Unexpected outcome (positive or negative) including adverse drug reactions

Watershed infarcts in a patient after carotid endarterectomy

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Summary

We report a case that developed watershed infarcts after undergoing carotid endarterectomy. Our patient had been having episodes of transient ischaemic attacks before admission. Carotid Doppler scan before admission showed severe unilateral carotid stenosis on left side. She underwent a carotid endarterectomy procedure. Her continuous blood pressure monitoring stayed normal during and after the procedure. One and half hour after the procedure, she developed right arm weakness. CT scan later revealed watershed infarcts in left cerebral hemisphere. The pathogenesis of watershed infarcts remain debated despite haemodynamic and embolic mechanisms being postulated.

BACKGROUND

Carotid endarterectomy is the surgical procedure employed to remove atherosclerotic plaque from the carotid arteries for stroke prevention. Serious complications include death, stroke and myocardial infarction. The incidence of these complications varies with various preoperative factors including recent history of ipsilateral stroke or contralateral carotid stenosis of more than 70%.^{1 2} Cerebral hyperperfusion can occur in 2–3% of cases which can progress to cerebral haemorrhage in 0.2–0.8%.³ Other complications include hypotension, hypertension, wound haematoma, infections and false aneurysm, cranial nerve damage and Horner's syndrome.

We report a case of a woman with unilateral severe symptomatic carotid stenosis (90–95% on left and 30–40% on right) who underwent carotid endarterectomy. The procedure was uneventful with normal continuous blood pressure recordings during and after the procedure. Patient developed right-sided hemiparesis after the procedure and CT scan of the head revealed cortical and deep watershed infarcts involving left hemisphere of brain only (figure 1 and 2). Although haemodynamic compromise and embolic mechanism are postulated in development of the watershed infarcts, their pathogenesis remains debated.⁴

CASE PRESENTATION

A lady in her late seventies was admitted for elective carotid endarterectomy. She had been having recurrent transient ischaemic attacks for some time prior to admission. Her medical history included ischaemic heart disease, peripheral vascular disease, type 2 diabetes mellitus, diabetic peripheral neuropathy, osteoarthritis and gastro-oesophageal reflux disease. Her medications at the time of admission were carvedolol, lansoprazole, paracetamol, simvastatin, losartan, solifenacin and subcutaneous insulin.

INVESTIGATIONS

Carotid Doppler imaging performed preoperatively showed 90–95% stenosis on left side and 30–40% stenosis on right side. Her haemoglobin was 8.3 gm/dl, white cell count was $9.3 \times 10^9/l$, platelets were $357 \times 10^9/l$, serum

sodium was 128 mmol/l, potassium was 4.8 mmol/l, urea was 4.4 mmol/l and creatinine was 37 mmol/l.

TREATMENT

She was taken to the theatre at 09:35 h in the morning and had carotid endarterectomy and patch angioplasty, under general anaesthesia, performed by an experienced surgeon. The procedure went uneventful. The continuous blood pressure monitoring records from theatre were in normal limits for her at all times and around 135/60 mm Hg. She was then taken back to recovery area at noon. The continuous blood pressure monitoring done in the recovery room was also noted to be normal with no drop in blood pressure. Postprocedure she was feeling well with no active complaints initially, until one and a half hour when she developed weakness in her right arm. The carotid duplex scan revealed emboli material in the

Figure 1 Cortical watershed infarcts in left cerebral hemisphere.

Figure 2 Cortical and deep watershed infarcts in left cerebral hemisphere.

left internal carotid artery. She was immediately taken back to the theatre at 15:35 h and underwent left sided carotid embolectomy/thrombectomy.

OUTCOME AND FOLLOW-UP

After the procedure, she was noted to have a dense right-sided hemiplegia with the power of 0/5 in arm and 1/5 in the leg on Oxford scale of muscle strength grading. Along with this she developed expressive aphasia and right-sided upper motor neuron type of facial weakness. A CT scan was performed which revealed areas of low attenuation consistent with watershed infarcts in anterior and middle cerebral artery territory. Please see the attached figures. She was started initially on aspirin 300 mg daily which was later changed to clopidogrel 75 mg daily for secondary stroke prevention. She was transferred to inpatient rehabilitation unit for further rehabilitation.

DISCUSSION

Infarcts which involve the junction of the distal fields of two non-anastomosing arterial systems are called watershed infarcts.⁴ Classic neuropathological studies⁵ describe two distinct supratentorial watershed areas referred to as cortical watershed (CWS) area and internal watershed (IWS) area. The CWS area is in the cortex between territories of anterior cerebral artery, middle cerebral artery and posterior cerebral artery. The IWS area is in the white matter along and slightly above lateral ventricle, between deep and superficial arterial system of middle cerebral artery or between superficial arterial system of anterior and middle cerebral arteries.

Although the pathological,^{6 7} and imaging characteristics,^{8–10} of watershed infarcts are well-described, their pathogenesis remains debated.⁴ Haemodynamic failure is classically thought to cause watershed infarcts in internal carotid artery disease.^{9–12} Susceptibility of the watershed areas is thought to result from their situation of 'distal field', where perfusion pressure is lowest.¹³ This

interpretation is further supported by radiological studies showing that watershed infarcts distal to internal carotid artery disease are more likely with a non-competent circle of Willis.^{14–16} In contrast, embolism from internal carotid artery preferentially affects the stem and large branches of the middle cerebral artery, producing carotid 'wedge shaped' and/or deep striato-capsular infarcts.^{14–16}

Despite widely prevalent interpretation that hypotension leads to watershed infarcts, several pathological reports emphasise their association with micro embolism arising from unstable carotid plaques or from the stump of an occluded internal carotid artery. Jorgensen and Torvik,¹⁷ and Torvik and Skullerud,⁶ first reported that most of the occlusion observed in the leptomenigeal arteries over watershed infarcts distal to internal carotid artery occlusion resulted from micro emboli occluding the terminal vascular field, rather than being secondary to slowing of the cerebral blood flow. Beal *et al*,¹⁸ reported a patient in whom arm paresis developed after multiple transient ischaemic attacks distal to ulcerative carotid plaque and who was found at autopsy to have multiple pial arteries occluded by cholesterol emboli in the border zones. Pollanen and Deck¹⁹ reported three cases in which embolisation of thrombotic material (from the heart in two cases and from internal carotid artery in one) caused CWS infarcts. Furthermore, Masuda *et al*²⁰ found atheromatous embolism to cause CWS infarcts by occlusion of the terminal cortical branches with small emboli (50–300 μ m) mostly composed of cholesterol crystals, whereas, territorial infarcts were related to large fibrin emboli. Importantly, there is experimental evidence that small thrombi travel preferentially to watershed areas because of their distinctly small size.²¹ Interestingly, cerebral amyloid angiopathy has been recently been proposed as a risk factor for micro infarcts in CWS areas.²² Though these studies mainly apply to CWS infarcts, Lammie GA,²³ suggested that ischaemic lesion observed in the IWS area may also involve an embolic mechanism, either cardiac or artery to artery.

Isabelle and Jean-Claude⁴ concluded in their study that evidence strongly favours a haemodynamic mechanism for IWS infarcts; however, the relationship between CWS infarcts and haemodynamic compromise appears more complicated. Thus, although severe haemodynamic compromise appears to underlie combined cortical and IWS infarcts, artery to artery embolism may play an important role in isolated CWS infarcts.

Learning points

- ▶ Although haemodynamic compromise and embolic mechanism are postulated in development of watershed infarcts, their pathogenesis is still debated.
- ▶ Our case did not have severe carotid stenosis on contralateral side and did not suffer from hypotension during or after the procedure which could lead to hypoperfusion and infarction, but still developed unilateral (left side only) watershed infarcts.

Competing interests None.

Patient consent Obtained.

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