EDITORIAL

MANAGEMENT OF STROKE

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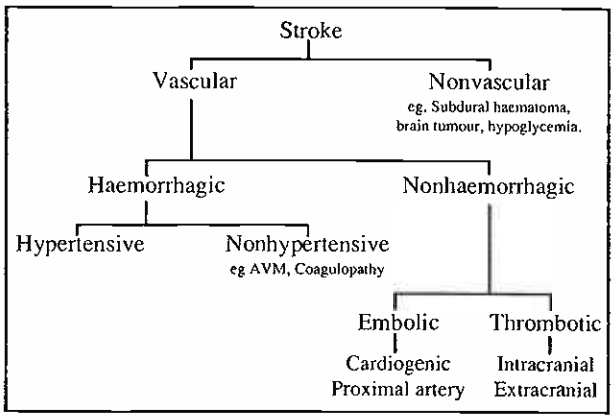
SINGAPORE MED J 1994; Vol 35: 26-28

A recent paper by Broderick and his colleagues[1] reported a disconcerting increase in the incidence of cerebral haemorrhage and infarct among residents of Rochester and the surrounding areas. The increase reversed a declining trend which began in the 1950s, and may be attributable to increased survival of patients with coronary heart disease, diabetes and hypertension. Stroke patients also have a better chance of survival[2,3]. Earlier diagnosis, better supportive treatment, control of risk factors and rehabilitation are probably contributory factors to lower mortality and morbidity.

The stroke syndrome is defined as a focal neurological deficit of sudden onset. Fig 1 gives a classification according to aetiology and pathology. It is a guide to a diagnostic approach to a patient with suspected stroke. The syndrome may be due to vascular or nonvascular causes. Examples of nonvascular causes include subdural haematoma, neoplasm, Todd's paralysis and hypoglycemia. A vascular cause may not be recognised when a proper history is unavailable, or the patient presents with confusion, dysphasia or dizziness and dysequilibrium with little or no motor signs. Some other interesting presentations of inobvious stroke are sudden onset of unilateral hand tremor, cortical wrist drop and paralysis of cranial nerves 9 to 12 without accompanying long tract signs. The latter may be due to dissection of the internal carotid artery[4].

Fig 1 – Classification of Stroke Syndrome



An accurate history and thorough clinical examination are essential factors for a correct diagnosis. Important points to note in the history are the mode of onset, progression, previous history of stroke and coronary artery disease and family history of strokes. Patients often omit to mention brief attacks of limb weakness preceding the stroke by a few days to weeks. Physical examination should include not only the neurological system but also the cardiovascular system. As an error in diagnosis may lead to inappropriate treatment which may be disastrous, a high degree of certainty is demanded in diagnosis. Thus most patients require CT Head or MRI for evaluation. When the patient is seen after an acute stroke, it has been my practice to have a CT Head done in the first 24 hours mainly to exclude cerebral haemorrhage and to plan further management. Although the CT is often negative in the first 24-48 hours for an ischaemic infarct, subtle changes such as a slight increase in attenuation in a focal area, or localised effacement of sulci may be present. Recently Tomsick et al[5] and Schuknecht et al[6] have reported increased density in an occluded cerebral artery in patients with cerebral infarct.

The differential diagnosis between haemorrhage and infarction is easily made with the CT scan. It may be difficult however to separate an embolic stroke from cerebral thrombosis. Most neurologists would accept a large haemorrhagic infarct in the area of a cerebral artery as indication of an embolic occlusion. Large subcortical infarcts involving the striatum and internal capsule are usually due to embolic occlusion of the middle cerebral artery[7]. Multiple infarcts in different vascular territories indicate cerebral embolism. The presence of a cardiac lesion raises the possibility of embolism but does not exclude other pathogenetic mechanisms.

Localising the vascular territory involved is of more than academic importance:

i) Subsequent strokes may occur in the same vascular supply or a different vessel suggesting progressive thrombosis or embolism respectively.

ii) It has been the author's experience that progressive vertebrobasilar strokes respond more readily to heparin than carotid territory stroke.

iii) If the patient has a classical lacunar stroke, the prognosis is usually better as it indicates small vessel disease and there is absence of intracranial hypertension from cerebral oedema.

iv) MRI Head is the preferred mode of neuroimaging if posterior territory stroke is suspected.

When should a patient be admitted? It is probably good practice to hospitalise for observation and management all acute strokes except those that have been stable for at least two weeks. Even a patient who appears to have only a mild stroke should be considered for admission as his condition may deteriorate rapidly. It is good practice to inform the patient and his family of this possibility.

Management of the patient with a cerebral infarct

a. *Drug management.* Aspirin or ticlopidine is usually used to reduce the risk of further progression, inspite of the lack of evidence to support this. Although the usefulness of other drugs such as pentoxifylline, co-dergocrine mesylate, dipyridamole, piracetam and citicholine are not proven, they are often used as well to either limit the neurological damage or prevent further stroke. There have been several reports on the use of nimodipine in ischaemic stroke[8,9]. The results have not been dramatic, and further studies involving larger number of patients are required. I would only use nimodipine in a patient with subarachnoid haemorrhage to prevent vasospasm. It should be used with care

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when cerebral oedema or raised intracranial hypertension are present. As the drug interacts with a number of anti-hypertensive agents careful titration of the dose is required. Thrombolytic agents, such as streptokinase if used early enough may reopen occluded arteries. However there is a risk of haemorrhage into the infarcted area which may be fatal, and its general use cannot be recommended at present. Cerebral oedema in cerebral infarction is best treated with intravenous mannitol. Steroids are not indicated in the treatment of cerebral oedema from infarction.

Patients with cerebral infarction from cardiogenic embolism should be considered for immediate anticoagulation if the infarct is not large (ie lobar, or accompanied by midline shift), and initial CT scan shows no haemorrhagic infarction. In large infarcts one should wait for 48 to 72 hours to repeat the CT scan, and begin anticoagulation only if the scan is negative for haemorrhage. If haemorrhage has occurred into the infarcted area, anticoagulation should be deferred for 4 to 6 weeks.

Progressive stroke from thrombosis is usually treated with intravenous heparin although its effectiveness, particularly in carotid territory strokes, is not proven.

Hypertension in the first few days of an acute ischaemic stroke should be treated gently[10]. It is acceptable for the blood pressure to be in the range of 150-170 mmHg systolic and less than 100 mmHg diastolic. If the patient has been hypertensive, then readings of 170/100 are quite acceptable. In cases of severe hypertension (diastolic >140mmHg) infusion of sodium nitroprusside (0.5–10 µg/kg/min) should be commenced. Unless the patient is in left ventricular failure or has intracerebral haemorrhage, rapid lowering of blood pressure is not wise. Labetalol (oral 200-300mg bd or tds), captopril (12.5-25 mg bd) or nifedipine (10mg 6 hourly) may be used. It should also be remembered that blood pressure may rise because of airway obstruction, cerebral oedema or a distended bladder. Unless these possibilities are excluded, iatrogenic hypotension may result from overdosage.

b. *Excellent nursing care* plays a vital role. Patients may require the insertion of a nasal gastric tube for feeding, and catheterisation. Attention to posture of the limbs in bed, skin care and care for bowels are also important. The nursing team must also be alert to any change in the neurological status of the patient.

c. *Physiotherapy* should begin early to minimise disability from pain and contractures. It preferably should be supervised by a rehabilitation physician and individualised for the patient.

Cerebellar haemorrhage

This category of stroke deserves special mention as it can be life-threatening. Yet appropriate management is often satisfying. Usually the patient presents with sudden onset of vertigo, headache and vomiting. Neurological examination may reveal only severe ataxia, although unilateral limb ataxia, ipsilateral gaze palsy and facial palsy may be present as well. Unless the physician is aware of this presentation the diagnosis may be mistaken for vestibular neuronitis or Meniere's disease. An urgent CT scan is indicated. Once the diagnosis is made, the patient should be admitted to a hospital where neurosurgical expertise is available 24 hours. Haematomas greater than 3 cm diameter or in the midline of the cerebellum should be considered for surgical evacuation[11]. As sudden respiratory arrest may result from brainstem compression, such patients must be monitored closely.

Basal ganglia haemorrhage

These may be treated conservatively if the patient is stable. Evacuation of the haematoma may be indicated to save life if the patient is deteriorating and the haematoma is large. The quality of life after surgical evacuation may be quite good. The prognosis for small basal ganglia haemorrhages is excellent, with many patients recovering fully.

Management of transient ischaemic attacks (TIAS)

Current opinion favours the use of anti-platelet drugs such as aspirin or ticlopidine. The dose of aspirin is controversial, but best evidence to date indicates 150 mg – 300 mg a day as effective. Aspirin is probably less effective in women. Dipyridamole is commonly used in combination with aspirin although there is no evidence that this combination is superior to aspirin alone. Ticlopidine at the dose of 250 or 500 mg a day has the advantage of being effective in both men and women, but there is a risk of bone marrow depression especially in the first 3 months[12]. I tend to favour ticlopidine over aspirin when the patient is female, or has vertebro-basilar TIAs, or continues to have TIA inspite of aspirin.

Warfarin is not indicated unless the TIAs continue inspite of antiplatelet agents, or when cardiogenic emboli are suspected. Some patients, especially those with vertebro-basilar TIAs, appear to have less attacks when on warfarin than when on antiplatelet agents. However the effectiveness of warfarin in the long term prevention of stroke remains unproven. A reasonable approach would be to give warfarin for six months to a year, then check the patency of the basilar artery using transcranial doppler or magnetic resonance angiography. If the artery is not stenosed, then warfarin may be tailed off and replaced by ticlopidine.

The work up should include doppler studies of carotid arteries to screen for severe stenosis of 70% or more in the common carotid artery or internal carotid artery in the neck. Endarterectomy may be considered for such patients[13] as well as those with 50% stenosis and large ulcer, or those who failed medical treatment. Transcranial doppler sonography may detect stenosis of the proximal segments of intracranial arteries as well as the distal portion of the internal carotid artery and is particularly useful in monitoring during the acute stage (to identify haemodynamically significant stenosis of major extra- and intracranial vessels), and in the follow-up of results of surgical treatment such as carotid endarterectomy or superficial temporal-middle cerebral artery bypass. Patients with asymptomatic stenosis of the carotid artery can have the progress of the lesion followed with repeat ultrasound studies.

Angiography is usually not necessary unless the patient is young or when carotid endarterectomy is being considered. Angiography carries a risk of stroke (up to 4%).

Extracranial-intracranial surgical bypass has not been shown to reduce incidence of stroke[14], and is now only done in special circumstances such as aneurysm surgery, moya-moya disease or when a severely stenosed middle cerebral artery causes TIA inspite of medical treatment.

The quality of intravenous digital subtraction angiography has improved considerably. It may be a useful alternative to conventional angiography in the future. Magnetic resonance angiography appears promising. It has not yet replaced angiography and is mainly used to select patients for angiography.

As part of the management of TIA or stroke, patients should be advised to reduce stroke risk with control of hypertension and diabetes, to stop smoking and control their diet.

Conclusion

We have in recent years seen an increase in understanding the stroke syndrome. This has led to more active and earlier interven-

tion in treatment. With present methods of diagnosis, treatment and rehabilitation the pessimistic attitude towards stroke has slowly given way to a new hope for the stroke patient.

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