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In this issue, we present you expert articles describing ischemic stroke concerned with carotid lesions & their link with coronary heart disease, along with the pivotal role played by carotid endarterectomy in times of ‘urgency’. In order to improve neurological & functional outcomes & quality of life, preventive and therapeutic strategies must control both cerebral and systemic risk. Neurosurgeries like decompressive hemicraniectomy can produce good functional outcomes in such conditions with minimally compromising functional decline of the patient especially concerning disability & aphasia. We also present an interesting article studying brain swelling following stroke & its associated complications.

We sincerely thank our readers for supporting us in this endeavour. We hope our readers find this issue as interesting as the previous one, helping doctors to take management & treatment of stroke to the next level.

Regards
Dr. S.M. Hastak
Dr. Abhishek Srivastava

A PIECE OF EDITOR’S LEIXS

Dear Friends,

We welcome you to this new issue of Stroke Talk in continuation with our last issue entitled “Neurology Stroke. When and How?”. We were overwhelmed by the response from our readers from last issue where we discussed different neurosurgeries currently in use.

In this issue, we present you expert articles describing ischemic stroke concerned with carotid lesions & their link with coronary heart disease, along with the pivotal role played by carotid endarterectomy in times of ‘urgency’. In order to improve neurological & functional outcomes & quality of life, preventive and therapeutic strategies must control both cerebral and systemic risk. Neurosurgeries like decompressive hemicraniectomy can produce good functional outcomes in such conditions with minimally compromising functional decline of the patient especially concerning disability & aphasia. We also present an interesting article studying brain swelling following stroke & its associated complications.

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Regards

Dr. V.G. Pradeep Kumar
Dr. D. Nagaraja
Dr. Dheeraj Khurana

Stoke Talk

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10-15% of all MCA territory ischemic strokes are hemorrhagic infarctions. Subsequent neurological deterioration and death occurs in 40-80%. It is typically seen in patients with ICA or mainstem MCA occlusions.

Clinically the most common findings are hemiplegia, global or expressive aphasia, severe dysarthria, neglect, gaze preference, and a visual field defect. An initial National Institutes of Health Stroke Scale score of > 20 with dominant hemispheric infarction and <15 with nondominant hemispheric infarction is a sensitive but not specific marker. Papillary abnormalities indicate significant brainstem compression and develop between 3 to 5 days. Acute carotid artery occlusion or dissection may present with an early Horner’s syndrome.

The most specific sign of significant cerebral swelling after stroke is a decline in the level of consciousness, usually occurring within 72-96 hours. However combined MCA and ACA infarctions may have diminished responsiveness from the beginning. The presence of cerebral pressure (right MCA occlusions) may give a false suggestion of a decreased level of consciousness. The other clinical parameters associated with development of a malignant infarction include early nausea vomiting, younger age (intracranial compliance is less as compared to older patients), female sex, congestive heart failure and leukocytosis. Haemorrhagic conversion of the infarct may produce a new mass effect and lead to worsening.

On neuroimaging progressive cerebral edema and mass effect is characterised by isipilateral ucal effacement, compression of the ipsilateral ventricular system, and then a shift of the midline structures such as the septum pellucidum and the pineal gland. The blockage of foramen of Monro or the third ventricle might lead to entrapment and dilatation of the contralateral lateral ventricle and obstructive hydrocephalus. Brainstem displacement may lead to widening of the ipsilateral ambient cistern. These cisterns become effaced when swollen tissue eventually fills the cisterns. Infarction in the territories of the anterior or posterior cerebral arteries may be seen in some patients. Serial CT findings in the first 2 days are thus useful to identify patients at high risk for developing symptomatic swelling. CT findings that predict malignant edema and poor prognosis include frank hypodensity on head CT within the first 6 hours, involvement of one third or more of the MCA territory and inhomogeneous dense MCA sign or midline shift ≥5 mm within the first 2 days is also associated with neurological deterioration and early mortality.

Angiographically a “Tocclusion” of the distal internal carotid artery (TIA) and a complete circle of Willis leading to involvement of multiple vascular territories is also predictive of development of malignant edema and worse outcome. On MRI, acute DWI volume (MRI done within 6 hours of stroke onset) of > 80 ml and DWI volume of ≥ 145 ml (MRI done 14 hours after stroke onset) is predictive of a progressive downhill course.

Such patients should be managed in an intensive care or...
Surgical treatment of the swelling associated with cerebral infarction is performed by removal of the skull or expansion of the dura as is known as decompressive craniectomy with dural closure. Three prospective, randomised trials (i.e., Decompressive Surgery for the Treatment of Malignant Infarction of the Middle Cerebral Artery (DESTINY)10, Decompressive Craniectomy in Malignant Middle Cerebral Artery Infarction [DCMCA]12, and Hemicraniectomy After Middle Cerebral Artery Infarction With Life-threatening Edema Trial [HAMLET]13) and 14 case series have studied patients with supratentorial treatments treated with decompressive craniectomy within 48 hours of stroke onset. On the basis of a pooled analysis, it is recommended that patients <60 years of age with unilateral MCA infarction who deteriorate neurologically within 48 hours after stroke onset, should undergo decompressive craniectomy with dural expansion.10 The effect of later decompression is not known, but it should be thoroughly considered. The above trials have shown a significant reduction in mortality (22% versus 71% mortality) with decompressive craniectomy compared to medical management. The residual disability remains significant as only 14% of the survivors could look after their own affairs without assistance (mRS score 2).10

All prior clinical trials involved patients <60 years of age (mean age, 45 years) and it remained unclear whether older patients would experience a similar effect until the results of DESTINY 2 became available. The trial concluded that early hemicraniectomy significantly increased the probability of survival even among patients older than 60 years of age with malignant middle cerebral artery infarction (proportion of patients who survived at 6 months without severe disability was 38% in the hemicraniectomy group compared with 18% in the control group), but majority required assistance with most bodily needs (7% had modified rankin score of 3, 32% had a score of 4 and 28% had a score of 5 compared to 3%, 15% and 13% respectively in the control group).12 Postoperative concerns include wound dehiscence and requirement of tracheostomy and gastrostomy in the initial postoperative period. The timing of cranioplasty after decompressive craniectomy is determined by a wide variation in clinical practice. Early cranioplasty (within 10 weeks of craniectomy) results in higher chance of complications compared to cranioplasty, and particularly in patients with a ventriculoperitoneal shunt at the time of cranioplasty. Late cranioplasty can produce a communicating hydrocephalus requiring ventriculoperitoneal shunt placement.10

Indian perspective

In a prospective observational cohort study on 60 patients of large MCA infarcts, decompressive hemicraniectomy lead to markedly improved survival (45% risk reduction in death in 1 year compared to mortality in the management alone) and better functional outcome (motor and language) at 1 year (20% patients achieved good outcome that is modified rankin score ≤ 3 as compared to none in the other group). The benefit of surgery in motor and apahra recovery was progressive and sustained until 1 year.14

Malignant cerebellar infarction

Few, if any antecedent symptoms and signs can contribute to stratter cerebellar stroke patients across a continuum of clinical severity. Swelling after cerebellar infarction may result in failure of compression causing ophthoparesis, breathing irregularities, and cardiac dysrythmias. It may produce acute hydrocephalus secondary to obstruction of the fourth ventricle. Often both pontine compression and hydrocephalus occur together. The most reliable clinical evidence is in the post stroke swelling associated with loss of consciousness. Peak swelling occurs several days after the onset of ischemia. Elevation of the fovea on the midline is a radiologic marker, followed by basal cistern compression, followed by brainstorm deformity, oculomotor dysfunction, sleep disturbance, and upward transtentorial herniation. Predictive MRI based infarcts have not been clearly defined for cerebellar strokes.

Decision of surgery (Bicocaplan craniectomy) is based on a combination of clinical and radiologic worsening. The time interval to surgery does not seem to affect outcome. The value of preemptive surgery (that is when swelling and hydrocephalus progress on CT scan in a clinically stable patient) and the best neurosurgical approach (i.e., removal of necrotic tissue versus decompression alone versus decompression and ventriculostomy) are not known.

Surgery in spontaneous ICH

For most patients with ICH, the usefulness of surgery is uncertain. As per the 2010 AHA/ASA guidelines,15 patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible. Initial treatment of these patients with ventricular drainage alone rather than surgical evacuation is not recommended. For patients presenting with low lobar clots >30 mL and within 1 cm of the surface, evacuation of supratentorial ICH by standard craniotomy allows for the effectiveness of minimally invasive clot evacuation by stereotactic or endoscopic aspiration with or without thrombolytic usage is uncertain and considered investigational. Although intraventricular administration of recombinant tissue plasminogen activator in IVH appears to have a fairly low complication rate, efficacy and safety of this treatment is uncertain and is again considered investigational.

Conclusion

To conclude, brain swelling following stroke can cause secondary cerebral ischemia, herniation syndrome, and brain death, and immediate surgical treatment is necessary to prevent detrimental outcomes. Important questions such as the acceptable degree of decompression and its post surgery, long-term effect of chronic incapacity, the importance of apahra and the best timing of surgery require further research.

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OPEN SIZE: W-17” X H-11.5”
ISCHEMIC STROKES RELATED TO CAROTID-ILDENSIONS, AND LINK WITH CORONARY HEART DISEASE

Dr. S. Ravikumar and Dr. K. Shrinivasan

It is now realized that extracranial carotid disease (ECCD) as a cause of stroke is less common in the Tropics compared to the west. Intracranial (ICD) and cardiovascular causes proved in more than 20-30% of patients. 1-3 Earliest studies have reported that urgent CEA showed decreased with time from event to surgery. 4 As a consequence of the above findings, clinical guidelines were revised and widely implemented in clinical practice.

A study was done by P Gajin, Dj Radak, S Tanaskovic, S Babic and D Nenezic to analyze the outcome of urgent CEA in patients with crescendo TIA and stroke in progression, and was performed within less than six hours after index event occurred. 58 urgent CEs were done in all 46 patients with crescendo TIA and 12 patients with stroke in progression. Carotid stenosis was estimated by the means of computed tomography angiography and color duplex scan according to ECST criteria. Carotid stenosis was considered significant (>70%) if peak systolic velocity (PSV) was >150 cm/s and end diastolic velocity (EDV) <90 cm/s. Median follow-up was 42.2 ± 16.6 months. In the crescendo TIA group (TIA, stroke), stenosis was considered as ‘early’ but not ‘urgent’ Natural course of carotid related ischemic stroke is poor. Re-infarction rate is about 12% and mortality 19% within the first year after index event

Our study: results - see Table

Adult ischemic strokes (346pts). CABA 120 patients as a preliminary study. Carotids assessed by USG, MRA and in CABA cases by catheter angiography. 1. Symptomatic ICA lesions with compelling need for CEA or CAS in only less than 10%. Significant carotid atheroma in only 20%. Carotids normal in 50-60% of Ischemic Strokes and CHD. 2. Link between carotid and coronary disease more than strong in the west. However carotid bruit should alert both the neurologist and cardiologist. 3. Less well studied but relevant: Morphology of plaques, (CMR recorded), Haemodynamic factors, Haemorheology etc. 4. Common risk factors (RF), e.g. Diabetes, Hypertension, Lipids can be decisive blamed in only less than 50% of CVD or CHD. Especially with the increased intracranial and carotid embolic causes more common in the East than the west.

Patients ask: Why did I need CABA? Or why did I experience TIA or stroke. I do not have any PE, smoking, drinking, Diabetes, H.T., Lipids, etc. We blame Genes, Stress and Life style etc.

EXTRACRANIAL EMERGENCY CAROTID ENDARTERECTOMY

Carotid artery endarterectomy (CEA) aims to remove atherosclerotic plaque from the carotid artery, preventing thrombus formation on the plaque, which could otherwise lead to a stroke or transient ischemic attack (TIA). Crescendo TIA is defined as two or more episodes within 24 hours, with complete recovery after each episode, while stroke in progression is defined as gradual deterioration of neurological deficit that took place for over at least six hours. CEA can be termed ‘urgent’ only if done immediately after or during arising neurological ischemic event (within six hours or less). In the early postoperative time, the ischemic (event, TIA, stroke) can be termed as ‘early’ but not ‘urgent’. Natural course of carotid related ischemic stroke is poor. Re-infarction rate is about 12% and mortality 19% within the first year after index event

APPENDIX

Image of Carotid Endarterectomy

After the three large multicentric randomized trials, namely North American Symptomatic Carotid Endarterectomy Trial (NCCT), European Carotid Surgery Trial (ECST) and Veterans Affairs Cooperative Studies Program Trial Group, published their results, it became evident that carotid endarterectomy (CEA) is beneficial for stroke risk reduction in symptomatic and asymptomatic patients with high-grade internal carotid artery (ICA) stenosis. 15-18 Earliest studies have described urgent CEA with a high rate of postoperative mortality in the range of 42-60%. 19-20

Later studies reported that urgent CEA showed decreased rate of postoperative complications. 21 Most recent studies demonstrated that urgent CEA performed in patients with crescendo transient ischemic attack (TIA) as well as patients with stroke in evolution could have favorable outcome 8-9 Increased perioperative risk has been described in patients with crescendo TIA and stroke in evolution compared with the outcome after elective CEA. 22-23

After the publication of results from the North American Symptomatic Carotid Endarterectomy Trial (MASCET) and the European Carotid Surgery Trial (EACT), guidelines for stroke care in the USA, Europe and elsewhere recommended operating on patients with symptomatic carotid stenosis within 6 months of the presenting ischaemic event. In 2004, a pooled analysis of data from MASCET and EACT was conducted to further the effect of surgery, including surgery timing, outcomes in various subgroups etc. The study concluded that patients benefited most from an operation within 2 weeks of an ischaemic event and thereafter, this benefit decreased with time from event to surgery. As a consequence of the above findings, clinical guidelines were revised and widely implemented in clinical practice.

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