Stroke Talk

DIALOGUE OF EXPERTS

Indian Stroke Association

An Official Publication of ISA

ISSUE - 7

A PIECE OF EDITOR'S LEXIS

Dear Friends,

Indeed, as we step into 2011, some wonderful strokes have won us the much-awaited 2011 World Cup Title and a wonderful year at Indian Stroke Association.

Indian Stroke Association has had some great innings with its score of members increasing from 220 to 510 in just one year. Testimony to the fact, that stroke movement is gaining ground.

In the gamut of achievements were three stroke meetings held during the year that helped to clarify objectives and channelize our goal towards enhancing stroke management.

Empowering us in this resolution 'Stroke Talk' aimed to strengthen our efforts with more updates, useful news and stroke protocols to enhance stroke management.

The ISCON conference attended by an intellectual faculty of 350 members, of which 10 were of international standings, helped to encourage and contribute to the recent advances in the management of stroke, dynamism in facing challenges, more agility in detection & diagnosis and more velocity in action.

Championing the cause with her heart and soul is Rani Mukherjee, whose dad suffered from a massive cardiac arrest. Rani has unconditionally pledged her support and contribution towards awareness and treatment of stroke across India and has joined hands with an NGO working for the cause.

We have a huge task ahead of us to raise awareness about stroke and possible risk factors that can lead to it. During stroke, the golden hour being crucial it is important that the patient is empowered to make the right choice of stroke units. At Indian Stroke Association, we strongly believe this can bring on a paradigm shift in stroke management and create a more positive change for our patients.

We are working at stroke unit endorsement through quality controlled measures and protocols. An increased awareness of the right stroke unit will go a long way to strengthen their decisions. Fortifying the cause are preventive strategies that are being designed for post-stroke care.

We have aimed to grow in order to get our voice and the voices of the hundreds of thousands of stroke survivors heard. We want to sincerely thank our supporters and those who have recently joined us in taking this work forward.

The warm response received is witness to the fact that our work has been well received and we have a long way to go.

Regards

PANEL OF ISA

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To contribute to Stroke Talk

Please send your articles/ experiences/ stroke related activity to : stroketalk.isa@gmail.com

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FROM THE PRESIDENT'S DESK
STROKE PLAN / VISION 2011-2012

DR. M M MEHNDIRATTA

Dr. M.M.Mehndiratta

Dear ISA Members,

At the outset, I wish to congratulate my immediate predecessors Dr. Subhash Kaul and Shirish Hastak for maintaining high standards and taking stroke activities in India to greater heights. Once again, I wish to thank you all for bestowing on me the privilege and responsibility of this post. I will like to mention certain principles of my vision for the year 2011-2012 (my tenure as president of ISA-13.03.2011 to March 2012. I have mentioned some of these points earlier but will like to reemphasize my view point

- It will be my effort to pursue academically driven agenda.
- I will stand committed to the cause of stroke
- I will like to follow qualitative principle to achieve stroke agenda.

Some of the activities I have planned will be as follows:

A) Education and Knowledge Enhancing Activities in the medical fraternity

B) Awareness Project

C) Broadening the Indian Stroke Association outreach

A) Education and Knowledge Enhancing Activities in the medical fraternity

1) STROKE TEACHING PROGRAMS

NEUROLOGIST TEACHING / Hands on WORKSHOPS

- The keen neurologists – young and interested ones could be taught on managing stroke, intervention and thrombolysis.
- This could be achieved by conducting special workshops like those of Hacke Meeting.
- We could now explore some other speaker to do the same meeting.

"Stroke Training Programme-STP" ISA will like to develop a

"Stroke Training Programme-STP"

- This could be rolled out at the National and Regional Meetings like APICON etc and the primary target audience would be GP's and Physicians.

2) DNB / DM STUDENTS TRAINING

- We could have a day or two day intensive stroke training workshops for the DM and DNB Neurology students.
- We could look at a method of paying them a fixed charge to cover their travel and stay to the venue.

3) MENTORSHIP PROGRAMS

- This activity can be continued in a more aggressive way by involving many more hospitals.

B) AWARENESS PROJECT

- MEDIA AWARENESS
  a. ISA as an organization could approach the various media channels to promote the symptoms of stroke and that they need to present early of they see those symptoms.
- SCHOOL AWARENESS
  a. The Schools and School children seems to be a very impactful media of communicating the Stroke, not only to educate them but alto to use them as a medium to educate their parents and grandparents as well.
  b. We could explore a School / College Students awareness campaigns.

C) Broadening the Indian Stroke Association outreach

a. International Advisors: New advisors suggested to make
ISA truly international.
b. Honorary membership
c. Fellowship of Indian Stroke Association-FISA
d. Honorary Membership of ISA
e. Tenure of Office bearers

Yours,

Dr. M.M.Mehndiratta

President-Indian Stroke Association

"FROM THE VICE PRESIDENT'S DESK"

DR. M.R.SIVAKUMAR

Dr. M.R. Sivakumar

It gives me great pleasure in addressing all the members of ISA, as your Vice-president. At the outset, I wish to thank the members of the executive committee and all the members of the ISA, for electing me to the Post and I sincerely look forwards to serve the ISA and the Stroke community. Dr. Sirish Hastak has worked hard and taken the ISA to dizzy levels, multiplying the membership of the ISA by double to more that 500 members. Dr. Katpal had organized an excellent meeting at Indore with a galaxy of eminent speakers from India and Abroad. I am sure Dr. Mehndiratta has many plans for the current years and will lead the Association as effectively as Dr. Sirish. We are steadily progressing and all of us should work with a mission to reduced the mortality and morbidity associated with Stroke. Our vision should be to create a network of Stroke units, connected by Telestroke to deliver tissue Plasminogen Activator to all eligible patients with acute ischemic stroke. At present very few patients are treated with tPA, the only approved treatment for acute ischemic stroke. We shall push through all the pending agenda and all the members are welcome to make suggestions regarding improving the ISA. I request every member to play an active role in the activities of the ISA and in the management of Stroke victims. I am always available to everyone at profmrs@gmail.com, Cell: +919840017893.

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LETTER TO EDITOR DRAFT

P.M. DALAL

The World Stroke Day 2010 theme "One in Six-Act now" is a warning bell on the rising stroke burden particularly in the developing countries. As life expectancy increases, India will face enormous socioeconomic burden to meet the costs of integrated rehabilitation of subjects with a stroke. Caring for stroke patients leads to caregiver strain. In this context, I would like to draw readers' attention on caregiver stress which is a neglected domain in stroke patient care and rehabilitation.

Recovery of stroke patient is enhanced by a supportive environment and healthy caregiver. There is evidence to suggest that caregiver stress may impact the recovery and successful rehabilitation of stroke patients. 1

The recently concluded Mumbai Stroke Registry provided the basic framework for the Caregiver study conducted by M. Bhattacharjee, J. Vairale et al in the H-ward area of Mumbai 2. The level of stress in caregivers was ascertained by two scales: Oberst Caregiving Burden Scale (OCBS) and the Caregivers Strain Index (CSI) 3,4 at 28 days after stroke and follow-up at 6 months and1 year.

The study revealed that patient factors leading to increased caregiving burden included patient being female ($p=0.0183$), moderate to severe neurological deficit by NIHSS scale on admission ($p=0.0254$), morbidity at 28 days by Modified Rankin Scale ($p=0.0051$), poor recovery at 28 days with Barthel Index score of less than 50 ($p= 0.0000015$). However, patient's age, type of stroke, presence of risk factors like hypertension, diabetes mellitus, raised cholesterol, ischemic heart disease etc were not related to increased caregiver burden.

Caregiver factors which were related with high caregiving stress included younger age < 45years ($p=0.0021$), female gender ($p=0.0524$), long caregiving hours, on an average 8.4 hrs per day ($p=<0.000001$), anxiety ($p=<0.000001$), disturbed night sleep ($p=<0.000001$), financial stress ($p=0.0000108$), caregivers being daughter-in-laws ($p=0.012$).

Most of the caregivers in the study were immediate family members and none of them had access to any social network/ or support group for additional help, which is available in western countries 5. This also reflects the tolerant nature of Indians and Asians and emphasizes strong family bonding 6. Here, it is worth mentioning the excellent Stroke Support Group initiative started by Dr. Shirish Hastak which is a welcome step.

Likewise, Prof. G. Arjundas and Dr. Deepak Arjundas have initiated an important rehabilitation programme right in the wards with participation of near relations who will be caregivers of the patient

Planned studies of caregiver stress vis-a vis stroke burden with well defined protocol at multiple centres need to be studied to arrive at some general planning as well as identify local situation.

Burden of stroke as well as that of caregivers needs emphasis in terms of planning health care delivery system. In absence of organized health care, it is difficult to define preventive planning measures; but with intensive awareness programmes it is anticipated that more strokes will reach health care centres. Some kind of support system is mandatory but has received no attention by the Planning Commission in the Health Budget. This requires strong political will. Stroke rehabilitation services should also address caregiver issues, and include practical training in nursing skills and counseling sessions which will help in reducing the caregiver burden and improve patient recovery. [7,8,9]

References

1) Rigby H, Gubitz G, Eskes G, Reidy Y et al. Caring for stroke survivors: baseline and 1-year determinants of caregiver burden. Int. J of Stroke. 2009; 4: 152-158.
2) Dalal PM, Malik S, Bhattacharjee M, Trivedi ND, Vairale J, Bhat P et al. Population based stroke survey in Mumbai, India: Incidence and 28-day case fatality. Neuroepidemiolgy 2008; 31(4): 254-261.
3) Oberst, 1990; Oberst et al., 1989 Oberst, M.T. (1989). Perspective on Research in Patient Teaching. Nursing Clinics of North America. 24. pp. 621-628.
4) Zarit SH, Reever KE, Bach –Peterson J. Relatives of the impaired elderly: correlates of feelings of burden. Gerontologist.1980; 20(6):649-55.
5) Anderson CS, Linto J, Stewart Wynne. A population based assessment of the impact and burden of caregiving for long term stroke survivors. Stroke 1995; 26:843-9.
6) Das S., Hazra A, Ray BK, Ghosal M et al. Burden among stroke caregivers-results of a community based study from Kolkata, India. Stroke.2010; 41:00-00.
7) McCullagh E, Brigstocke G, Donaldson N, Kalra L. Determinants of caregiving burden and quality of life in caregivers of stroke patients. Stroke.2005; 36:2181-2186.
8) Hankey G. Informal caregiving for disabled stroke survivors. BMJ.2004; 328:1085-86.
9) Kalra L, Evans A, Perez I, Melbourn A, Patel A, Knapp M, Donaldson N. Training care givers of stroke patients: randomized controlled trial. BMJ. 2004; 328:1099–101.

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A CASE FOR INTRA ARTERIAL TPA IN STROKE

Introduction –

Thrombolytic therapy with tissue plasminogen activator (tPA) has been shown to improve outcome in ischemic stroke patients treated within 4.5 hours from symptom onset. (1, 2) Two large European studies and 2 trials from the United States have clearly shown that the time window for fibrinolytic therapy is rather narrow, 1,3– 6 although the results of the second European study 4 suggest some benefit for patients who undergo thrombolysis, even when treated within 3 to 6 hours from symptom onset.

Factors that determine outcome in acute ischemic stroke include

- Recanalization
- Speed of Recanalization
- Symptomatic hemorrhage
- Collateral Circulation
- Stroke Sub-Type
- Previous Stroke
- Co-morbid Conditions

Case presentation –

A 29 year old female presented in the Emergency Department within 1 and half of stroke onset. She presented with aphasia, right hemiparesis and neglect. She had a history of untreated Rheumatic Heart Disease. As per protocol, she underwent NCCT, CTA and CTP, demonstrating a M1 occlusion and a large Penumbra on the CTP

She was given full dose IV rt-PA while waiting for intraarterial intervention. There was no resolution of the MI occlusion. The National Institutes of Health Stroke Scale (NIHSS) score was 17 and the ECG was normal. Computed tomography (CT) 30 minutes after arrival demonstrated a hyperdense MCA, and CT angiography confirmed a 1.4-cm M1 segment MCA occlusion. Early ischemic changes were already present but a larger area of potentially salvageable "tissue at risk" was identified on CT perfusion imaging. (Figure 1)

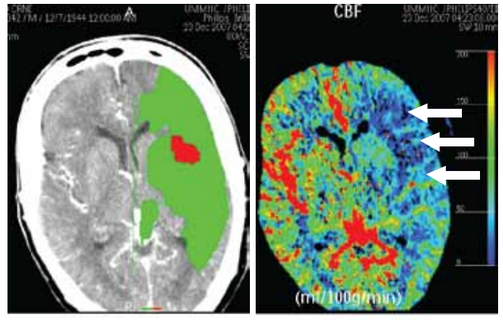
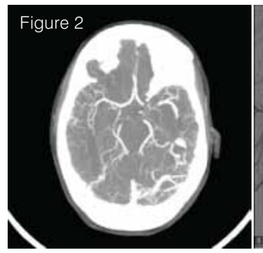


Figure 1: Baseline CT perfusion imaging confirms (right) preservation of cerebral blood volume except for some reduction within the area of hypoattenuation (white arrowhead), surrounded by reduced cerebral blood flow and prolonged mean transit time within a larger distribution of the right middle cerebral artery territory (including temporo-occipital and frontal lobes). This mismatch between reduced blood flow and the area of preserved cerebral blood volume is consistent with an ischemic penumbra.

A 0.6-mg/kg intravenous dose of tPA (alteplase) was administered, with 15% given as a bolus, followed by a 30-minute infusion (symptom onset-to-needle time was 89 min). Patient finished the IV rt-PA and had a catheter in place by 4.2 hours. Results from initial CT scan showed no resolution of thrombolysis. (Figure 2)



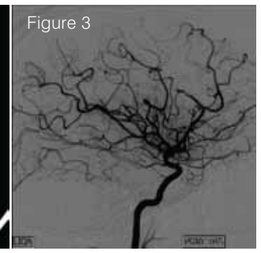


Figure 2: Enhanced CT head after intravenous rt-PA shows the area of occlusion. **Figure 3:** Procedural catheter digital subtracted angiogram. Post-treatment angiogram shows complete recanalization (TIMI 3), which correlated with rapid neurological recovery.

Thereafter she received intra-arterial rt-PA and achieved quick reperfusion and complete resolution of symptoms. The patient was transported to the angiography suite 105 minutes post-onset, where a diagnostic angiogram confirmed that the M1 segment occlusion was still present (thrombolysis in myocardial infarction [TIMI] classification 0) (Fig. 3).

Following the procedure, complete recanalization was achieved (TIMI 3) (Fig. 3) with dramatic clinical recovery. There were no clinical complications. Post-angiography CT scan showed contrast enhancement of the right basal ganglia, which cleared the following day. (Figure 4).

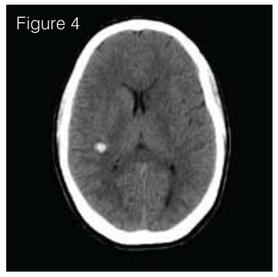


Figure 4: Unenhanced CT scan results showing resolution

The patient was ambulating the next day and discharged home with no detectable neurologic deficit. Re-examination 3 weeks later confirmed a complete neurologic recovery: NIHSS score 0, Folstein Mini Mental State Examination 30/30, Barthel Index 100/100, and normal performance on the Sunnybrook Neglect Assessment Procedure for hemispatial inattention. She was able to resume all her independent daily activities. She was prescribed oral anticoagulation for secondary stroke prevention.

Discussion –

This case demonstrates that acute stroke thrombolysis can be very effective in the appropriate candidate and often intrarterial thrombolysis can help in complete resolution. Without treatment, spontaneous recovery would not have been expected given the extent of the proximal MCA thrombus and the early neuroimaging signs of evolving infarction. A hyperdense MCA sign on baseline CT3 correlates with severe stroke and predicts a poor outcome, even after treatment with IV tPA. (7) We used an acute stroke imaging protocol consisting of plain head CT, CT angiography, CT perfusion, and post-contrast head CT (total scan time approximately 2 min), which can facilitate patient selection for thrombolysis by providing rapid information about the vascular lesion, extent of infarction and presence of potentially salvageable brain tissue.

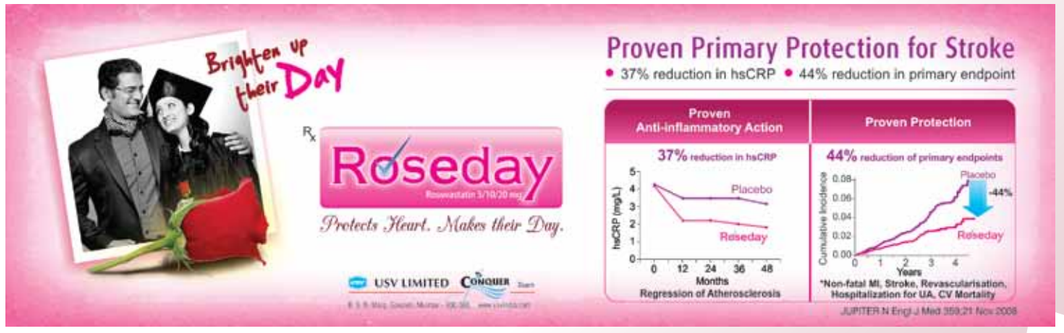
Early recanalization of occluded cerebral vessels is believed to improve clinical outcomes in patients with acute ischemic strokes. Clinical experience also suggests greater recanalization rates with intraarterial thrombolysis than when using the intravenous route. (8, 9) The PROACT-I (10) was the first randomized, double-blind, mul-ticenter trial, in which the safety and efficacy of intraarterial delivery of recombinant pro-urokinase (r-proUK) was compared with placebo. The trial was conducted using a homogeneous patient population; patients presented with an acute middle cerebral artery occlusion of fewer than 6 hours duration. The PROACT-I study demonstrated that intraarterial infusion of r-proUK was associated with higher rates of arterial recanalization compared with placebo infusion.

The follow-up PROACT-II 24 study was a multicenter, randomized trial in which 180 patients of 12,323 screened patients were randomized. The study patients were randomized in a ratio of 2:1 (treatment:control), and the study was not placebo controlled because of concerns regarding the infusion of a placebo drug into an occluded middle cerebral artery. In addition, the dose of r-proUK was increased from 6 to 9 mg and the trial was changed to an open design with blinded follow-up study. From the primary efficacy data analysis, 40% of r-proUK patients compared with 25% of control patients had a modified Rankin score of 2 or less at 90 days after stroke onset ($p = 0.04$). The rate of symptomatic ICH was 10% in the treatment group compared with 2% in the control group, although this did not reach statistical significance at 10 days. The increased number of ICHs did not change the mortality rates between the two groups. Despite the lack of statistical significance of the hemorrhage rate, the clinical significance is considerable. (11) Despite the lack of FDA approval for this treatment, numerous academic centers, and some small community hospitals, (12) are treating patients with intraarterial thrombolysis. Direct comparison between these uncontrolled studies is difficult given that different dosing regimens, stroke types, and time intervals were examined.

A major concern regarding the use of intraarterial thrombolysis is the amount of time needed to obtain an emergency cerebral angiogram. Intravenous tPA may be given with minimal delay once a head CT is obtained, but many patients are still left with residual neurological deficits. (1) In addition, many patients treated with intravenous tPA have residual occlusion of the involved vessel. (13) An innovative treatment approach in which an attempt was made to combine initial intravenous tPA with local inraarterial thrombolysis was used in the Emergency Management of Stroke Bridging Trial.4 This was a double- blind, randomized, placebo-controlled, multicenter feasibility study with 35 enrolled patients, in which intravenous plus intraarterial tPA (IV/IA) treatment was compared with placebo plus intraarterial tPA (placebo/IA). The study was conducted prior to the FDA approval of intravenous t-PA. Despite randomization, patients who were found not to have an arterial thrombus at the time of the angiogram did not receive intraarterial tPA treatment. The rate of symptomatic ICH at 72 hours ranged from 5.5% (IV/IA Group) to 11.8% (placebo/IA Group). Primary outcome measures were not different between treatment groups at 90 days of follow-up study. This study showed a direct correlation between the National Institutes of Health stroke scale score and the presence of thrombus in a "major cerebral artery." This finding could be explored in future studies as a marker to identify patients who may benefit from intraarterial thrombolysis based on the PROACT data. (11) Because the number of patients was so small in the Emergency Management of Stroke Bridging Trial, no definitive conclusions can be drawn except that combination intravenous/intraarterial therapy is feasible and appears safe and deserves further investigation. These preliminary reports are interesting but need further exploration in rigorous clinical trials.

References

1. The National Institute of Neurological Disorders and Stroke rt-PA Study Group. Tissue plasminogen activator for acute ischemic stroke. N Engl J Med. 1995;333:1581–1587.
2. Kwiatkowski TG, Libman RB, Frankel M, Tilley BC, Morgenstern LB, Lu M, Broderick JP, Lewandowski CA, Marler JR, Levine SR, Brott T, for the National Institute of Neurological Disorders and Stroke rt-PA Study Group. Effects of tissue plasminogen activator for acute ischemic stroke at one year. N Engl J Med. 1999;340:1781–1787.
3. Hacke W, Kaste M, Fieschi C, Toni D, Lesaffre E, von Kummer R, Boysen G, Bluhmki E, Höxter G, Mahagne M-H, Hennerici MG, for the ECASS Study Group. Intravenous thrombolysis with recombinant tissue plasminogen activator in acute hemispheric stroke. JAMA. 1995;274: 1017–1025.
4. Hacke W, Kaste, Fieschi C, von Kummer R, Davalos A, Meier D, Larrue V, Bluhmki E, Davis S, Donnan G, Schneider D, Diez-Tejedor E, Trouillas P, for the Second European-Australasian Acute Stroke Study Investigators. Randomized double-blind placebo-controlled trial of thrombolytic therapy with intravenous alteplase in acute ischemic stroke (ECASS II). Lancet. 1998;352:1245–1251.
5. Marler JR, Tilley BC, Lu M, Brott TG, Lyden PC, Grotta JC, Broderick JP, Levine SR, Frankel MP, Horowitz SH, Haley EC Jr, Lewandowski CA, Kwiatkowski TP, for the NINDS rt-PA Stroke Study Group. Early stroke treatment associated with better outcome. Neurology. 2000;55: 1649–1655.
6. Clark WM, Wissmann S, Albers GW, Jhamandas JH, Madden KP, Hamilton S, for the ATLANTIS Study Investigators. Recombinant tissue-type plasminogen activator (alteplase) for ischemic stroke 3 to 5 hours after symptom onset. The ATLANTIS Study: a randomized controlled trial. JAMA. 1999;282:2019 –2026.
7. Tomsick T, Brott T, Barsan W, et al. Prognostic value of the hyperdense middle cerebral artery sign and stroke scale score before ultraearly thrombolytic therapy. AJNR Am J Neuroradiol 1996;17:79-85.
8. del Zoppo GJ, Pessin MS, Mori E, et al: Thrombolytic intervention in acute thrombotic and embolic stroke. Semin Neurol 1991;11:368-384
9. Sasaki O, Takeuchi S, Koike T, et al: Fibrinolytic therapy for acute embolic stroke: intravenous, intracarotid, and intra-arterial local approaches. Neurosurgery 1995;36:246-53.
10. del Zoppo GJ, Higashida RT, Furlan AJ, et al: PROACT: a phase II randomized trial of recombinant pro-urokinase by direct arterial delivery in acute middle cerebral artery stroke. PROACT Investigators. Prolyse in Acute Cerebral Thromboembolism. Stroke 1998;29:4-11.
11. Furlan A, Higashida R, Wechsler L, et al. Intra-arterial prourokinase for acute ischemic stroke. The PROACT II study: a randomized controlled trial. Prolyse in Acute Cerebral Thromboembolism. JAMA 1999;282:2003-11.
12. Edwards MT, Murphy MM, Geraghty JJ, et al: Intra-arterial cerebral thrombolysis for acute ischemic stroke in a community hospital. AJNR 1999;20:1682-7.
13. Brott TG, Haley EC Jr, Levy DE, et al: Urgent therapy for stroke. Part I. Pilot study of tissue plasminogen activator administered within 90 minutes. Stroke 1992;23:632-40.



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POST-LUMBAR PUNCTURE INTRACRANIAL HYPOTENSION AND CEREBRAL VENOUS THROMBOSIS: CAUSAL OR A MERE ASSOCIATION.

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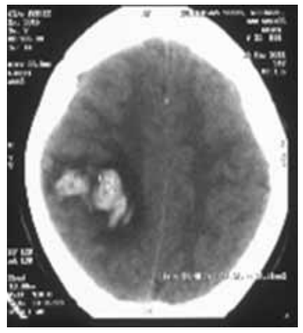
Dr. Sylaja P.N.

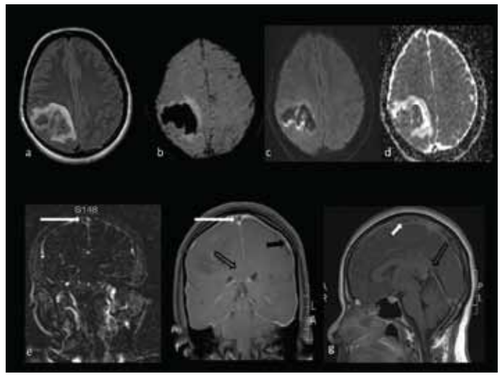
Introduction:

Post-lumbar puncture headache (PLPH) is common occurring in 10-30% of patient due to persistent CSF leak performed either for anaesthesia or diagnosis[1]. Post- lumbar puncture intracranial hypotension syndrome (PPIHS) with persistent headache and neurological deficits is rare. PPIHS usually has benign course with most patient making complete recovery. Few patients (4-10%) may develop complications in the form of subdural hygroma and hematoma, neurological deficits due to brainstem compression, recurrence of IHS, Parkinsonism mental deterioration and coma. Rarely PPIHS can be associated with cerebral venous thrombosis (CVT). We discuss a case with PLPH in a post-partum female who later developed (CVT).

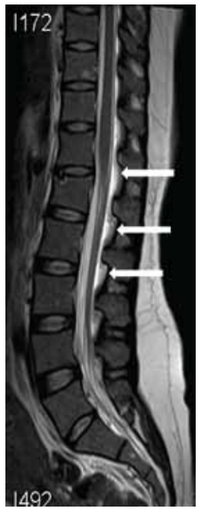
Case report:

A 31-yr-old female with pregnancy induced hypertension (PIH) underwent caesarean section (CS) at 35weeks of pregnancy (as the fetus had developed growth retardation). Spinal anaesthesia was used for the procedure. On the second day when she tried to get off the bed she noticed diffuse occipital headache and neck pain, which improved on lying down. This postural headache was persistent over next 8days following which it changed character and became continuous. A diagnosis of post-lumbar puncture headache (PLPH) was made and she was discharged with analgesic medication alone. On 12th post-partum day she had a generalized tonic clonic seizure after which she persisted to be drowsy. Prior to this episode she had been detected to have PIH in earlier two pregnancies and the first pregnancy resulted in intrauterine death. During the present pregnancy she was started on low molecular weight heparin from local maternity centre with suspicion of anti-phospholipid syndrome it was continued till 8th post-partum day. There was no history of abortions or deep vein thrombosis in past.





On examination in emergency after 12hours of seizure she was conscious and oriented. She did not have papilledema, visual acuity and field were normal. She had weakness and appendicular ataxia of the left upper limb and mild dysarthria. Her NIHSS on admission was 3 and mRS of 1. She was investigated with a CT scan head(Fig:1) after 8 hours of ictus at local hospital which showed right high parietal haemorrhagic infarct. After which she was referred to our comprehensive stroke care centre with diagnosis of cortical venous thrombosis.



MR imaging brain (Fig: 2 a-g) and spine (Fig:3a) (Sequences: T1W, T2W, Flair, SWI, DWI, ADC and MRV) was done after 14hours of ictus. It showed right posterior high parietal lobe hematoma with mass effect and right side superficial cortical veins and partial sagittal sinus thrombosis. In addition there was evidence of intracranial hypotension with CSF leak at lumbar puncture site as evidenced by corpus callosum sagging, pachymeningeal enhancement, diffuse prominence of cortical vein and rim of CSF seen in the epidural space from D10 to L3 vertebra. Her routine blood investigation including hemogram, liver function test and renal function test were within normal limits. ESR was elevated with 40mm at one Hr. Her coagulation parameters including prothrombin time and antithromboplastin time were normal. Prothrombotic work-up sent prior to starting of heparin therapy revealed reduced Protein C and Protein S activity of 31% (normal range 67-195%, plasma-clotting time based assay) and 26 % (normal range 55-123%, plasma-clotting time based assay) respectively. Antithrombin III antigen level was normal 209mg/L (Normal range170-300mg/L, chromogenic assay). Factor V Leiden mutation (Real Time PCR method) was not detected. The vasculitic work up (ANA, dsDNA, APLA IgG and IgM and ANCA) was negative.

Low molecular weight heparin was started along with bed rest and caffeine. She responded early and was discharged after 6days on warfarin. On discharge she did not have any headache and neurological deficit had subsided with NIHSS of 0 and MRS of 0.

Discussion:

We describe a case of post lumbar puncture intracranial hypotension as suggested by the typical postural headache of the patient and followed by the change of pattern of headache which could have been the point when cortical venous thrombosis developed . The MR brain and spine imaging features are also supportive of the above fact, showing classical features of intracranial hypotension and cortical venous thrombosis. Our patient had multiple predisposing conditions, like early post-partum period, history of intrauterine death and reduced activity of Protein C and Protein S. These predisposing conditions increased the likelihood of occurrence of thrombosis in our patient with PPIHS. Similar findings have been also described by Wilder-Smith et al[3], of their 5 patients three had predisposing prothrombotic condition. It is not only observed with spinal anaesthesia or epidural analgesia but also with post-myelography, diagnostic lumbar puncture, intrathecal injections, spinal injury and placement of lumbar drain.

The pathogensis of the PPIHS induced CVT can be explained by Monro-Kellie-Abercrombie doctrine, it gives the concept that the skull is a rigid structure, the brain volume, venous blood and the CSF are in state of equilibrium, reduction or increase of either element leads alteration in the volume of the other two. In IHS the CSF volume and pressure are reduced significantly. Two changes occur as a result of this, first in the venous compartment, there is increase in the intracranial venous volume and secondly decent of the brain and brainstem structures. The venous volume expansion is due to the venous stasis and dilation of the sinuses, cortical and spinal veins. This change occurs first in the meninges both pachymeninges and leptomeninges. The pachymeninges do not have a blood brain barrier this leads to contrast extravasation and hence the post-contrast enhancement. Further due to the decent of the brain there is distortion and stretching of the veins. All the above changes are further aggravated in erect posture when there is acute dilation of the veins and further stretch on the venous walls which leads to postural headache. This along with venous stasis may lead CVT in some patient. This hypothesis has been further bolstered by the study by Canhao et al[2] who showed reduction in the velocity of blood flow by approximately 50% in the straight sinus in patients after lumbar puncture.

In a recent review by Miglis et al[4], of 29 patients reported in the literature ,26 had postural headache prior to the development of the CVT. This clearly suggests that the IHS occurred first followed by CVT. The outcome of the patient developing CVT after IHS is good, most responding to either epidural blood patch or heparin injection. Though long term follow-up studies are not available most of the patients do not have any persistent neurological deficits.

Conclusion:

Therefore, in a setting of PPIHS, if there is change in the headache pattern this should alert the treating clinician that the patient may have developed CVT. Further this propensity is increased in patient with prothrombotic conditions.

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Disclosure: None

References:

1. Evans RW, Armon C, Frohman EM, Goodin DS. Assessment : Prevention of post – lumbar puncture headaches Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2000:909-914.
2. Canhao P, Batista P, Falcao F Lumbar puncture and dural sinus thrombosis--a causal or casual association? Cerebrovasc Dis. 2005;19(1):53-6
3. Wilder-smith E, Kothbauer-margreiter I, Lämmle B, et al. Dural puncture and activated protein C resistance : risk factors for cerebral venous sinus thrombosis. J Neuol Neurosurg Psy. 1997:351-356.
4. Miglis MG, Levine DN. Intracranial Venous Thrombosis After Placement of a Lumbar Drain. Neurocritical Care. 2010:83-87.

Legends

Fig 1: CT scan head shows right high parietal haemorrhagic infarct with surrounding edema.

Fig 2: MR imaging a-Flair b-SWI, c-d DWI and ADC map respectively show right high parietal hematoma without diffusion restriction, e-MR venogram shows thrombus in the sagittal sinus thrombus (white solid arrow), f- post contrast T1W coronal sequence shows enhancement of the pachymeninges (black solid arrow), sagging of the corpus callosum (black arrow) and filling defect of the sagittal sinus(white solid arrow), g- Post-contrast T1W sagittal sequence shows thrombus in the sagittal sinus (white solid arrow), narrowing of angle between the vein of Galen and straight sinus(black arrow).

Fig 3: MR imaging of the spine T2W sequence shows rim epidural CSF collection in the posterior aspect D11 –L3 vertebra suggestive of CSF leak (white solid arrow).

Size : W - 17" X H - 11.5"