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Recent Studies in Cerebrovascular Diseases in IndiaAnnual Stroke Masterclass 2017: the Summary of Meeting Proceedings:

Annual Stroke Masterclass 2017: The Summary of Meeting Proceedings

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The "Annual Stroke Masterclass 2017 (ASM, 2017)" focused on providing a comprehensive knowledge on medical and interventional management of ischemic stroke with specific emphasis on "How to?" handle dilemmas, workshop on vascular neuroimaging, live endovascular session, live transcranial Doppler workshop demonstration of findings in specific pathological conditions, and "hands on" Botox in spasticity. The masterclass was held at Taj Dwarka, New Delhi on the 23rd and 24th of September 2017. It was organized by the "Stroke and Neurovascular Interventions Foundation" and "Artemis Agrim institute of Neuroscience" under the aegis of Indian Stroke Association, Indian Academy of Neurology and the Society of Therapeutic Neurointervention. The conference chairmen were Dr. Sumit Singh and Dr. Vipul Gupta and the meeting was organized by Dr. Rajsrinivas Parthasarathy.

Preconference e-learning commenced in June 2017 and was conducted for a period of three months through electronic media that included more than 500 doctors with neuroscience background from across India. The meeting was highly subscribed and attended by over 300 neurologist and neurointerventionist from India and the neighbouring countries. The ASM, 2017 was granted 14.5 credit hours by the Delhi Medical Council. The meeting started at 8.30 AM on the 23rd September 2017 with "Imaging in Stroke," the topics discussed were: NCCT and CTA is the gold standard—lessons learnt. "The pitfall—I felt further imaging would have helped in selected cases" (Dr. David Liebeskind), perfusion imaging in acute ischemic stroke—lessons learnt; "When not to rely on it?"(Dr. Kenneth Butcher) and Vessel wall imaging—How to differentiate Intracranial atherosclerosis/vasculitis/moyamoya? Case scenarios! (Dr. A. R. Chatterjee). The dilemma theme was "Acute stroke: To intervene or not based on Imaging" (Dr. Jayanta Roy). The second session was on "Heart and Brain"; the theme was "In pursuit of atrial fibrillation." Is "30-day' cardiac monitoring justified?" the "pros" and "cons" (Dr. Manjinder Sandhu). The dilemma addressed was "Short (very brief) duration AF and stroke" (Dr. Rajsrinivas Parthasarathy). The next session was on non-vitamin K oral antagonists and the subject matter for discussion was "Individualizing NOAC therapy 'How to choose your drug?'(Dr Kenneth Butcher), 'Stopping the ooze' while on NOAC; How to monitor anticoagulation on NOAC therapy" (Dr. Kenneth Butcher), "Left atrial appendage exclusion—a nonpharmacological alternative?" (Dr. Manjinder Sandhu) and "Early initiation of NOAC" (Dr. Kenneth Butcher). The brain teaser themes were "Dual antiplatelet and anticoagulant—when and why? & is it safe?" (Dr. Rajsrinivas Parthasarathy) and "When NOACS don't work" (Dr. Jayanta Roy). This was followed with a debate session on thrombolytic therapy in "Acute Ischemic Stroke". The debate was on (1) tenecteplase: "the elephant in the room?" Can we ignore the evidence anymore? (Dr. Kameshwar Prasad) versus "Large body of evidence for Alteplase trumps the evidence in favor for tenecteplase" (Dr. Subhash Kaul) and (2) "M1 occlusion low NIHSS/borderline core/delayed window period—One should consider intervention in this group of patients' (Dr. Dheeraj Khurana) versus "I will employ a conservative approach as they are less likely to benefit" (Dr. Rohit Bhatia). The problem scenarios discussed were "Reversing NOAC effect prior to thrombolysis (Dr. Kenneth Butcher)," "MI and Stroke—How to save the brain and the heart? (Dr. Binay Kumar)," "Sherlock Holmes and Doctor Watson in Scarlet: the Dooms day; a case of aortic dissection and stroke (Dr. Srinivasan Paramasivam)," "Thrombolytic therapy and ICH—What can one do?" "Scientia potential est" and "Legs giving way—Is it the Brain or the Spine; cranial and spinal dural arteriovenous fistula presenting with spinal cord ischemia" (Dr. Rajsrinivas Parthasarathy), and "Visual loss—role for thrombolytic therapy; rule out the mimic" (Dr. Aviraj Deshmukh). The 16th session was on intracranial hemorrhage and the talks were on 'Hematoma expansion—Is it of concern? & Intensive Blood pressure control—Is it

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overemphasized" and "clotting factors in ICH, Surgery in ICH & tPA in Intraventricular hemorrhage?" (Dr. Rohit Bhatia). The brain teaser themes were "Since the time of the Mummies; cocaine vasculopathy induced hemorrhage" (Dr. Anand Vaishnav), "The Pandora's box; cranial dural arteriovenous fistula presenting as subarachnoid hamorrhage, recurrent brain stem bleed, rapidly expanding vasogenic oedema mimicking dural sinus thrombosis, obstructive hydrocephalus & seizures" (Dr. Aviraj Deshmukh and Dr. Vipul Gupta), "Sulcal hemorrhage—what could it be?, a case of Takayasu arteritis" (Dr. Mahesh Kate) and "The bleeding heart! 'Sublata Causa, tollitur effectus', mycotic aneurysm with ICH" (Dr. Aviraj Deshmukh). The inauguration speech was by Dr. Ashok Panagariya on: future direction for young aspiring neurologists—"The Road not Taken." The first session after lunch was on carotid disease and the following topics were debated: "Argue the toss: Asymptomatic carotid stenosis: I intervene in this subset?" (Dr. Vipul Gupta), "Clearance for surgery in carotid disease? CABG and other!" "Dr. P. N. Sylaja" and "CREST 2 trail—Hear it from the investigator" (Dr. Jordi Blasco). The dilemma themes discussed was "Blind following carotid revascularisation—'What went wrong?' (Ocular ischemia resulting in neovascularity which then resulted in Ocular hyperperfusion and glaucoma following revascularisation)" (Dr. Rajsrinivas Parthasarathy), "APLA and stroke—My head is spinning!" and "When the power house malfunctions!" (Dr. Anand Vaishnav). Then, the treatment concept in basilar artery occlusion was discussed by Dr. Jordi Blasco. The day ended with simultaneous workshops for two hours each (1) neuroimaging workshop (early ischemic signs, ASPECTS scoring, posterior circulation ASPECTS, collaterals: leptomenineal, willisian and external to internal carotid, carotid plaque and stenosis, intracranial stenosis, and dissections) and (2) botox in spasticity (live hands on workshop on patients with upper and lower limb spasticity).

The second day started at 8 am with flow model based endovascular workshop demonstrating the technique of carotid revascularisation and mechanical thrombectomy. This was followed with talks on wake up and interventional management in stroke; the subject matter discussed was "Wake up stroke: My approach" (Dr. Dheeraj Khurana), "BGC—my first choice: Why? What do I resort to when it fails?" (Dr. Jordi Blasco), "ADAPT, SOLUMBRA, TRAP—Technical Nuances & How effective is it?" (Dr. Aquilla Turk), "Tandem occlusions—ICA stenosis and M1 occlusion & ICA dissection with M1 occlusion—My approach" (Dr. Vipul Gupta) and "Concurrent stenting in acute ischemic stroke—Expert opinion" (Dr. Jordi Blasco). The 12th session was a panel discussion on fellowship opportunities which included platform presentation of the top 4 abstracts by Dr. Arun, Dr. Charandeep Singh, Dr. Sai Sripad Rao, and Dr. Daniel Sweetson. The panellist were Dr. Ashfaq Shuaib (University of Alberta), Dr. Adnan Qureshi (Zeenat Qureshi Stroke Institute), Dr. Kenneth Butcher (University of Alberta), Dr. Vijay Sharma (National University of Singapore), Dr. Bijoy Menon (University of Calgary), Dr. David Liebeskind (UCLA), and Dr. Bernard Yan (Royal Melbourne hospital). The next session was on secondary prevention and the topics discussed were "Refractory symptomatic carotid artery dissection: I reconstruct parent artery in this subset" (Dr. Vipul Gupta), "Vertebral artery stenosis—When to stent?" (Dr. Adnan Qureshi) and "Intracranial atherosclerosis—Technical Considerations" (Dr. Vipul Gupta). The 14th session was on "Mechanical thrombectomy and Carotid & Intracranial stenting—complications encountered & how I handled them?" (Dr. Adnan Qureshi) and the brain teaser theme was "On the edge"—"trust your instinct—should you?" (Dr. Tariq Matin). This was followed with a session on dural sinus thrombosis; the subject matter for discussion was (1) "Technical considerations: compare techniques for opening the outflow pipes" (Dr. Rajsrinivas Parthasarathy and Dr. Srinivasan Paramasivam) and "NOAC in CVT" (Dr. Kenneth Butcher). The preworkshop session on neurosonolgy included the following topics: "Insonation 'Tips and Tricks'—Trade secrets revealed" (Dr. Amit Batra), "Interpretation of Waveform; 'The laws guiding flow pattern'; Pulsatility Index: what is it?"(Dr. Arvind Sharma), "Pattern Recognition: 'Proximal occlusion, High resistance pattern etc.—The underlying basis!'" (Dr. Vijay Sharma) and "My management decision was influenced by flow monitoring"—"Vasomotor reserve testing & HITS" (Dr. Vijay Sharma). Post lunch was the endovascular workshop during which a live carotid artery stenting procedure was transmitted from Artemis hospital to the venue. The delegates had a chance to view the live surgery and discuss their questions (90 min). The last session was a work shop on transcranial Doppler. Normal and waveforms in pathological condition such proximal occlusion, and focal and long segment stenosis were demonstrated on live patients.

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Differential Distribution of Cerebral Microbleeds in the Subtypes of Ischemic Minor Stroke and TIA and Association with Vascular Stroke Risk Factors

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Background

Cerebral microbleeds (CMBs) are considered as predictors of recurrent stroke and poor functional outcomes following an ischemic stroke, however, there are very few studies intriguing the distribution of CMB in the various ischemic stroke subtypes.

Aims

To determine the prevalence of CMB in the subtypes of ischemic minor stroke and transient ischemic attack (TIA) as per the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification as well as to identify the vascular risk factors associated with location and number of CMB.

Methods

Patients with ischemic minor stroke (NIHSS of 5 or less) or TIA, but without any significant pre-existing disability (mRS > 2), traumatic/spontaneous intracerebral hemorrhage or dementia were recruited. The presence and distribution of CMB were assessed utilizing the microbleed anatomical rating scale.

Results

488 subjects (79% minor stroke) were included with mean age (SD) being 57.5 years (14.4) and males constituting 77.7%. The most common TOAST subtype was undetermined etiology (31.6%) followed by large artery atherosclerosis (LAA-28.5%), lacunar (25.6%), and cardioembolic (CE-8.6%) subtypes. CMB were noted in 140 (28.7%) of the study population, with 42 (8.6%) having four or more CMB. A 35% of those with CMB had a lacunar etiology, whereas LAA and CE subtype constituted 33.6% and 10.7%, respectively (*P* value 0.000). Lacunar subtype was more likely to harbor multiple CMB (four or more) as well as CMB in all locations (lobar, deep, or infratentorial). On multivariate analysis, systemic hypertension [P value: 0.025; OR 0.335 (95% CI 0.129–0.874)], serum triglyceride levels below 150 mg/dl [P value: 0.001; OR 3.70 (95% CI 1.698–8.072)], and presence of white matter hyperintensities on MRI brain [P value: 0.026; OR 2.18 (95% CI 1.096–4.337)] were associated with presence of CMB. Those with serum triglyceride levels of less than 150 mg/dl were more likely to harbor lobar (*P* value 0.002) or infratentorial CMB (*P* value 0.022), whereas those with serum creatinine levels of 1.5 mg/dl tend to have underlying lobar CMB (*P* value—0.033).

Conclusion

Our study provides evidence on the differential distribution of CMB in ischemic stroke subtypes in the Indian subcontinent, and association of vascular stroke risk factors with the presence, number, and location of CMB.

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Study of the Clinical, Ecg, and Biochemical Spectrum of Cardiovascular Complications in Patients of Aneurysmal Subarachnoid Hemorrhage—an Initial Experience at a Tertiary Centre in India

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Introduction

Subarachnoid hemorrhage (SAH) following aneurysmal rupture is an extremely fatal condition with mortality as high as 45%. Apart from the intracranial causes, the cardiovascular events which add to the morbidity and mortality are myocardial ischemia, arrhythmias, and heart blocks. These can manifest with deranged cardiac biomarkers. However, quantitative assessment of these biochemical markers and its correlation with prognosis in patients of aneurysmal SAH has not been adequately studied.

Methods

After obtaining ethical clearance, we conducted a prospective observational study in our department. The study included all patients of aneurysmal SAH with ictus less than 48 hours at the time of admission. Excluded were patients with past history of coronary artery disease or cardiac surgery. Hemodynamic and neurological findings along with the Hunt and Hess/WFNS grade of SAH were noted on admission. Serum cardiac enzymes (troponin T, total creatine phosphokinase, cardiac specific creatine phosphokinase), serum brain natriuretic peptide (BNP), and C reactive protein levels were done for six consecutive days from the day of admission. Patient also underwent a 12-lead ECG and 2D echocardiography on days 0, 1, and 5.

Results

ECG abnormalities were observed in 76% of the cases (n–25) which included ST depression/elevation, T wave inversion/tall peaked T waves, prominent U wave, prolonged QTc interval, sinus bradycardia or tachycardia, and RBBB. Prolonged QTc interval was the most common abnormal ECG finding among the patients who died. Elevated serum levels of troponin T, total creatine phosphokinase, and BNP were statistically associated (p value < 0.05) with poor outcome (Troponin T, p-0.04, day 4; total CPK, p-0.01, day 3; BNP, p-0.045 on day 3). There was no association seen between cardiac specific creatine phosphokinase and C reactive protein with outcome. One patient developed 3rd degree heart block following aneurysmal SAH and another patient developed left ventricular dysfunction with an ejection fraction of 40% (1/25 patients).

Conclusions

Cardiovascular complications are common in patients of aneurysmal SAH. Serum quantitative levels of troponin T, total creatine phosphokinase, and BNP show statistical significant association with outcome and can be incorporated in the battery of serum tests in SAH patients for predicting outcome.

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Cerebral Venous Sinus Thrombosis Presenting with Headache and Papilledema Without Focal Neurological Deficits

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Introduction

Cerebral venous sinus thrombosis (CVST) presents with a wide spectrum of clinical manifestations, which may be grouped into four clinical syndromes: isolated intracranial hypertension (IIH), focal syndrome, diffuse encephalopathy, and cavernous sinus syndrome. The most common symptom is headache, which may be associated with seizures, focal neurological deficits, and/or papilledema.

Objective

The aim of our study was to determine the percentage of patients with CVST, who presented with raised intracranial pressure without localizing signs (IIH pattern), in a tertiary care hospital in south western Karnataka.

Methods

Medical records of all cases admitted under the Department of Neurology, Kasturba Medical College, Manipal, over a 2-year period (May 2015 to May 2017), with a diagnosis of CVST were reviewed.

Results

A total of 61 patients were identified (male: 40; mean age: 35 ± 13). Fifty-four patients had headache as a symptom (n = 61). Sixteen patients presented with IIH pattern, and only five had isolated headache. Median duration of headache was seven days. Parenchymal lesion was noted in 59% (n = 61). Patients with headache and localizing signs were more likely to have a parenchymal lesion (71% vs. 19%, $p < 0.001$), as compared to those with IIH pattern.

Conclusion

Clinical presentation with IIH pattern in CVST is not rare. In our study, 1/4th of patients presented with IIH pattern. Significantly less parenchymal lesions were noted in these patients. Hence, it is important to consider CVST when patients present with IIH pattern and short duration of headache.

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Intra-Aneurysmal Balloon Catheter Looping Technique in Ruptured Wide Neck Complex Middle Cerebral Artery Aneurysm for Coil Embolization: a Case Report

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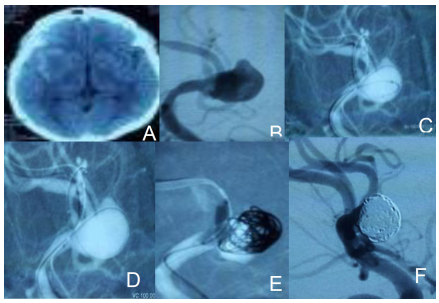
Objective

Aneurysm in middle cerebral artery (MCA) bifurcation is common; however, trifurcation aneurysm is a rare presentation. Wide necked aneurysms are the most difficult to treat by the endovascular method. Microcatheter looping technique in unruptured aneurysms has been described. We present here a balloon catheter looping technique in a ruptured wide necked MCA trifurcation aneurysm.

Case

A middle-aged woman presented with sudden onset of severe headache and vomiting. CT-scan (A) of the patient showed subarachnoid hemorrhage in right sylvian fisher (fisher grade II). The Glasgow coma scale score was 15. Digital subtraction angiography (DSA) showed an aneurysm (B) with a size of 12 mm × 8 mm with a neck of 5 mm at the trifurcation of right MCA. A single lumen super complaint balloon catheter was introduced over microguide wire (0.014 inch) and placed at M1 segment. Avoiding the rupture point, the microwire was looped (C) within the aneurysm sac and navigated distally into the M2 segment of right MCA and placed distally. Balloon microcatheter was advanced over the wire slowly up to the M2 segment. Then, the microwire was pushed distally to have adequate support and balloon catheter was pulled back slowly, so that the balloon was at the neck of the aneurysm sac (D). A microcatheter was placed at the center of the aneurysm and multiple GDC coils were deployed with assistance of the inflated balloon (E). Due to coil loop exposure, there was nonocclusive thrombus in M2 segment that resolved with adequate heparinization. The global DSA showed no branch occlusion. A follow-up check DSA was done after three months and showed near total occlusion of the aneurysm with patent arteries (F).



Conclusion

Balloon catheter looping technique may be considered even in ruptured large wide neck aneurysms. However, careful planning to avoid the potential rupture spot is the key to successfully loop the catheter within the aneurysm sac.

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Flow Diverter in Wide Neck Internal Carotid Aneurysm with Fenestration

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Introduction

Intracranial aneurysms result in serious morbidity and mortality. Fenestration of the internal carotid artery is an extremely rare entity, which may be associated with saccular aneurysms, either arising from the site of fenestration or from another arterial site. Endoluminal flow-diversion devices have been associated with high rates of complete aneurysm occlusion, even in giant aneurysms. The flow diverter device placed across the aneurysm neck, disrupts, and diverts the flow creating stagnation and eventually leads to aneurysm thrombosis with compression or mass effect. Flow diversion should be considered a viable treatment for symptomatic, cavernous internal carotid artery aneurysms in selected patients.

Case

A 33-year-old male presented with complaints of headache on and off for past 3 years. MRI brain showed giant left cavernous internal carotid artery aneurysm with minimal eccentric thrombosis and aneurysm measuring 2.3 × 2.7 cms arising from the cavernous segment of left internal carotid artery. Digital subtraction angiography showed left cavernous saccular aneurysm of 11.7 mm diameter, 14.9 mm length with a fenestration in parent artery. A 4 × 40 mm surpass was deployed across the aneurysm neck. Care was taken to appose the flow diverter against the wall. Dynamic CT showed good opposition of stent to wall even at the site of fenestration. No per/post procedural complications were noted. Repeat CT angiogram and digital subtraction angiography showed complete thrombosis of the aneurysm. Cavernous internal carotid artery aneurysms appear well-suited to treatment by flow diversion, given the side wall morphology and lack of critical perforating side branches of the aneurysm in the region of the aneurysm cavity.

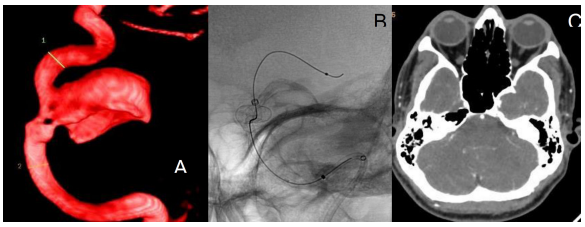

Figure 1. (A) Cavernous part of internal carotid artery showing saccular aneurysm with parent artery fenestration; (B) post procedure CT showing the position of flow diverter; and (C) follow-up CT showing thrombosed aneurysm and patient stent.

Conclusion

Flow diversion may be a safe and an effective option in treating challenging wide neck intracranial aneurysms even when there is a fenestration in the parent artery. Care should be taken to appropriately size and appose the stent on to the wall of the artery to achieve complete aneurysm occlusion.

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