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ABSTRACT

Background: Even after the failure of EC-IC bypass trial, EC-IC bypass can help many patients in preventing future stroke. Here we present a case series of patients with cerebral ischemia from different etiological modes who underwent EC-IC bypass with positive end results.

Methods: Patients in these cases series with TIA/stroke/recurrent stroke were evaluated clinically for the history of TIA or recurrent/hemodynamic TIA (in rest or during work) or progressive hemiparesis/aphasia/visual disturbances or sudden hemiplegia/hemiparesis/aphasia with subsequent significant (days to a week) recovery. MRI of the brain was done in ischemic protocol in all cases. To see the arterial pathology dynamic CTA was also done in all cases except one case. DSA was done in 03 cases. When clinical features, cerebral ischemia on MRI and arterial stenosis/occlusion on angiogram were concordant with each other, only then cerebral revascularization was done. After bypass, all patients were followed up regularly. All recorded data were reviewed retrospectively.

Results: Total no. of cases were 08. The most common presentation was hemiparesis. Etiologies were infective thrombosis of ICA, orbital cellulitis, thrombosed giant ICA aneurysm, single & multiple vessel occlusion and MCA stenosis. High flow EC-IC bypass was done in one case. STA-MCA bypass was done in rest of the cases. All patients were ambulant with static neuro-status without new stroke till last follow up. All bypasses were patent and functioning till last follow up (clinical, Doppler/Imaging).

Conclusion: In carefully selected cases cerebral revascularization in ischemic conditions can result positive outcome.

INTRODUCTION

After introduction by Yasargil in 1967, cerebral revascularization by EC-IC bypass has become an indispensable tool for managing patients with hemodynamic ischemia, or for managing patients with complex aneurysms or skull base tumors; that are not amenable to radical resection; because of major vascular involvement. [2,14] Patients with

Keywords
cerebral revascularization,
EC-IC bypass,
ischemic conditions,
different etiologies



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hemodynamic ischemia have an annual stroke rate of 25%, which increases by 2% every year.[14] They can develop a fatal stroke. This category also includes Moya moya disease. [14,18] After failure of EC-IC bypass trial, neurosurgeons were in search of cases where EC- IC bypass would help the patients in preventing future stroke and neurological improvement. Here we present a case series of patients with cerebral ischemia from different etiological modes underwent EC-IC bypass with positive end results.

METHODS

Patients in these cases series with TIA/stroke/recurrent stroke were evaluated clinically for history of TIA or recurrent/hemodynamic TIA (in rest or during work) or progressive hemiparesis / aphasia / visual disturbances or sudden hemiplegia / hemiparesis / aphasia with subsequent significant (days to week) recovery. Permanent hemiplegia cases were not included in this case series. Then the cases were evaluated neuro-radiologically for cerebral ischemia with or without infarct/s and possible cerebral revascularization by EC-IC bypass. CT scan of head was done to exclude hemorrhage and other pathology such as tumor. MRI of brain was done in ischemic protocol (All images including DW, ADC, PW, DTI and MRA&MRV including neck vessels) to see cerebral ischemic zone/s (DW& PW mismatch), cortico-spinal tract & other major tracts and intracranial or extracranial arterial stenosis. To see the arterial pathology dynamic CTA was also done in all cases except one case. DSA was done in 03 cases. When clinical features, cerebral ischemia on MRI and arterial stenosis/occlusion on angiogram were concordant with each other, only then cerebral revascularization by EC-IC bypass was done. After bypass all patient were followed up regularly (clinically and radiologically). All recorded data were reviewed retrospectively. All bypass operation was done from January 2015 to March 2018

REPRESENTATIVE CASES

Case 1 (Table 1, 2; Figure 1 - a, b, c)

A 38 years old male presented with headache, vomiting, dropping of left eyelid and visual disturbance. Left sided visual acuity reduced to finger count with left sided complete ophthalmoplegia. CT scan of head showed pan-sinusitis with skull base osteitis. MRI of head showed

pan-sinusitis with both cavernous sinus involvement and right parietal infarct (Figure 1a-A,B&C). He underwent diagnostic endoscopic rhino-sinuscopy, which showed rhino-sinusitis involving all sinuses & nose with pseudo-membranes. Biopsy reported inflammatory sinusitis. Fungal and tubercular cultures were negative. Routine bacterial culture-revealed methicillin resistances *Staphylococcus aureus* (MRSA). Then he was put on high dose injectable antibiotic for three week that resulted significant radiological improvement (Figure 1a-D,E&F) and then patient was discharged with oral antibiotic for three month.

Three weeks after discharge he again presented with altered level of consciousness and right sided hemiplegia (UE>LE, Muscle power in right UE MRC grade 1/5 and in lower limb 2/5) and motor aphasia. MRI of head showed resolving rhino-sinusitis with patchy infarcts and ischemic zone involving fronto-parieto-occipital zones especially on left side and old infarct on right parietal area (Figure 1b). MRA showed bilateral complete occlusion of ICA and whole brain was perfused by basilar artery (Figure 1c-A&B). On urgent basis the patient underwent left-sided STA-MCA bypass.

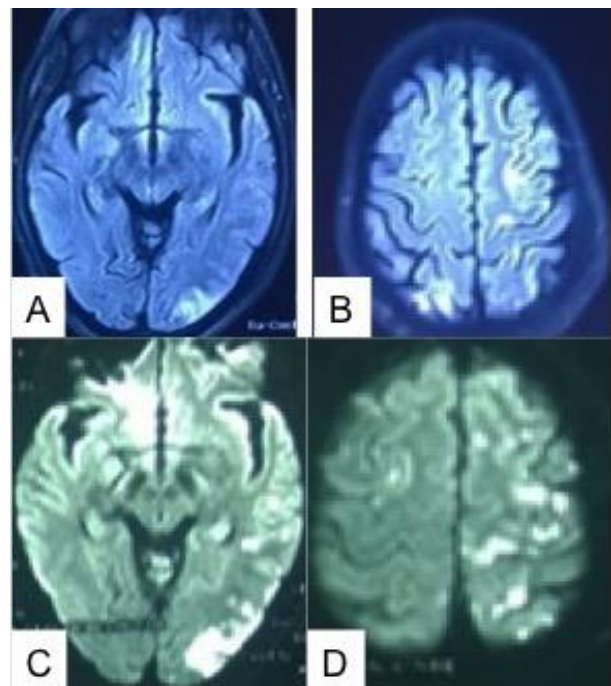


Figure 1.

Operation

Under GA the patient was positioned typically supine

with the head turned more than 60°. At this point, we used digital palpation technique and a handheld Doppler probe to map out the course of the STA both frontal and parietal branches.

The incision was started at the level of the zygoma and carried up to near midline behind the hairline. Both branches were procured up to superior temporal line very carefully to avoid thermal damage or avulsion injury for branches. Papaverine solution and plain local anesthetic agent (2% lidocaine) was used to irrigate the STA for prevention of vasospasm. A mini pterional craniotomy was done very carefully (not to damage the procured STA). After durotomy a small posterior Sylvian split was done to find out a suitable M3 as a recipient vessel for bypass. Among the frontal and parietal branch the suitable and larger frontal branch was used to make a STA-MCA anastomosis. After bypass patency was checked clinically and with micro-doppler. Dura was loosely closed around the STA (not water tight). Along the temporal margin of bone flap a portion was removed so that the STA would not be kinked or compressed by the bone. Mini plates and screws were used to fix the bone flap. Rest of the wound was closed accordingly without drain.

Postoperative course

Patient recovered well from anesthesia. In postoperative days the patient recovered hemiparesis quickly. By the end of 7th POD patient became ambulant. By the end of 04 weeks after operation he returned to his professional work and muscle power on right side body improved at the level of MRC grade 4+/5. Post-operative CT scan showed no hematoma or new infarct. CTA showed patent STA-MCA bypass on left side (Figure 1c-C&D).

Case 2 (Table 1, 2; Figure 2 - a, b, c)

A 55 years old policeman presented with recurrent occasional episode of fall during walking without loss of consciousness or convulsion and unable to move right side of body with aphasia lasting for 7-10 minutes. The frequency of events increases last few months and reached 3-5 times/day for last few days before presented to us. He was on adequate anti platelet therapy. He was a smoker but non-diabetic and non-hypertensive. CT scan of head showed multiple infarcts especially in left cerebral hemisphere. CTA showed absent both VA and left ICA (Figure 1a) with scarcity of left. MCA, PCA and

posterior fossa vessels. MRI of brain showed multiple old infarcts with ischemic areas especially in left hemisphere and ischemic zone in left PICA area. He was advised for urgent cerebral revascularization. But the patient developed left PICA infarct 06 hours before the scheduled 'urgent' operation (Figure 2b-A&B). In this situation we proceeded for cerebral revascularization by left-sided CCA-RA-MCA high flow bypass by keeping in mind that posterior fossa decompression might be needed at any time.

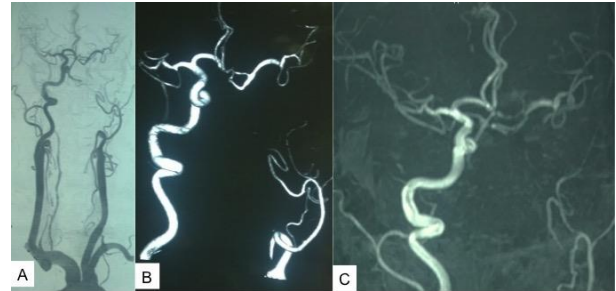


Figure 2a.

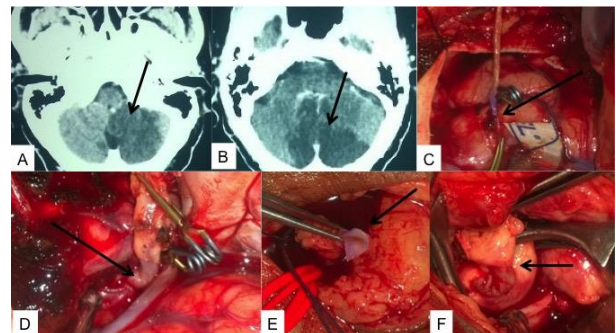


Figure 2b.

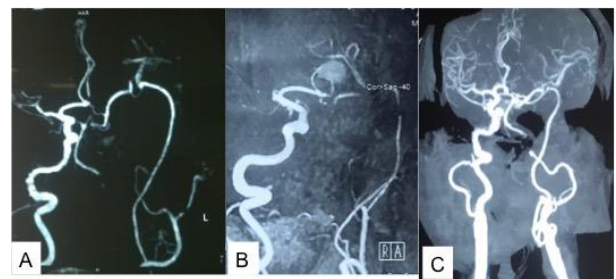


Figure 2c.

Operation

Under general anesthesia with endotracheal intubation patient was placed in supine position. Head was fixed with 3-pin head holder with neck extension and head turning to the opposite (right) side (30°). Head end of the table was elevated (20°).

Eye, ear, pressure points and nerves areas were protected. Left upper limb placed on a side 'limb rest' in extended elbow, 30° abducted from the trunk in supine for radial artery procurement. After preparation, left front of the forearm, right side of the neck and left pterional areas were draped properly.

With longitudinal incision, radial artery was harvested from brachial bifurcation at elbow to wrist (20 cm). The artery was distended with intraluminal injection of heparin and papaverine mixed normal saline. Then the artery was kept in heparin and papaverine mixed normal saline. The forearm wound was closed with a drain.

A curve incision on the left side of the neck was made from the tip of mastoid and extended downward & medially 2cm posterior to the angle of mandible to the mid line. After cutting platysma and investing deep fascia sternocleidomastoid muscle was retracted laterally. With further dissection posterior belly of digastric muscle, hypoglossal nerve, internal jugular vein, common carotid, internal carotid and external carotid artery with its branches were identified.

A left-sided pre-coronal post hairline curvilinear incision was made and superficial temporal artery (STA) and its parietal branch was procured and prepared for STA-MCA insurance bypass as donor artery. A temporally extended pterional craniotomy was done. Temporal bone was removed down to the middle fossa floor. In the cervical wound, a blunt index finger dissection was made in between the digastric muscle and hypoglossal nerve upward and superiorly to styloid process and then finger dissection was continued upward, medially & anteriorly to lateral pterigoid plate. A curved medium sized artery forceps was passed from middle fossa floor to the fingertip and with finger guidance the arterial tip was brought out into the cervical wound and then A 26Fr thoracostomy tube was passed from cervical wound to the middle fossa floor. Radial artery (RA) graft was passed from middle fossa floor to cervical wound through the tube. With stabilization of both ends of RA graft thoracostomy tube removed. RA graft was made twist free by injecting heparinized solution into the lumen.

After durotomy a STA -MCA (Temporal M4) 'insurance bypass' was done with 10/0 nylon and checked for patency and function with micro Doppler (Figure 2b-C). After Sylvian dissection temporal M2

was identified and prepared for bypass. Cranial end of RA graft was also prepared for bypass and the RA graft and temporal M2 bypass was made after systemic heparinization with 3000 unit of injection heparin (Figure 2b-D). The patency of anastomosis was checked by retrograde flow of blood through the caudal end of RA graft in cervical wound.

With the control of common carotid artery (CCA) an anastomosis was made between caudal end of RA graft and CCA (Figure 2b-E&F). The patency and flow through the anastomoses and RA graft were checked with micro doppler. Cervical wound and craniotomy wound were closed with drains.

Postoperative course

Postoperatively, he was on tab. Aspirin and inj. Heparin. CT scan on 1st postoperative day (POD) showed no infarct or any gross hematoma (Figure). CT angiogram on second POD showed left ECA-RAG-M2 bypass with pre-operative PICA infarct without further swelling. Post operatively he became non-communicable and he used shout with inappropriate & slang words especially at night and did not want to take food. Gradually these symptoms improved within three weeks and became communicable but improvement of intellectual functions was slow and incomplete. He became continent by three weeks. Within one week he could stand and walk though there was some instability. He became fully ambulant without any cerebellar dysfunction by three months. By the end of one year after operation his intellectual and cognitive function returned to near preoperative level and he returned to his job without any further hemodynamic TIA. CT scan and CTA after one year showed patent bypass without any new infarct.

RESULTS

Total no. of cases were 08. Six were male and 02 were female. Age range was 25-57 (average 38 years). Follow up period was 12-48 months. Details of all cases were shown in Table 1 & 2 (Figure 1-7).

Most common presentation was hemiparesis. Infection was the etiology of thrombosis of cavernous ICA in two cases. In one case orbital cellulitis spread in CS and ICA with aneurysm was thrombosed (Figure 6-A,B&C). In the other cases pan rhino-sinusitis (of MRSA) spread to both cavernous sinus and both ICA was occluded. In one interesting case giant partially thrombosed ICA bifurcation

aneurysm thrombosed totally with distal ICA, A1 and M1 (Figure 7). Acute thrombosis of ICA with aneurysm in CS occurred in two female cases where both developed hemiparesis and one was 3 months pregnant. In one case there was intractable TIA with impending major stroke where whole brain was supplied by only right sided ICA and he developed PICA infarct 12 hours before the 'scheduled urgent' revascularization operation; Only high flow EC-IC bypass was done in this case (Figure 2a,b&c). Post operatively he developed 'behavioral, intellectual and? psychogenic' symptoms that recovered slowly.

STA- MCA bypass was done in rest of the cases. MCA stenosis was the etiology in two cases; one was of 27 years of age (Figure 3). Average ischemic time was 28 minutes (range 25-35 minutes). There was no clamp related infarction. In one case patient developed postoperative insulo-frontal infarct that not related to temporary clamping (Figure 6-C, D & F). All patient improved neurologically. All patient were ambulant with static neuro-status without new stroke/TIA till last follow up. All bypass were patent till last follow up (clinical, Doppler/Imaging).

Table 1. Particulars of cases (age, sex, clinical presentations, investigations and image finding/s).

No	Age/ sex	Presentation/s	Investigation/ s	Image finding/s
1. (Figure1)	38/M	Headache, vomiting, dropping of left eyelid, visual disturbance. Left sided visual acuity reduced to finger count with left sided complete Opthalmoplegia, Three weeks later (on antibiotics): presented with altered level of consciousness and right sided hemiplegia (UE>LE, Muscle power in right UE MRC grade1/5 and in lower limb 2/5) and motor aphasia	CT MRI Diagnostic sino-nasal endoscopy MRI MRA	Pansinusitis with skull base osteitis pansinusitis with both cavernous Sinus involvement and right parietal infarct Rhino-sinusitis involving all sinuses with pseudomembranes *Biopsy-Inflammatory sinusitis *Culture: <i>Fungal-Negative, Tubercular-Negative, Routine bacterial culture- Multiple antibiotic resistance Staphylococcus Areus</i> Resolving rhino-sinusitis with patchy infarcts and ischemic zone involving fronto-parieto-occipital zones especially on left side. Old infarct on rt. parietal area. Bilateral complete ICA block (whole brain is perfused by basilar artery)
2. (Figure2)	55/M	Recurrent episode of fall during walking and unable to move lt. side of body with aphasia,5-8 times/day before presentation even with	CT scan CTA	Multiple infarcts specially in lt. hemisphere Absent both VA and lt. ICA. Scarcity of lt. MCA, PCA and posterior fossa vessels

		adequate anti platelet therapy	MRI MRA	Multiple old infarcts with ischemic areas especially in Lt hemisphere. Ischemic Lt PICA area. The pt. developed Lt. PICA infarct 12 hours before the scheduled 'urgent' operation.
3. (Figure3)	27/M	H/O TIA (recurrent hemiparesis and aphasia), sudden rt. Hemiplegia (MRC grade-2/5), and global aphasia	MRI CT & CTA DSA	Cerebral infarcts and ischemic zones in left hemisphere especially Parieto-occipital lobe. CT-Cerebral infarcts and ischemic zones in left hemisphere. CTA- left M1 bifurcation stenosis. Scarcity of Lt. MCA vessels Left M1 bifurcation stenosis/occlusion with delayed filling of MCA territory.
4. (Figure4)	45/M	Headache, left hemiparesis (UE>LE, Grade1/5) and motor aphasia. H/O TIA. One-week later motor power on left side 2/5.	CT CTA MRI MRA	Right parieto-occipital and right periventricular (corona radiata) infarcts. Complete occlusion of right-sided ICA from neck to ICA bifurcation with decrease vasculatures on right MCA zone. Right parieto-occipital and right periventricular (corona radiata) infarcts/ischemia with diffusion -perfusion mismatch. Tractography showed intact major tracts. Complete occlusion of right sided ICA from neck to ICA bifurcation with decrease vasculatures on right MCA zone
5. (Figure 5)	27/F	Sudden headache, eyeache, vomiting, rt. ptosis with complete ophthalmoplegia, Lt. hemiparesis, 2 nd trimester pregnancy	MRI & MRA DSA	MRI-right cavernous sinus thrombosis with 'target' sign. MRA-occlusion of rt. ICA with giant CS ICA aneurysm (thrombosed). Scarcity of rt. MCA vessels Rt. ICA occlusion with scarcity of blood flow in rt. MCA
6. (Figure 6)	25/F	Fever, Lt. proptosis, headache, eyeache, vomiting, Lt. ptosis with complete ophthalmoplegia, rt. hemiparesis	CT scan MRI & MRA	CT-Lt eye proptosed otherwise normal MRI-Lt cavernous sinus thrombosis with 'target' sign. Lt eye was proptosed.

				MRA-occlusion of lt. ICA with CS ICA aneurysm (thrombosed). Scarcity of lt. MCA vessels with M1 narrowing
7.	57/M	H/O TIA with motor dysphasia and hemiparesis. Rt. Hemiparesis (3/5) and global aphasia	CT scan & CTA MRI MRA	CT-left ganglio- capsular, periventricular, parietal and frontal small infarct/ischemias. CTA- left M2 (upper trunk occlusion) and M2(lower trunk stenosis) Left ganglio- capsular, periventricular, parietal and frontal small infarct/ischemias. Left M2 (upper trunk occlusion) and M2(lower trunk stenosis)
8. (Figure7)	35/M	Initially headache, visual disturbance. Later visual problem improved with progressive left. Spasticity and hemiparesis (3/5)	CT & CTA MRI CT & CTA (4month later) DSA	Right ICA bifurcation Giant partially thrombosed aneurysm Right sided giant partially thrombosed aneurysm Aneurysm size decreased to less than half. Anterior corona radiata infarct. No aneurysm on CTA with occlusion of rt. ICA bifurcation. Scarcity of rt. MCA vessels Non-visualization of right ICA bifurcation, A1 and M1 with aneurysm. Delayed filling of right MCA territory through cortical anastomoses. Right ICA only supplied the right PCA through PCom

[M-male,F-female, H/O-history of, Rt/rt-right, Lt/lt-left, UE-upper extremity, LE-lower extremity, TIA-transient ischemic attack, MRC-medical research council, CT-computed tomography, CTA-CT angiogram, MRI-magnetic resonance imaging, MRA- MR angiogram, DSA-digital subtraction angiogram, ICA-internal carotid artery, MCA-middle cerebral artery, PCA-posterior cerebral artery, Pcom-posterior communicating artery, CS-cavernous sinus, PICA-posterior inferior cerebellar artery, VA-vertebral artery]

Table 2. Etiology of infarct/ischemia, indication of operation, surgical treatment, complication/s and outcome of bypass.

No	Etiology	Indication/s	Surgical treatment	Complication	Outcome
1. (Figure 1)	Bilateral complete occlusion of ICA due infective inflammatory involvement of cavernous sinus	Acute on chronic hemodynamic ischemia of whole brain specially ICAs territory.	Left STA-MCA bypass	None	Ophthalmoplegia, hemiparesis and aphasia recovered completely. Rhinopansinusitis resolved completely

2. (Figure 2)	Chronic occlusion of three out of 04 intracranial arteries caused hemodynamic stroke and ischemia	Chronic and hemodynamic ischemia specially left hemisphere and cerebellum	Radial graft high flow EC-IC (CCA-RA-M1) bypass with insurance STA-MCA bypass	Early post operative period-behavioral changes	Behavioral changes recovered. No hemodynamic TIA.
3. (Figure 3)	Acute occlusion of M1 bifurcation on Chronic MCA stenosis	Acute on chronic ischemia	Left STA-MCA bypass (parietal)	None	Almost near total recovery of neuro-deficit.
4. (Figure 4)	Complete occlusion of Right ICA from neck.	Ischemic right cerebral hemisphere (with infarcts) specially MCA territory	Right STA- MCA bypass	None	Hemiparesis improved (MRC grade-4/5). Aphasia improved to dysphasia
5. (Figure 5)	Sudden ICA occlusion with occlusion of giant aneurysm in CS ICA	Ischemic hemiparesis (with CS thrombosis)	STA-MCA bypass with decompression of CS	None	Hemiparesis and ophthalmoplegia recovered completely. Normal delivery of healthy baby
6. (Figure 6)	Sudden ICA occlusion with occlusion of aneurysm in CS ICA due to orbital cellulitis spread in to lt. CS	Ischemic hemiparesis (with infective CS thrombosis)	STA-MCA bypass with decompression of CS. C/S of CS purulent content Injectable antibiotic (long term)	Anterior insular cortex and Broca's area infarct-motor dysphasia	Hemiparesis, dysphasia, ophthalmoplegia recovered completely
7.	Acute occlusion of M2 on chronic stenosis	Acute on chronic ischemia	Left double STA -MCA bypass (parietal and temporal)	None	Motor recovery-near normal, Global aphasia converted to sensory aphasia
8. (Figure 7)	Thrombosis of giant aneurysm with progressive occlusion of ICA bifurcation	Right MCA territory ischemia	Right STA- MCA bypass	None	Hemiparesis improved (MRC grade-4/5).

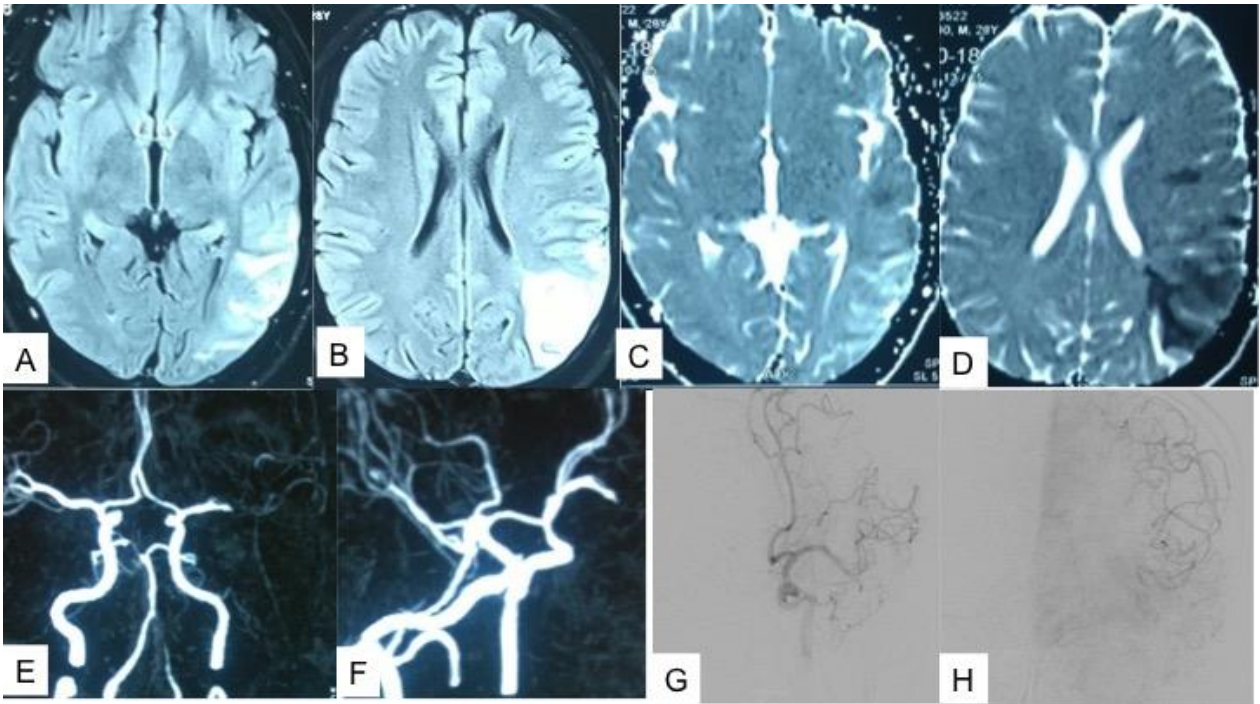


Figure 3a.

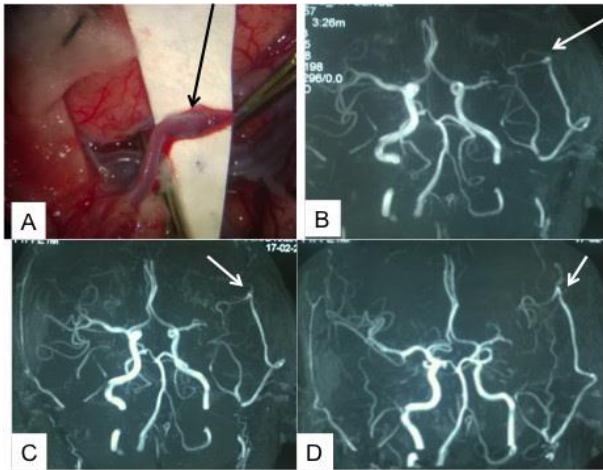


Figure 3b.

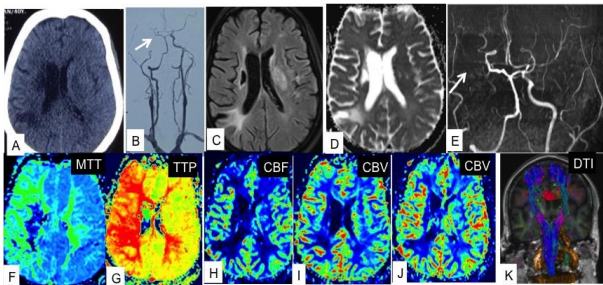


Figure 4a.

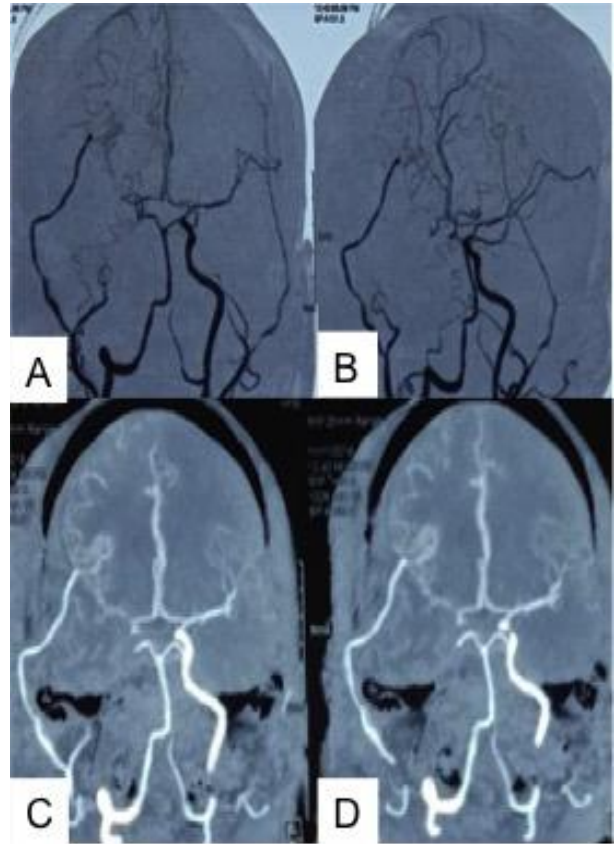


Figure 4b.

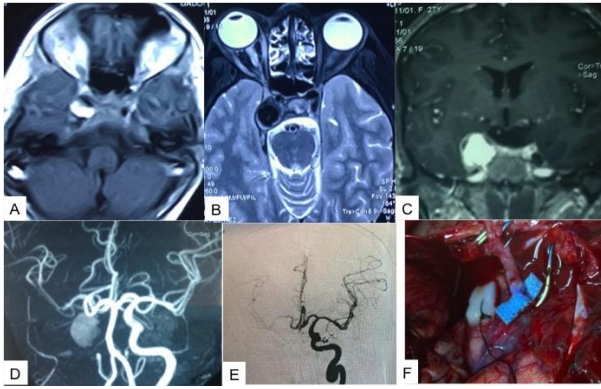


Figure 5a.

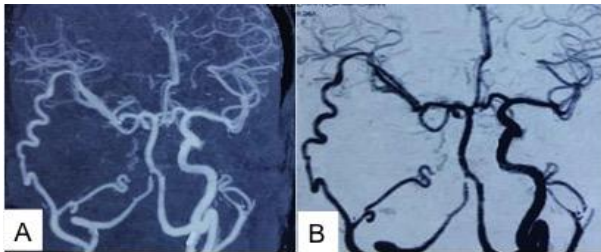


Figure 5b.

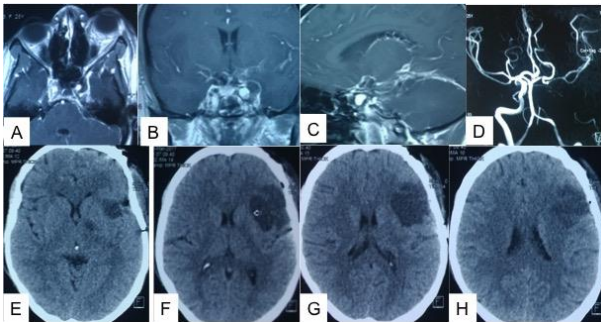


Figure 6.

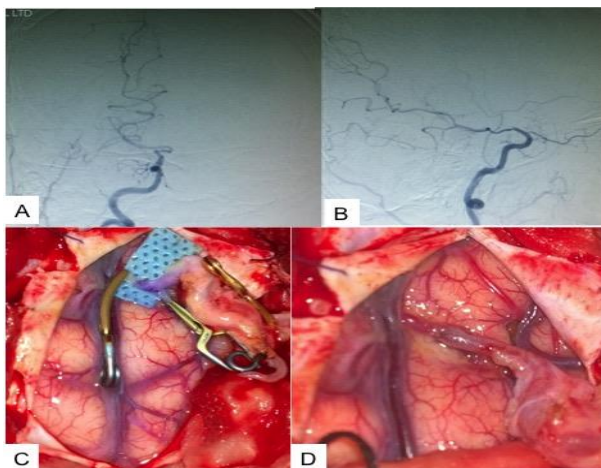


Figure 7.

DISCUSSION

In 1961, Pool and Potts [2,9] first attempted cerebral revascularization with a synthetic graft using a plastic tube to create a superficial temporal artery (STA) to anterior cerebral artery shunt but angiography showed thrombosed tube though patient recovered and survived. In 1963, Woringer and Kunlin [11] performed the first extracranial to intracranial (EC-IC) bypass of the common carotid artery (CCA) – intracranial (IC) internal carotid artery (ICA) using a saphenous vein (SV) graft but patient did not survive, while the graft was patent on autopsy.

In 1967, Yaşargil performed the first EC-IC bypass in a patient with an occluded ICA.[10] In 1972 Yaşargil also started STA-middle cerebral artery (MCA) bypass for moyamoya disease. In 1971 Lougheed did the first EC-IC bypass using an SV graft, while in 1978 Ausman performed EC-IC bypass using a radial artery graft. In the 1970s, Sundt et al.[3] and others performed posterior circulation revascularization to treat steno-occlusive disease, vertebrobasilar insufficiency, and unclippable complex aneurysms.

EC-IC bypass study (1977-1985) [15] evaluated the result of EC-IC bypass as a means to decrease the subsequent stroke rate for the treatment of “symptomatic atherosclerotic lesions of the ICA and/or MCA.” But the study failed to show significant difference between the EC-IC bypass group and medically treated group. The study identified two important subgroups that appeared to do EC-IC bypass; 1. Patients with severe MCA stenosis and 2.those with the persistence of ischemic symptoms in known ICA occlusion.

IC or MCA atherosclerosis results to an ischemic cerebrovascular event through: (1) Hypo-perfusion, (2) thrombosis at the site of stenosis, (3) thromboembolism, and (4) direct occlusion of small perforating vessels. [9,16]

The clinical presentation may vary from an acute ischemic deficit to intermittent neurological symptoms. The pathophysiologic changes of chronic hypo-perfusion/ischemia have been categorized into three stages-

Stage 0: Normal hemodynamics; Stage 1: Reflex vasodilation in response to inadequate collaterals and a falling perfusion pressure with resultant increases in cerebral blood volume and prolongation of mean transit time, but with preservation of cerebral blood flow (CBF) and normal oxygen extraction fraction (OEF); Stage 2: Misery perfusion in

response to cerebral perfusion pressure falling below the range of auto-regulatory capability exemplified by falling CBF and increasing OEF and maintenance of the cerebral metabolic rate of oxygen (CMRO₂). [9]

Progressive MCA stenosis, severe stenosis (>70% stenosis), female gender, National Institutes of Health Stroke Scale score >1, concurrent diabetes, borderline body mass index values, hyperlipidemia, white ethnicity, and the presence of hemodynamic stenosis increases the risk for stroke. [9]

The beginning assessment of transient ischemic attack (TIA) or ischemic stroke should include computed tomography (CT) of head to exclude the possibility of IC hemorrhage (and rarely other pathologies such as cavernous sinus mass, giant thrombotic aneurysm etc.) and estimate the extent of ischemic change in consideration of thrombolysis. Diffusion-weighted (DW) magnetic resonance imaging (MRI) with magnetic resonance angiography (MRA) including neck vessels, and perfusion studies can be subsequently obtained to better delineate the extent of infarct and/ ischemia as well as provide more information for further management strategies. [16] TCD, angiography, MRA, and computed tomographic angiography [CTA] or parenchymal perfusion (CT perfusion, positron emission tomography [PET], single-photon emission computed tomography [SPECT], and magnetic resonance perfusion studies are the main neuro-radiological evaluations to see the intra cranial flow related pathology. MRA and CTA both provide excellent detail regarding the caliber of vessels; although, MRA has been shown to overestimate the degree of stenosis in some cases. These noninvasive techniques were sufficiently accurate to exclude more than 50% stenosis, but further confirmatory studies were needed to characterize the stenosis and digital subtraction angiography (DSA) is the gold standard. Perfusion studies [16] (include PET scans, SPECT scans, xenon CT perfusion studies, CT perfusion, and perfusion MRIs) permit the extrapolation of CMRO₂, OEF, and CBF and provide information on the perfusion of the brain. The Carotid Occlusion Surgery Study [17] employs measuring OEF by PET.

Since the first STA-MCA procedure was described by Yasargil, [2,18] many variations have been reported but STA -MCA bypass remains the main workhorse of a vascular neurosurgeon. Many of

these variations have been developed in dealing with complex intracranial aneurysms and skull base tumors. These variations include anastomoses between the bilateral anterior cerebral arteries; occipital artery-to-posterior, inferior cerebral artery (PICA), anterior and inferior cerebral artery (AICA). Others includes PICA to PICA, vertebral artery to PICA, STA to SCA or PCA, subclavian artery to PCA, PCA to SCA, and even a tandem occipital artery to AICA and PICA anastomoses. [9]

Complications in STA-MCA bypass are limited and include early postoperative TIA, delayed stroke, development of a pseudo-aneurysm, and wound dehiscence. High-flow bypass grafts is more prone to develop complications than low-flow STA-MCA bypass. Radial artery grafts may suffer vasospasm or intimal hyperplasia and eventually occlude. Pro-atherogenic changes can occur in SV grafts, which eventually leads to occlusion. After parent vessel occlusion thromboembolic complications are common after high flow bypass mainly due to the change in intracranial hemodynamics. Preoperative antiplatelet medications, as well as intraoperative anticoagulation, can prevent these thromboembolic events. In patients without vascular reserve, prolonged temporary occlusion times can lead to territory infarcts without changes in the neuro-monitoring. So it is important to minimize occlusion times in these patients. In longstanding perfusion deficiency, reperfusion hemorrhage may be problematic after revascularization, though the incidence is low. Other complications involve the site of graft harvests such as infection, ischemic hand, or hematoma. [9]

Here in representative case 1 (high flow bypass), there were postoperative behavioral, intellectual and cognitive dysfunctions that recovered slowly. There may be hyper-perfusion of chronically ischemic brain tissue but postoperative radiology did not show any hyper-perfusion signs. Retrogradely we thought, could we avoid such complication by giving low flow bypass.

Most of the ICA and other intracranial arterial stenosis or occlusion (acute or chronic) leading to cerebral infarct/ischemia is caused by atherosclerosis predisposing and precipitating by many factors. Other causes include dissection, vasculitis, vasospasm and Moyamoya disease. [5,13]

In this series most of the cases are unique from of etiological point. Spontaneous occlusion of

cavernous ICA was reported in the literature but such occlusion associated with cerebral ischemia was not reported.[8] Infection from paranasal sinuses or orbit usually spread into cavernous sinus but usually does not occlude the ICA; in case of fungal infection, fungus may erode the arterial wall and can cause mycotic aneurysm with distal embolism or occlusion of ICA.[4,12]

Giant partially thrombosed ICA bifurcation aneurysm case, at initial presentation patient refused operation for aneurysm then he returned with left hemiparesis with complete occlusion aneurysm as well as occlusion of parent ICA with A1 and M1 (proximally up to PCom). ICA was only supplying right PCA through PCom. Young patient with MCA stenosis recovered near to normal neurology.

In our series, in a few cases, we did revascularization on urgent basis. Nussbaum ES et al [6] did emergency EC-IC bypass in patients with acute ischemic injury, which was seemed to be safe and effective where patients were relatively young. They found bypass was successful in arresting progression of stroke, and in some cases resulted in rapid neurological improvement. Although the study on EC-IC bypass failed to show a benefit from the bypass procedure Nussbaum ES et al [7] continued to perform the operation in selected cases. Carefully selected individuals with occlusive cerebrovascular disease and persistent ischemic symptoms, despite maximal medical therapy, seem to obtain demonstrable and durable benefit from cerebral revascularization.

CONCLUSION

In this small series, cases with concordant clinical features, MRI findings and angiographic finding underwent bypass surgical procedure and we found such concordances ended up with positive result (though short term) with cerebral revascularization.

ABBREVIATIONS

ADC-axial diffusion coefficient
AICA-anterior inferior cerebellar artery
CBF-cerebral blood flow
CCA-common carotid artery
CT- computed tomography
CTA- CT angiography
CS -cavernous sinus
DW-diffusion weighted

DSA-digital subtraction angiography
ECA- external carotid artery
EC-IC extracranial-intracranial
GA-general anesthesia ICA-internal carotid artery
IC-intracranial hemorrhage
LE-lower extremity
MCA-middle cerebral artery
MRI-magnetic resonance imaging
MRA-magnetic resonance angiography
MRS-methicillin resistance staphylococcus aureus
OEF-oxygen extraction fraction
PCA-posterior cerebral artery
Pcom-posterior communicating
POD-post operative day
PICA-posterior inferior cerebellar artery
PW-perfusion weighted
PET-positron emission tomography
RA-radial artery
RAG-radial artery graft
SCA-superior cerebellar artery
SPECT-single photon emission computed tomography
STA-Superficial temporal artery
SV-shapenous vein
TCD-transcranial doppler
TIA-transient ischemic attack
UE-upper extremity
VA-vertebral artery

DECLARATION

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DISCLOSURE

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