.E., Lawne, G.M. and Glaeser, D.H. Patterns of atherosclerosis and their surgical significance. Ann. Surg,,1985;201:115-31.
24. Callow, A.D. and Mackey, W.C. Optimum results of the surgical treatment of carotid territory ischemia. Circulation, 1991;83:1190-95.
25. Giordano, J.M., Trout, H.H., Kozloff, L. and Depalma, R.G. Timing of carotid artery endarterectomyafterstroke. J.Vasc.Surg., 1985;2:250-55.
26. Eastcott, H.H.G., Pickering G.W. and Robb, C. Reconstruction of internal carotid artery, in a patient with intermittent attacks of hemiplegia. Lancet, 1954;2:994-96.
27. Zuccarello, M., Yeb, H.S. and Tew, J.M. Morbidityand mortality of carotid endarterectomy underlocal anaesthesia, a retrospective study. Neurosurgery. 1988;23:445-50.
28. Browse, N.L and Ross-Russell, R. Carotid endarterectomy and the Javid shunt; the early results of 215 consecutive operations for transient ischaemic attacks. Br.J.Surg., 1984;71:53-57.
29. Piepgras, D.G., Marsh, W.R. and Sundt, T.M. Recurrent carotid stenosis. Results and complications of 57 operations. Ann. Surg., 1986;203:205-13.
30. DeWeese, J.A. and Green, A.M. Anastomotic neointimal fibrous hyperptasia in complications in vascular surgery. Edited by V.M. Bernhard and J.B. Towne. New York. Grune and Stratton, 1980, pp. 153-65.
31. Shuaib, A., Hunter, K.M. andAnderson, M.A. Multiple intracranial haemorrhagesafter carotid endarterectorny. Can.J. Neurol.,Sci.,1989; 16:345-47.
32. Gutrecht, J.A. and Jones, H.R. Jr. Bilateral hypoglossal nerve injury after bilateral carotid

endarterectomy. Stroke. 1988; 19:261-62.
33. Colgan, M.P., Kingston, V. and Shanik, G. Stenosis following carotid endarterectomy. Its implication in management of asymptomatic carotid stenosis. Arch. Surg., 1984;119:1033-35.
34. Nicholls, R.C., Phillips, D.J. and Bergelin, R.O. Carotid endarterectomy. Relationshipof outcome to early restenosis. J.Vasc.Surg., 1985;2:375-81.
35. Stoney, RJ. and String. S.T. Recurrent carotid stenosis. Surgery. 1976;80:705-10.
36. Newman, D.C. and Hicks, R.G. Combined carotid and coronaryartery surgery; a review of the literature. Ann. Thorac., Surg., 1988;45:574-81.
37. Zurbruegg. H.R. Seiler, R.W., Grolimund, P. and Mattle, H. Morbidityand mortality of carotid endarterectomy. A literature review of the results reported in the last 10 years. Acts Neurochir. (Wien), 1987;84:3-12.