The Newsletter & e-journal
Kairaly Neurosciences Society
NSI Kerala Chapter

Volume I, Issue 2 - November 2020

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HEADLINES

- The Kairaly Neurosciences Society meeting Essence 2020 was conducted at Perinthalmanna on February 22nd and 23rd
- Dr.K.A.Salam and Dr. Madhusoodanan.M received the life time achievement award from Kairaly Neurosciences Society (NSI Kerala Chapter) for the year 2020.
- The Kairaly Neurosciences Society launched the Official DIGITAL PLATFORM for virtual meetings and academics in July 2020
- Mid– term Meeting of Kairaly Neurosciences Society 2020 is being held in the Virtual Digital Platform for the first time in November 13th , 14th & 15th
- The Society expressed deepest condolences in the sad demise of Dr.Simon Hercules who lost his precious life during his service in Covid –19 Pandemic at Chennai.

Dr. Gnanadas .K

PRESIDENT`S MESSAGE

I am delighted to know that second edition of Bodhi is coming out. As you all know learning is a continuous process and the role played by Information Technology is very great.

I have no doubt that the second edition of Bodhi will enrich our knowledge in Neurosciences. Dr.Sreehari and team ,has been putting lot of effort to popularize and educate us through website, journal and News letter.

I am happy to announce the materialization of the Kairaly Neurosciences Digital Platform for Academic meetings and education. No doubt it will facilitate learning in various ways through interactions, teaching programs ,mock tests etc.I request all institutions to take up workshops and small conferences thereby cutting edge technology can be imparted to youngsters and also programs for the benefit of students. I request all members to participate to achieve maximum benefits.

I am extremely happy to work with dynamic youngsters Dr.Raj as Secretary and Dr.Tinu Abraham as Treasurer and with presence of esteemed Patrons Dr.Sreekumar Sir and Dr.Mohanlal Sir.

Friends, we all are going through one of the very bad times in the history because of Corona Virus infection. I appeal to all members to do everything possible to prevent escalation and spread of Corona Virus infection at the same time by taking all precautions for self protection

On behalf of Kairaly Neurosciences Society and on my behalf from the bottom of my heart ,let me congratulate Dr.Sreehari and team for the dedicated work for the organization and I Wish all success in this endeavor.

Thanking you

Dr.Gnanadas.K
President KNS

Secretary's Message

Dr. Raj.S.Chandran

Kairaly Neurosciences Society – NSI Kerala chapter as before has been actively supporting the academic activities and organizational progression for Neurophysicians, Neurosurgeons and related sciences in all times. The unprecedented Covid-19 pandemic has affected every service in the world and we are gradually
accepting the realistic remedial measures in our academic format also. The Society has taken an immediate step of launching the DIGITAL PLATFORM for regular meeting and Academic Porgrammes. With the support from the Senior members and Patrons of the society we could redeem and revive the activities to a great extend. This has paved way for the first Midterm meeting in virtual plat form which the first of its kind from our Society ,this was supposed to be at Kollam. The Virtual Platform also could be used for academic purpose ,PG discussions ,online classes, telemedcine etc., which could be organized by contacting through the website contact form.

The second edition of NEWS LETTER and e- journal "BODHI" was another tough task to materialize in this difficult times which could also be sorted out with help of our Senior members and editorial team.

The society expressed heartfelt condolences in the sad demise of Dr. Simon Hercules during his duties during Covid-19 pandemic and condemned the act of the antisocial elements during the burial.

I request all of the members and post graduates to actively publish their work and case reports to enrich the learning process which is the main goal of our society. I thank all the Editorial Board members who worked hard in this hard times for bringing out the second edition of BODHI.

I wish all the members and readers all success and request all to stay safe.

Dr.Raj S.Chandran
Secretary ,Kairaly Neurosciences Society

Minutes of Annual NSI Kerala Governing Body at Perinthalmanna 22/02/2020

Dr.Raj S.Chandran
Secretary , KNS

President Dr Girija A S chaired the meeting. Secretary Dr Raj S Chandran presented the report of the previous meeting of NSI Kerala Chapter held at Kochi and the minutes of previous governing body meeting. After discussions, the minutes was passed.
Treasurer Dr Tinu Ravi presented the accounts of the society, gave the details of fixed deposits and income from memberships and additional expenses for the up gradation of the website and Newsletter which is to be released in the inaugural function. He expressed concern over the fact that the profit share of last meeting was not handed over in time. Dr Sandeep, organizing committee secretary of last conference soon handed over the cheque of 1.5 lakhs . Also the huge increase in income tax to be paid was informed to the general body. The general body insisted that the profit share should be transferred to the association in time.

Dr Mohanlal proposed to change the official address to Kairaly Neurosciences Society ,House No 14/122/1, Divyasree, Kasthurba Lane,Koorkenchery, Thrissur 680007, and the general body accepted the proposal

The detail of the newsletter which is to be released along with the inauguration function was explained by Dr Sreehari, the webmaster in charge and the general body congratulated the people behind it.

The secretary explained the need to increase the registration fee for the state conferences and accepted by the GB.

Election of New office bearers was conducted by Dr Balakrishnan who was elected as the returning officer by the GB

Dr Gnanadas , the existing vice president to take over as the new president.
Dr P K Muhammed Kunju elected as the vice president proposed by
Dr Mohan lal
Dr Raj S Chandran to continue as the secretary
Dr Tinu Ravi Abraham to continue as the treasurer
Dr Balakrishnan, Dr Reghu and Dr Jyohish to continue as executive committee members , which also includes the immediate past president and treasurer

<u>Next year's academic meetings</u>

Mid term meeting at Kollam accepted by Dr Thomas Varghese
Annual Meeting at Alappuzha accepted by Dr Biju Bhadran
Meeting came to an end at 2.00 PM.

BODHI , Volume I , Issue 2 ,November 2020

Prof Dr AS Girija,
Former Prof and Head Dept of Neurology, Medical College, Calicut
Former Prof of Neurology, CMC, Vellore,Prof and Head, Dept of Neurology, Malabar Medical College, Modakkalloor, Calicut

My reflections on Koch's disease of the nervous system

Koch's disease or tuberculosis came into the world ever since man started befriending animals in the Agriculture revolution70000 years back [1].First described by Willis in the 17th century, tuberculous meningitis (TBM) is the most severe form of tuberculosis (TB).[2] The disease continues in modern era and the advances in technology has not produced a remarkable impact in its diagnosis unlike in other diseases . When looking back over my career of 40 years, this disease baffles a lot.

I remember my encounter with this disease in my undergraduate days in the general medical setup in medical and surgical ops especially in surgery when we were taught of the matted lymph nodes and sinus tracts seen along with it, the cases of chest tuberculosis we were taught in medical wards, in the TB hospital at Pulayanarkotta, and the difficulties we had in diagnosing cavities in the lung by auscultation . The daily injection of streptomycin which we as housesurgeons had to give to the patients with TB was something which needs special mention. This was at a time when the he/she had to do along with other jobs like collecting blood of each admitted patient and sending blood samples to the list of investigations ordered by the ward chief and Unit Head , collecting the reports from the labs in person as even telephonic communication was almost nil in the ward at that time.

As a post graduate student in the same institute, I was exposed to a greater level to this disease and my knowledge was sharpened thanks to my expert teachers there. However it was in the Institute of Neurology at Madras Medical College when I got training in Neurology that I became aware of the complexities of the nervous system tuberculosis and the challenge it poses at clinical level, in diagnosis and management. I will hereafter enumerate a few cases which I still thought peculiar and rare in this entity.

I was the first year resident of DM in Madras Medical College when this happened. A young man in his early twenties presented with a two week h/o weakness of both lower limbs without any gross sensory complaints . The clinical discussion with the professor during rounds finalised it to be a straight forward case of Guillain Barre Syndrome (GBS)and was advised to investigate on those lines. Nerve conduction studies was done . LP and CSF study was also done. As the results did not come automatically from the respective labs, I had to go in person to get it. I was surprised the CSF showing a relatively high count with lymphocytic pleocytosis. The protein was mildly elevated and

I felt he was convinced of the diagnosis of GBS. Patient had already been started on steroids as it was the norm on those days for the treatment of GBS and I had seen many cases improving with it. I left for my home on a short leave of 10 days to Kerala. On my return, I was back to the ward for rounds. It gave me a shock to find this patient in an unconscious state . He was on ryles tube and bladder catheter. He had developed a right hemiparesis. My Prof started him on ATT and the patient was transferred to the ICU .

The same year, it was during a CME programme I heard the HOD of our institute describing the various presentations of CNS tuberculosis. Something which caught my attention was its presentation mimicking GBS. A literature review revealed this rare presentation of TBM [3].It was also during my training period there, we used to make a provisional diagnosis of tuberculoma in many cases of single enhancing CT lesions which by an important study at CMC, Vellore turned out to be due to neuro cysticercosis in a high percent. I also saw cases of arachnoiditis with high protein and with cobweb coagulum .

Later I was working as assistant in Kottayam Medical College . A 54 yr old lady presented as an acute left hemiplegia and without meningeal signs. Her CSF study showed lymphocytic pleocytosis with high protein and low sugar. It was pre scan era in Kerala and a rt carotid angiogram showed only paucity of Rt MCA vessels. She was put on ATT and recovered slowly.

It was when I was in Aleppey as Asst Prof , I came across real unusual presentations of tuberculosis. We were permitted private practice at the time and one day late in the night a young male patient in twenties was brought . The relatives were in real panic along with the patient as he had developed sudden visual loss both eyes .With history of fever and mild neck stiffness ,fundus being normal with total visual loss , I considered optochiasmic arachnoiditis (OCM). Being one with a serious complaint and due to lack of facilities at MCH Aleppey, I referred him to SCTIMST, Trivandrum. The prognosis for recovery of vision is not good in OCM[4]

Sometimes later I happened to see one young boy in late teens with c/o wasting of left hand . There was clinical evidence of C7,8 radiculopathy. LP was done which was normal. Considering this as one like neuralgic amyotrophy, a short course of oral prednisolone 40 mg per day was given for a period of 10 days and tapered off. Neither CT nor MRI were available locally. There was no significant improvement but instead patient developed headache and later Rt hemiparesis . Was admitted and repeat lumbar puncture revealed rise of CSF protein with lymphocytic pleocytosis. He was put on ATT. While on ATT he developed progressive symptoms with hemiparesis on opposite side. In this state he was referred to SCTIMST where they continued ATT.

But his neurological deficits never recovered fully. What would have been initially as localised granuloma at cervical level might have progressed to meningitis with immunosuppression.

It was while in Aleppey a case of tuberculoma of brain with long duration symptoms was published[5]. He had focal motor seizure involving the Rt hand and later developed weakness of the hand . The seizure had started 8 years back on coming to India from Nepal. CT head scan was done at Cochin and showed a small enhancing lesions in the left frontal region corresponding to the hand area(fig1). He was put on ATT and anti epileptics. While on treatment there was increasing weakness of hand which is well known as an immune reconstitution syndrome (IRIS) or paradoxical reaction in tuberculoma which typically occurs within the first six months after initiating treatment.[6] This was managed with short course of steroid and he recovered.

The next patient was an old lady with multiple tuberculomas , one in frontal region and another in cerebellum on CT scan(fig 2,a and b). She presented with ataxia and pyramidal signs on same side. No evidence of pulmonary tuberculosis was picked up. This lady responded to ATT but the treatment had to be continued up to 2 years for the lesions to completely disappear[7]

Later when I shifted my professional activity to Calicut, I was more privileged to have better facilities of staff, investigations like CT etc. I remember a young lady presenting with isolated 6th nerve palsy. CT was normal. No long tract signs. No signs of meningeal irritation. The CSF study was un remarkable except for a few lymphocytes. She was empirically started on steroids along with ATT . She recovered from 6th palsy . On follow up after 4 weeks we reviewed the case and stopped ATT as there was no definite indication. After about 2 weeks patient presented with headache, rt sided hemiparesis. CSF showed definite features of TBM and she was restarted on ATT. But the recovery of the hemiparesis was not satisfactory.

A young lady presented with large multiple tuberculomata with papilloedema and despite surgical intervention could not be revived(fig 3 a and b). The diagnosis can be so challenging sometimes. We had an18 year old boy who had recurrent seizures. CT head scan revealed a contrast enhancing lesion in the temporal region. He was put on ATT. The recovery was not satisfactory. Hence a neurosurgical opinion was sought. They considered the possibility of brain abscess as it had become larger and more cystic. It was operated and the surgeons opined that it looked like pyogenic abscess . He was put on antibiotics. However the pus culture demonstrated AFB. The patient succumbed to the illness. Cerebral abscess may form either from focal tubercular cerebritis or from liquefaction of a tuberculoma in an immuno compromised or even in a immune competent patient[8]

A young girl with recurrent focal seizures came for consultation. The CT head scan showed a

sizable multi loculated lesion with variable enhancement in the left frontal region. It was the pre MRI era and as no definite conclusion could be arrived at radiologically, she was referred to the neuro-surgeon. At surgery, it was diagnosed as tuberculoma . She was put on ATT and continued on AEDs. Despite effective treatment, the girl became a case of intractable epilepsy. This experience stresses the importance of medical treatment in all feasible situations as surgical intervention by it-self can produce a gliotic scar and cause intractable epilepsy.

In this instance it is most appropriate to mention about a 12 year old girl who presented with focal epilepsy . CT head scan showed ring enhancing lesion left frontal region . Her chest xray showed evidence of pulmonary tuberculosis with pleural effusion. The plural tap done in Paediatrics Dept showed high protein with lymphocytic pleocytosis . Mantoux was strongly positive. She was put on ATT along with anticonvulsants . The lung lesion and effusion improved. The ATT was continued for upto 1 year. By this time she developed recurrence of seizure. CT scan showed persisting left frontal lesion after initial decrease in size. The ATT was continued even up to 2yrs and she presented with recurrence of seizures at variable intervals without any other deficit .The problem was in managing the epilepsy and healing of the 'tuberculoma'

With HIV becoming prevalent in the society , we did not feel the upsurge in neurological complica-tions especially in infections like TB as most would have been managed by Medical specialities. Of course there were a few cases of CNS tuberculoma .

It was years later a lady presented with increasing headache and double vision of 2 weeks. She was 34 years, obese and o/e fundus showed bilateral papilloedema. There was no h/o fever, and no meningeal signs on examination. The reflexes were brisk in both lower limbs. Considering the sex and obesity (BMI 27) the possibility considered was IIH.MRI Brain excluded ICSOL. LP was done and it showed an elevated protein, fall in sugar and plenty of lymphocytes. After admission patient developed retention of urine and a catheter had to be put in. The plantars were now extensor though there was no weakness of the lower limbs or sensory deficit. MRI of the spine showed evi-dence of myelitis in the dorsal region. CSF aldolase, PCR test for Mycobacterium, culture for TB all were negative. She was put on ATT with steroids . The latter was tapered after 1 month. The papilledema subsided . The reflexes became normal . She was on ATT for 1 year. At follow up she is doing well . Her mother was also diagnosed with Potts paraplegia elsewhere and was on ATT.

The diagnosis of CNS TB is often made considering clinical and correlating it with CSF biochemistry and microscopy. Being a disease common to our country , a strong clinical suspicion should be

entertained in cases with lymphocytic pleocytosis in CSF with a less acute onset of symptomatology in TBM as against purulent meningitis. CSF of untreated TBM typically shows moderate lymphocytic pleocytosis, moderately elevated protein concentration and low glucose. Acellular CSF has been reported in the elderly and in patients with HIV. It is also not unusual to get polymorphonuclear pleocytosis. Presence of neurological deficits like multiple cranial nerve palsies, long tract signs with sub acute onset is a pointer to this . Papilloedema with or without meningeal signs also helps differentiation from viral or purulent meningitis. Of course in such instances TBM has to be differentiated from partially treated purulent meningitis, with other chronic meningitis like brucellosis, fungal meningitis,non infectious conditions like meningeal carcinomatosis etc.

In the evolution of investigations for diagnosing TBM, tests like CSF aldolase, stearic acid have been less commonly utilised due to the variability in specificity and sensitivity . Tests which were added later including PCR especially Rteal time PCR for antigen testing require minimum concentration of the bacillus in CSF. TB PCR has a sensitivity of 80.5% and specificity of 97.8% which makes it a mandatory test for all suspected CNS TB case[9].The ultimate gold standard tests are demonstration of organism by Zeil Neilson staining of centrifuged deposits of CSF or growing the organism on culture which may be either in a solid medium(Lowenstein Jensen) or in a liquid medium (BACTEC). The former takes up to 8 weeks and the latter 8 days. However the positivity of this test is low as TBM is a paucy bacillary disease[10]. Hence there is still no single diagnostic method that is both sufficiently rapid and sensitive.

Radiological innovations viz CT scan with contrast and MRI of brain has definitely added support to clinical diagnosis in TBM and tuberculoma . Presence of exudates in basal region, hydrocephalus, infarcts, tuberculoma are supportive. Dastur et al reported evidence of TB elsewhere in the body (especially in the lungs and lymphnodes)in almost all the autopsied cases of TBM[11]. In addition to MRI/C T, PET scan may be used to exclude pulmonary and extra pulmonary tuberculosis as its presence is an added supportive point in diagnosis of CNS tuberculosis. Hence clinical suspicion supported by Radiological investigations and CSF biochemistry and cytology help in a presumptive diagnosis of TBM. Empirical treatment with ATT in all suspected cases is the rule as to confirm it by culture or other tests takes time.

In the diagnosis of tuberculoma, there is a long list of differentials. Radiological investigations including MR spectroscopy help but are not conclusive and biopsy and demonstration and culture of the bacillus clinches diagnosis. Empirical treatment with anti TB treatment with clinical and radiological improvement is an indirect way and accepted way of diagnosing CNS tuberculoma. There is no

consensus in duration of treatment in tuberculoma. One view is to follow up the cases radiologically and to continue ATT till the lesion gets calcified. Another view is that radiological persistence implies only persistent immune response and specific drug treatment need be continued for a maximum of 18 months.

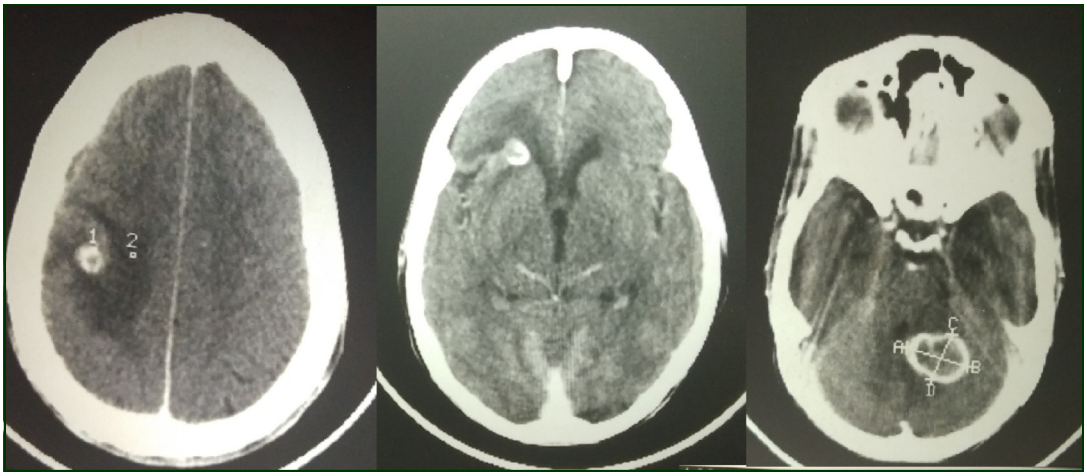

Fig 1 Lt frontal lesion on hand area**Fig2** a and b lesions over lt frontal region and rt cerebellum

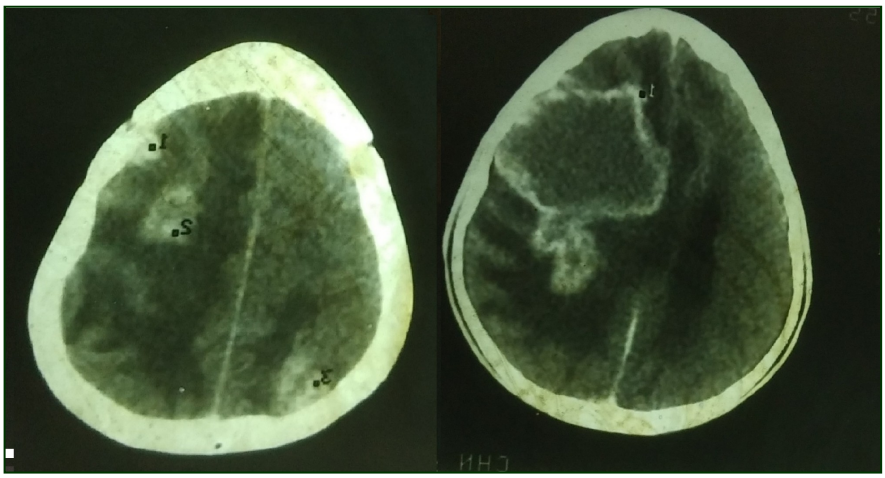

Fig3 a and b multiple ontrast enhancing lesions

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'Service was his life'

Dr. A Simon Hercules
MS, MCh, PhD, FRCS (Ed), MBA.
Medical Director,

A well-respected Neurosurgeon, Dr.Simon Hercules trained at Madras Medical College in Chennai and has been a Fellow of The Royal College of Surgeons of Edinburgh since 2001. In his role as Medical Director and Head of Neurosurgery at New Hope Medical Centre,Dr.Simon Hercules was described as a kind and affectionate man, who was a hardworking surgeon that went beyond the call of duty to help those in need.

The Members of Neurological Society of India Kerala Chapter are extremely shocked and saddened to hear the untimely demise of Dr.Simon, a prominent Neurosurgeon at Chennai.
On behalf of our Society we convey our heartfelt condolences to the bereaved family and pray to God , may his soul rest in peace and also to give strength to the family members to bear the great loss.
And also Society ,strongly condemn the attack by antisocial elements when body was brought and entrust our counterpart, requested the Tamil Nadu Association to do everything possible to persuade authorities to take strong action against the culprits.

The President Dr Gnanadas requested Dr Ram Narayanan and Dr Natarajan to convey our condolences to the family and initiate steps for legal action.
As IMA leadership announced light a candle wearing white Coat at 9pm on 22/4/2020 as our protest against the attack.

Dr Gnanadas -- President
Dr Raj S Chandran -- Secretary
NSI Kerala Chapter

Dr R Ramnarayan ,MCh. ,FRCS
Consultant Functional Neurosurgeon , New Hope Hospital Chennai

VAGAL NERVE STIMULATION (VNS) FOR DEPRESSION AND UNCONTROLLED SEIZURES.

Dr R Ramnarayan, Dr Simon Herculus (late)

Introduction

The vagus nerve, also known as the cranial nerve X, has the most extensive course and distribution of all the cranial nerves. It supplies the viscera. Stimulation of the vagal nerve has been approved as a treatment for refractory epilepsy and for treatment resistant depression. We describe a case of refractory epilepsy of many decades now developing resistant depression.

Case report

A 60 year old man with history of seizures diagnosed at the age of 8 years came to our clinic. He had the first GTCS when he was eight years of age. He has been on treatment for the same from that time. However the seizures were uncontrolled and he was added on more drugs over the last few decades. Presently he was on seven types of drugs some more than once a day. His family claimed he was very regular in medications but still used to get two or three seizures every month. The family complained that for the last decade or so he was having aggressive behavior with episodes of violence and abusive activity. Because of this his wife and children left him. From that time he was slowly becoming depressed and had treid committing suicide atleast 10 times in the last two years. He was on three anti- depressant medications. Clinical examination showed him to be conscious cooperative with no lateralising deficits. Neuropsychological assessment showed good level of intelligence with average memory. The aggressive behavior was assessed by the Modified Overt aggression scale (2). The weighted sum of the scores were 3 for verbal aggression, 4 for aggression against property, 3 for autoaggression and 8 for physical aggression, total of 18 out of 40. The Beck Depression index was 35/40. As he was having both uncontrolled seizures and depression a vagal nerve stimulation surgery was chosen as the best treatment option. He underwent left vagal nerve stimulation insertion and the post-operative period was uneventful. Slowly the device was programmed without much of side effects except some pain in the throat on the left side which disappeared after readjusting the parameters. Now at six months follow up, his aggression score has become 0 for verbal aggression, 1 for aggression against property, 3 for autoaggression and 1 for physical aggression totaling 5 out of 40. His Beck depression score has now become 18 and patient says he is feeling better and better with passing of time. He has now totally stopped three out of seven anticonvulsants he was on and tapering of atleast one or two is being planned. In the last six months he just had three episodes of GTCS that too in the initial phase of adjusting the drugs.

Discussion

The first vagal nerve stimulation was implanted by Penry et al in 1988. This was subsequently approved by FDA as an adjunctive treatment for refractory epilepsy in 1997 (1) and for severe, recurrent unipolar and bipolar depression in July of 2005 (3) The minimum age for vagal nerve stimulation therapy for epilepsy is 12 years, and for depression, 18 years. Hoarseness and cough are the most common side effects. If used as adjunctive therapy, vagal nerve stimulation has shown better control of seizures or depression at smaller doses of antiepileptic or anti-depressive medications and also results in decreased dose-dependent side effects. Vagal nerve stimulation therapy appears safe as an adjunctive treatment for drug-resistant epilepsy and depression (3).

Vagus nerve stimulator implantation is usually done on the left side so as to avoid cardiac complications (the right vagus nerve supplies the sinoatrial node, while the left innervates the atrioventricular node). This prevents the untoward effect of cardiac dysrhythmia. The mechanism of action is not still understood, including afferent vagal projections to seizure-generating regions of the brain and desynchronization of hypersynchronized cortical activity. The most common complications of VNS therapy include hoarseness, throat pain/dysphagia, coughing, and shortness of breath (4).

Boylan et al (5) reported that up to 63% of patients with resistant epilepsy had undiagnosed depression. This depression is associated with and a powerful predictor of reduced quality of life in epilepsy. Moreover antiepileptic medications can cause depression. In older patients, major depression has been shown as a risk factor for seizures. Spindler and coworkers (6) assessed 59 patients with different subtypes of disorders of depression as a comorbidity of epilepsy. Symptoms of all subtypes of depression ameliorated in response to VNS in all patients. Seizure reduction of at least 50% was achieved in two out of three of all patients two years after VNS. This study demonstrated the beneficial effect of VNS in the treatment of patients with resistant epilepsy associated with depression.

Yang and Phi (7) opined that VNS yields a more than 50% reduction in seizures in approximately 60% of recipients, with an increase in reduction rates as the follow-up duration increases. The complication rate of VNS is 3-6% and infection is the most important complication. They concluded that VNS is beneficial and effective in the treatment of epilepsy. Morace et al (8) looked at 32 patients with drug resistant epilepsy who underwent VNS. Resective surgery had previously been excluded in all cases. All patients had complex partial seizures and follow up was upto 9 years after VNS implantation. Patients were considered responders when a reduction of seizures of more than 50 % was reported. And twelv**e p**atients out of 29 followed up were responders. They confirmed that VNS is a safe procedure and a valid palliative treatment option for drug-resistant epileptic patients not suitable for resective surgery.

Mohr, Rodriguez, Slavíčková and Hanka (9) reviewed the available clinical evidence and neurobiology of VNS in treatment resistant depression. Four clinical trials with 355 patients were examined. . VNS demonstrated steadily increasing improvement with full benefit after 6-12 months, sustained up to 2 years. Patients who responded best had a low-to-moderate antidepressant resistance. Aaronson et al (10) compared whether adjunctive vagus nerve stimulation (VNS) with treatment as usual in depression has superior long-term outcomes compared with treatment as usual. This 5-year, prospective, open-label, nonrandomized, observational registry study was conducted at 61 U.S. sites and included 795 patients who were experiencing a major depressive episode (unipolar or bipolar depression) of at least 2 years' duration or had three or more depressive episodes (including the current episode), and who had failed four or more depression treatments (including ECT). The results indicated that the adjunctive VNS group had better clinical outcomes than the treatment-as-usual group, including a significantly higher 5-year cumulative response rate and a significantly higher remission rate. A subanalysis demonstrated that among patients with a history of response to ECT, those in the adjunctive VNS group had a significantly higher 5-year cumulative response rate than those in the treatment-as-usual group. A similar significant response differential was observed among ECT nonresponders.

In our study also the patient has significant reduction in the depression score and a good number of anticonvulsants have been reduced. The patient says his quality of life has improved significantly.

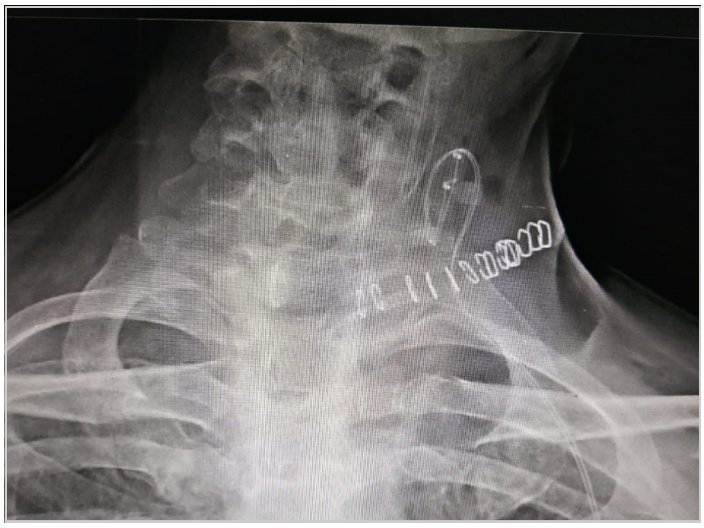

Figure 1

postoperative X ray showing the vagal nerve stimulator in position.

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Expanding Indications for Deep Brain stimulation

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Deep brain stimulation has been in use for more than three decades and its indications are expanding .DBS is used most commonly for control of motor symptoms of Parkinson's disease. Subthalamic nucleus was found to be most suitable target in controlling the symptoms like tremor rigidity and bradykinesia. Freezing and postural imbalance do not respond well to the procedure. Timing of surgery in PD patients is also undergoing reconsideration from advanced PD to early DBS for better results. Ever since its introduction DBS, it has been tried for other indications.DBS has qualified under the FDAs Humanitarian Device Exception (HDE) for a number of neurological disorders including stimulation of GPi and STN for dystonia in 2003, stimulation of the anterior limb of the internal capsule for obsessive compulsive disorder (OCD) in 2009and closed-loop stimulation for epilepsy in 2013. Expanding indications of DBS include obesity, Obsessive compulsive disorder, depression, tourette syndrome, trigeminal vascular cephalalgia, epilepsy and meigs syndrome and minimal cognitive state

Sub thalamic nucleus (STN) is found to be most useful and most frequently used target in Parkinson's disease. Several trials which used globus pallidus has done but none have demonstrate any superiority over subthalamic nucleus in Parkinson's patients.

The ideal candidates for DBS are below seventy years and having marked motor fluctuations, as evidenced by levodopa responsive off periods and drug induced dyskinesia, on attempting dose escalation of levodopa.DBS significantly prolongs the on phase in such patients. A second group of patients who will benefit from DBS are those with bad tremors unresponsive to drug therapy(Grill 2005). Levodopa induced reduction in motor symptoms as demonstrated by a 30% reduction in UPDRS (Unified Parkinson Disease Rating Scale) are best selected for DBS surgery.

FDA has approved thalamic DBS in 1997 for tremor and GPi and STN DBS in 2003 for Parkinson's disease. DBS has qualified under the FDAs Humanitarian Device Exception (HDE) for a number of other disorders which include stimulation of GPi and STN for dystonia approved in 2003, stimulation of the anterior limb of the internal capsule for obsessive compulsive disorder (OCD) in 2009, and closed-loop stimulation for epileptic patients in 2013(TEKRIWAL and BALTUCH 2015).

DBS for Parkinson's disease:

Neurosurgical procedure to control PD symptoms was in use even before the discovery of levodopa. Subcortical neurostimulation was used to localize the area to be lesioned prior to the ablative procedure (Albe-Fessard 1973). These procedures later became more acceptable because of the reversibility and therapeutic efficacy. (Benabid et al. 1987) .DBS has now largely replaced the lesional surgeries for treatment of PD (Groiss et al. 2009).

Deep brain stimulation is mainly done to improve motor control in PD. Factors considered for patient selection include age, duration of illness ,degree of functional disability, psychological status and any features of dementia in the patient(Rowland, Sammartino, and Lozano 2017).Symptoms resistant to levodopa treatments are resistant to DBS also. Freezing of gait and postural imbalance do not respond well to DBS unlike tremor and rigidity. Candidates should achieve an improvement of at least 30% in the preoperative L-dopa challenge measured by the United Parkinson's Disease Rating Scale (UPDRS) motor score. All patients need to undergo thorough neuropsychological evaluation to rule out psychosis, depression and hallucinations.

The symptoms best responding to bilateral DBS are the motor symptoms and L-dopa–induced dyskinesias in PD. Symptoms that are best controlled by DBS can be classified as L-dopa-sensitive OFF symptoms, L-dopa-induced dyskinesias (such as peak-dose hyperkinesia and biphasic dyskinesia), OFF-dystonia, and tremor.

Timing of surgery: In the initial years typical DBS cases were with long history of PD. Several randomized studies showed an advantage of DBS compared with best medical treatment in this group. However, very late stages of PD are often accompanied by axial motor symptoms and non motor symptoms like cognitive decline, which may not benefit from DBS and potentially even impede the overall improvement from DBS treatment. As such, improvement of appendicular motor symptoms does not necessarily result in a return of independency.

Careful patient selection is utmost importance to avoid unsatisfactory outcome after DBS. Careful clinical examination should be done to exclude patients with Parkinson plus syndrome as they do not respond to DBS.

German guidelines recommend the following guidelines for patient selection for DBS in PD.

1. Presence of motor fluctuations including levodopa-sensitive off symptoms or treatment-induced dyskinesia.
2. Tremor, which cannot be satisfactorily treated with medication
3. A levodopa-induced reduction of motor symptoms by >33% of the Unified Parkinson Disease Rating Scale (UPDRS), where tremor may be disregarded from the calculation as it may be refractory to levodopa treatment while still responding well to DBS;

Exclusion of dementia, relevant psychiatric or somatic comorbidity, or general contraindication to undergo neurosurgical interventions(Grill 2005).

Earlt stim study examined the efficacy of STN DBS treatment in PD patients suffering from early motor fluctuations and hence a less advanced stage of the disease and was compared with the best medical treatment (Cabrera, Goudreau, and Sidiropoulos 2018). In November 2015, the US Food and Drug Administration (FDA) approved the use of deep brain stimulation (DBS) therapy in people with Parkinson disease (PD) "of at least 4 years duration and with recent onset motor complications, or motor complications of longer-standing duration that are not adequately controlled with medication. Early stim was a randomized, prospective, multicenter, parallel-group clinical trial in Germany and France involving 251 patients with PD. The trial compared the effect of DBS of the subthalamic nucleus (STN) on quality of life in comparison to best medical treatment (BMT), with the specific aim "to assess the use of this therapy in earlier stages of the disease, when motor complications have just developed and before patients are significantly affected in their social and occupational functioning. The trial reported significant improvements for the primary and secondary outcome measures in the STN-DBS group compared to the BMT group. Another study also showed STN DBS with long and short disease duration was shown to improve quality of life(QOL) in patients with an age of more than 60 years (Dafsari et al. 2018).

Tremor

Patients with medically refractory tremor can be effectively treated with deep brain stimulation. The first indication for which dbs got FDA approval was for tremor in PD and got approved in 1997 (Lake, Hedera, and Konrad 2019).VIM thalamic stimulation, Zona Inserta of STN are commonly used sites of stimulation for tremor predominant PD.

Essential tremor which is inherited autosominal dominant is the most common type of tremor. Most of the cases respond well to oral medication but can rarely become medically refractory. Thalamic DBS (VIM Nucleus) gives significant improvement symptomatically in such patients. The quality of life improves significantly after DBS. Long-term follow up showed sustained benefit even after 10 years after surgery(Huss et al. 2015).Patients with distal tremor respond best to DBS.

Several types of tremor rating scales like The Fahn-Tolosa-Marin (FTM) tremor rating and The Essential Tremor Rating Assessment Scale (TETRAS) are used to assess the clinical severity. Moderate to severe tremor based on these scales are typically taken up for DBS surgery. Awake surgery is preferred as the clinician can assess the on table response of tremor for test stimulation. Appendicular tremor respond best to DBS but significant improvement in axial symptoms can also achieved (Lake, Hedera, and Konrad 2019).

Dystonia

Stereotaxic thalamic stimulation for torticollis was first described in late 1970s by Mundinger in 7 patients (Mundinger 1977). After this there were no major attempts of using this technique for almost two decades. A series of DBS studies, were attempted in 1999 using unilateral pallidal stimulation for focal cervical dystonia and bilateral pallidal stimulation for generalized dystonia, and were found to be giving successful in relief of Symptoms(İşlekel, Zileli, and Zileli 1999)(Kumar et al. 1999).FDA approved used of DBS in dystonia in 2003(Dietz and Neimat 2019). The best target in Dystonia was found to be the GPi and is the most commonly used one, and has repeatedly shown efficacy over the years(Dressler, Altenmüller, and Krauss 2018). Targeting GPi for primary generalized dystonia shows significant clinical improvements, ranging from 34% to 92% (Jill L Ostrem and Starr 2008)(Cersosimo et al. 2008).

STN stimulation has also been tried for dystonia and has also shown that as an appropriate alternative to the more commonly targeted GPi. A study report a significant improvement on the Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) for 9 patients with cervical dystonia who received bilateral STN DBS(J L Ostrem et al. 2011). Another study with 20 patients having primary dystonia to determine the long-term treatment efficacy and showed sustained clinical improvement over 3 years in 14 of the patients with isolated dystonia, reported 70% improvement in dystonia rating scale (Jill L Ostrem et al. 2017).

Epilepsy

In spite of conventional treatments like medical management and epilepsy surgery, many epilepsy patients have uncontrolled seizures. Since the 1970s interest has grown in invasive intracranial neuro-stimulation as a treatment for these patients. Intracranial stimulation includes both deep brain stimulation (DBS) (stimulation through depth electrodes) and cortical stimulation (subdural electrodes).The sites of stimulation tried with varying success rate include anterior thalamic stimulation, centro-median thalamic stimulation, Cerebellar stimulation, Hippocampal stimulation, Nucleus accumbens stimulation and cortical loop ictal stimulation (Sprengers et al. 2017). In 1997, VNS was approved by the FDA for the treatment of epilepsy, but with this treatment seizure freedom is rare and 25% of patients receive no benefit from the procedure.

The anterior nucleus of the thalamus (ANT) is a particularly interesting target, and Stimulation of the Anterior Nucleus of the Thalamus for Epilepsy (SANTE) trials was designed to test this. The study reported a 56% reduction in seizure frequency 2 years after implantation and although approval was granted in Europe and Canada, the FDA did not approve(R. Fisher et al. 2010)the procedure. Stimulation of the fornix and hippocampus are used in intractable mesial temporal lobe epilepsy. Another target of interest, is the centromedian nucleus of the thalamus (CMN), and is the neurologic gate-keeping which is altered in epileptics. Small-scale clinical trials have yielded encouraging results of reducing generalized seizures by > 50% up to a year after implant (R. S. Fisher et al. n.d.).

Gilles de la Tourette (GTS)

Imaging studies have implicated impaired thalamic Dopaminergic system in etiology of tourette syndrome (Worbe et al. 2012). The most common regions employed include the thalamus, globus pallidus and nucleus accumbens (NAc) .

Depression

Depression is a common psychiatric disorder in general population and often responds well to medical therapy. Sometimes depression is resistant to medical therapy, neurosurgical interventions such as lesion procedures or deep brain stimulation offer possible therapeutic options. Neuroimaging studies support the involvement of multiple interconnected regions that modulate different neural networks associated with various depressive symptoms; involved structures are both cortical and subcortical and include the subgenual cingulate, orbitofrontal cortex, medial prefrontal cortex, medial temporal lobe, ventral striatum containing the nucleus accumbens, and regions of the thalamus and brainstem (Schlaepfer and Bewernick 2013). The first wave of trials evaluated the anterior limb of the capsula interna (ALIC), anterior cingulated cortex (Cg25), ventral striatum, medial forebrain bundle (mFB) and subcallosal cingulate gyrus (SCG), ubcallosal cingulate gyrus targeted in 17 patients with refractory MDD by Holtzheimer et al., and showed reduction in reported levels of depression in 92% of patients (Holtzheimer et al. 2012) but these results were not reproduced in subsequent trials (Dougherty et al. 2015).

Obsessive Compulsive Disorder

Surgery for OCD is reserved for patients with the most severe cases of the disease, when pharmacological and psychotherapeutic alternatives fail. The targets presently reported in the literature on ablative and DBS procedures were selected empirically and/or as the result of an understanding of the presumed pathophysiology of OCD. Several such targets have been used, including the nucleus accumbens, the anterior limb of the internal capsule, and ventral portions of the striatum. The differential therapeutic success of these targets suggests that each may have a distinct role in OCD-relevant cognitive loops (Lipsman, Neimat, and Lozano 2007).

Obesity

Lateral hypothalamic stimulation is known to alter feeding behaviour. To assess the viability of DBS, a pilot study was conducted at the Pittsburgh, PA. In 2013, they reported three intractable obese patients who were implanted in an effort to see how safely such a procedure could be done in this patient population. Though the metabolic rate improved, no much reduction in obesity was noted (Whiting et al. 2013).

Headache

Specific activation patterns in various primary headaches have been revealed by Neuroimaging studies. In the trigeminal autonomic cephalalgias, neuro-imaging findings support the hypothesis that activation of posterior hypothalamic neurons have a pivotal role in the pathophysiology and prompted the idea that hypothalamic stimulation might inhibit this activation to improve or eliminate the pain in intractable chronic cluster headache and other trigeminal autonomic cephalalgias. Hypothalamic stimulation may also be beneficial in patients with SUNCT (short-lasting, unilateral, neuralgiform headache attacks with conjunctival injection and tearing)—a disorder with close clinical and neuro-imaging similarities to the cluster headache (Leone and Proietti Cecchini 2016).

Meigs syndrome

Meige syndrome is a type of segmental dystonia characterized by various subtypes of orofacial–cervical dystonia and blepharospasm. It mainly affects adults, and blepharospasm is the most common symptom in the initial stages of the disease. Both STN DBS and GPI DBS has been successfully used in Meigs syndrome (Wang et al. 2019). STN DBS is reported superior outcome when compared to GPI DBS by certain group (Wang et al. 2019).Patients with severe symptoms at the time of surgery respond poorly to DBS therapy.

Conclusion

Deep brain stimulation requires careful patient selection for favorable functional outcome in any disorders. The main targets of DBS for PD are STN and GPi. In addi-tion to these targets, VIM is an effective target for several types of tremor including Parkinson tremor. Gpi is the best target for

dystonia. Further studies will be required for fine tuning of targets in other disorders like depression, OCD and Tourette syndrome for optimal results.

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A brief review of surgically treatable causes of dementia

Abstract:

Background:

Dementia is a clinical condition of decline in cognitive function and loss of intellectual capacity in multiple domains. Some conditions which present clinically as dementia are reversible with treatment.

Objective, materials and methods:

Aim of the review is to update the current knowledge on the causes of dementia, which are surgically treatable. A search was done in Pubmed , EMBASE and Scopus.

Discussion:

Neurosurgical conditions among the causes are normal pressure hydrocephalus [NPH], chronic subdural hematomas(CSDH), tumors, and head injury. Imaging by CT(Computerised Tomography) or MRI (Magnetic Resonance Imaging) will help to diagnose the structural brain lesions.

Normal pressure hydrocephalus: Interstitial edema in periventricular white matter and prefrontal regions leads to psychomotor slowing. CT or MRI findings are ventriclomegaly, periventricular edema, and fourth ventricular flow void. Those patients who improve after the lumbar spinal tap test will have better outcome with Ventriculoperitoenal(VP) shunt.

Chronic subdural hematoma: This develops three or more weeks following mild to moderate trauma and the risk factors are cortical atrophy and consumption of oral anticoagulants and antiplatelets. Bilateral SDH can cause rapid progression of clinical features. Burr-hole craniostomy is the ideal surgical procedure.

Brain neoplasms: Intracranial tumors can presenting with cognitive impairment are glioma, metastasis, meningioma, and prolactinoma. High grade glioma can cause rapid deterioration. Most of the patients with meningioma have cognitive deficits which improve after surgery.

Head injury: Chronic traumatic encephalopathy (CTE), is a type of dementia. There is an association between moderate and severe TBI and dementia. Early surgery leads to favourable outcome in patients with traumatic intracerebral hematomas.

Conclusion:

Some causes of dementia respond to surgical treatment. They are normal pressure hydrocephalus, chronic subdural hematoma, brain neoplasms and traumatic brain injury. Patients with rapidly progressing symptoms should have early CT or MRI to rule out these conditions.

Key words:

Dementia, normal pressure hydrocephalus, chronic subdural hematoma, brain neoplasms, traumatic brain injury.

Introduction:

Dementia is a clinical condition of decline in cognitive function which results in impaired independent social or occupational functioning[1,2]. It is characterized by loss of intellectual capacity in multiple domains including learning and memory, language abilities, reading and writing, praxis, and appreciation of visuospatial information[3]. The clinical features depend on the lobes which are involved. Frontal dysfunction produces dysphasia, changes in personality, and difficulty in judgement. Parietal lobe involvement results in dysgraphia, dyscalculia and agnosia. Dysfunction of limbic system leads to impaired memory. Some conditions which present clinically as dementia are reversible with treatment[4,5].

Objective, materials and methods:

Aim of the review is to update the current knowledge on the causes of dementia, which are surgically treatable. A search was done in Pubmed , EMBASE and Scopus. All English articles are studied descriptively without any restriction of period.

Discussion:

Dementia is defined as memory impairment and one or more of the following cognitive disturbances:aphasia , apraxia, agnosia, and disturbance in executive functioning[4]. There are certain treatable disorders with clinical profile progressing to dementia. Early treatment can reduce the symptoms before dementia is established. Neurosurgical conditions among the causes are normal pressure hydrocephalus [NPH], chronic subdural hematomas(CSDH), tumors, and head injury[4,5,6,7,8]. Detailed history and cognitive evaluation are the first steps of treatment[5]. Imaging by CT (Computerised Tomography) or MRI(Magnetic Resonance Imaging) will help to diagnose the structural brain lesions[6,7,8].

Normal pressure hydrocephalus:

The clinical triad consists of apraxia of gait, urinary incontinence and dementia[7,9]. The pathology is enlargement of cerebral ventricles, which may be idiopathic associated with senility or secondary due to subarachnoid hemorrhage, meningitis, intracerebral hemorrhage, brain tumor or head trauma[9]. Interstitial edema in periventricular white matter and prefrontal regions leads to psychomotor slowing, impaired attention, and executive dysfunction[10]. CT or MRI findings are ventriclomegaly defined by Evan's index of greater than 0.3 (the maximal ventricular width divided by the largest biparietal distance), enlargement of temporal horn, periventricular edema, and fourth ventricular flow void(fig.1a). A lumbar spinal tap test with removal of 30 ml of CSF (cerebrospinal fluid) can predict the response to shunting. Those patients who improve after the tap test will have better outcome with Ventriculoperitoenal(VP) shunt(fig.1b). VP shunt placement leads to marked improvement of gait, but less of cognition[11,12].

Chronic subdural hematoma:

CSDH occurs in the elderly and can present with cognitive and memory disturbances and is a leading cause of reversible dementia[7,13]. This develops three or more weeks following mild to moderate trauma and the risk factors are cortical atrophy and consumption of oral anticoagulants and antiplatelets[13]. Rupture of the bridging veins is the starting point. Bilateral SDH can cause rapid progression of clinical features[14]. CSDH appear hypodense in CT scan[15](fig.2a). Bilateral isodense CSDH is better diagnosed by MRI. MRI can show multiple loculations and membranes also. Burr-hole craniostomy is the ideal surgical procedure routinely(fig.2b). Twist drill craniostomy is suitable for those who have high risk for surgery. Craniotomy is indicated for those with multiple membranes and organized SDH. The cognitive impairment is usually reversible with early surgical evacuation[16].

Brain neoplasms:

Intracranial tumors can present with cognitive impairment[7]. Common causes are glioma, metastasis, meningioma, prolactinoma etc[7,17]. High grade glioma and gliomatosis cerebri have been shown to present like dementia[17,18]. High grade glioma can cause rapid deterioration[17](fig.3a). Surgical decompression(fig.3b) followed by radiotherapy and chemotherapy may improve survival in case of high grade glioma. Cognitive impairment is common in brain tumor survivors after brain irradiation[19]. Most of the patients with mencial or occupational functioning[1,2]. It is characterized by loss of intellectual capacity in multiple domains including learning and memory, language abilities, reading and writing, praxis, and appreciation of visuospatial information[3]. The clinical features depend on the lobes which are involved. Frontal dysfunction produces dysphasia, changes in personality, and difficulty in judgement. Parietal lobe involvement results in dysgraphia, dyscalculia and agnosia. Dysfunction of limbic system leads to impaired memory. Some conditions which present clinically as dementia are reversible with treatment[4,5].

ingioma have cognitive deficits which improve after surgery[20]. Olfactory groove meningioma may present as pseudodementia due to compression of the frontal lobe[21]. The tumor may grow to a large size before diagnosis, since focal neurological deficits are often absent. Donepezil can improve cognitive functions in brain tumor survivors[22].

Head injury:

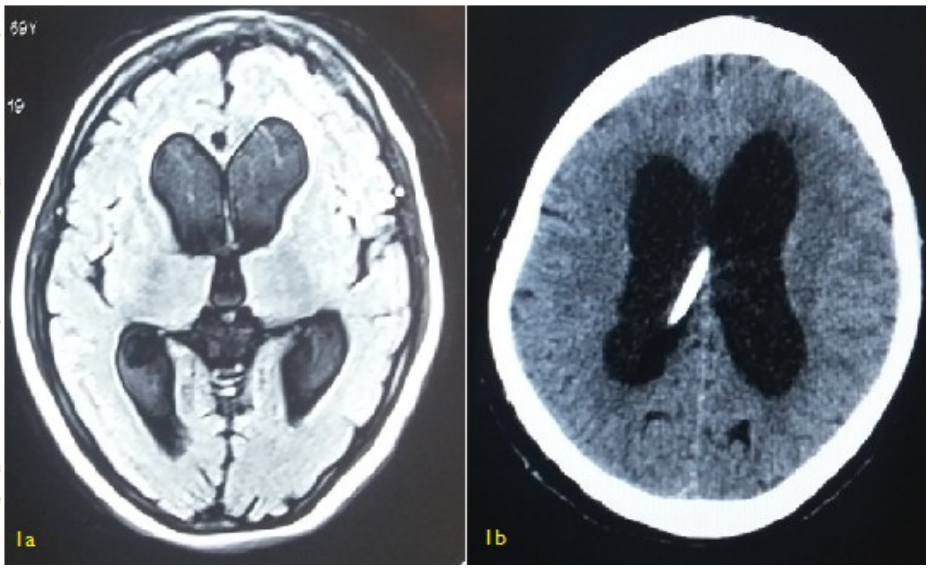
Traumatic brain injury(TBI) is associated with dementia[23]. Professional boxers experience repeated mild TBIs and get chronic traumatic encephalopathy (CTE), a type of dementia. There is an association between moderate and severe TBI and dementia[24]. Studies have shown that TBI leads to neurodegeneration. Multiple Intracerebral hemorrhages and contusions lead to impairment of consciousness and cognitive decline(fig.4a). Surgical Trial In Traumatic intraCerebral Haemorrhage (STITCH), showed that early surgery leads to favourable outcome in patients with traumatic intracerebral hematomas[25](fig.4b).

Conclusion:

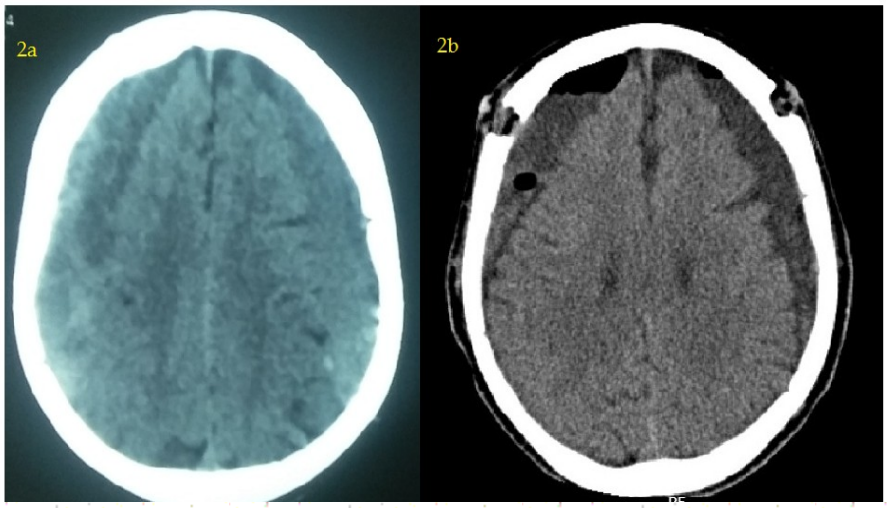
Dementia is usually a progressive decline in cognitive function. But some causes respond to surgical treatment. They are normal pressure hydrocephalus, chronic subdural hematoma, brain neoplasms and traumatic brain injury. Patients with rapidly progressing symptoms should have early CT or MRI to rule out these conditions.

Figures:

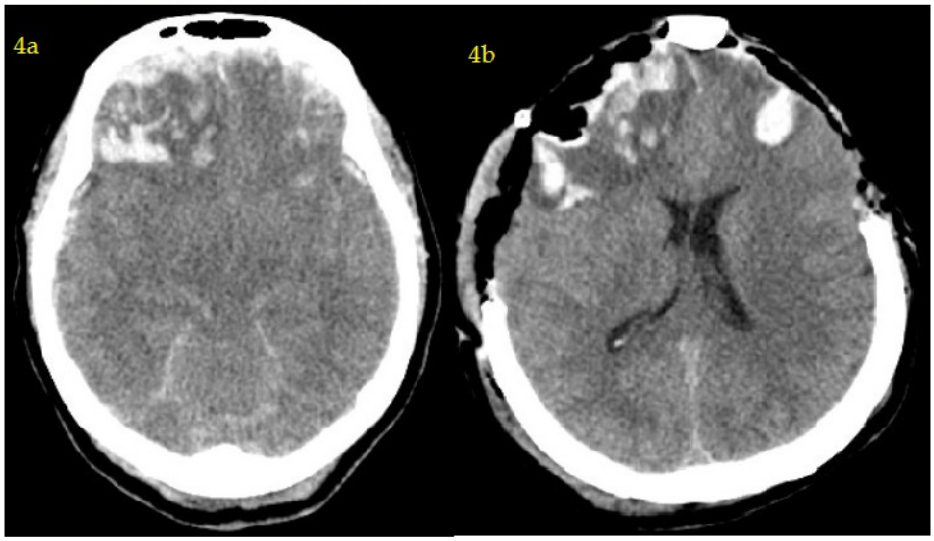
1a. MRI picture showing NPH; b. CT scan showing VP shunt.



2a. CT brain showing bilateral SDH; b. CT scan showing bilteral burrhole drainage.



4a. CT brain showing bifrontal hemorrhagic contusions;
b. CT brain after decompression.



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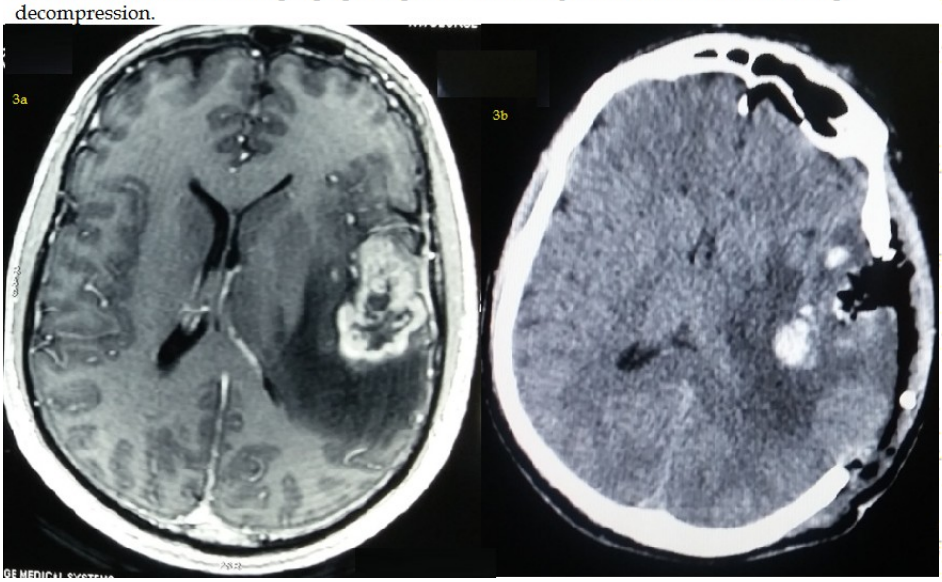
3a.MRI with contrast showing high grade glioma of left temporal lobe; b.CT brain showing decompression.



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Hashimoto Encephalopathy: A counterintuitive approach

Neurologists love to reason. Even though exhausting at times, the very process of collecting information via history taking, physical examination and pertinent investigations followed by a careful analysis and deductive reasoning form the core process of neurological diagnosis. At times, we go further and treat the patient and the response to the treatment forms the strongest pillar of our diagnosis. Hashimoto encephalopathy is a disorder known to us for the past half century is a good example of the above and it was rechristened to Steroid Responsive Encephalopathy associated with Autoimmune Thyroiditis (SREAT) in 2006. Lord Brain and associates originally described this entity in the year 1966 in a middle aged patient already diagnosed with Hashimoto's Thyroiditis.1 He had a episodic neurological illness with multiple clinical manifestations in the form of alterations in the mood and consciousness, stroke like episodes variably inducing transient aphasia, right or left hemiparesis, hemisensory deficits, hemianopia and monocular blindness. He also had involuntary movements, hallucinations and agitated confusion. He was worked up as diligently as possible in that era. The CSF study showed mild pleocytosis and protein elevation. Carotid angiography was unremarkable. The Electoencephalogram showed non specific theta and delta slowing. He was treated with prednisone and anticoagulant but little consistent clinical response was noted. The illness lasted more than one year and got resolved spontaneously. The next half century witnessed the establishment of this entity in the field of neurology while always remaining controversial for multiple reasons. A subacute progressive or relapsing encephalopathy was the core clinical feature described in literature. But seizures, status epilepticus, stroke like episodes, myoclonus, tremor and psychiatric manifestations were also considered to be characteristic clinical manifestations of this condition.However these features are not usually specific for any condition and meticulous workup is needed to rule out vasular, infectious, metabolic and toxic causes. When workup revealed nothing else and serum thyroid autoantibody titre was detected to be elevated a presumptive diagnosis of Hashimoto encephalopathy was made and steroid treatment was initiated mostly considering an immune mediated pathogenesis. A sustained clinical response established the firm diagnosis of Hashimoto encephalopathy. The usual investigations such as EEG, neuroimaging and CSF profile were either normal or abnormal in a nonspecific way. But the nosological status remained foggy, especially about similar cases without steroid responsiveness. The pathogenesis the disorder invoked multiple hypotheses including direct thyroid antibody mediated disorder, lymphocytic vasculitis, nonvasculitic inflammation and TRH toxicity but none have been proved till date. In 1996 in a review article two clinical types were described- a vasculitic type and a diffuse progressive type.2 A decade later Castillo et al argued that steroid responsiveness should be an absolute requirement for establishing this diagnosis.

They postulated the new term Steroid Responsive Encephalopathy associated with Autoimmune Thyroiditis (SREAT) in preference to the older term Hashimoto Encephalopathy.3 A comprehensive diagnostic criteria was also proposed. 1. Encephalopathy characterised by cognitive impairment and at least one of the following- neuropsychiatric features, myoclonus, seizures and focal deficits. 2. Elevated serum thyroid autoantibodies. 3. Euthyroid or mildly hypothyroid status. 4. No evidence in the blood urine or CSF of a infectious, toxic or a metabolic process. 5. No serological evidence of neuronal voltage gated calcium potassium or other 'currently' recognised antibodies. 6. No finding in neuroimaging studies that indicate vascular, neoplastic or other structural lesions to explain the encephalopathy. 7. Complete or near complete return to the patients baseline neurologic status following corticosteroid treatment.

All the criteria were required for establishing the diagnosis. The concepts and the term SREAT was widely accepted by medical community in general. Many experts especially endocrinologists were skeptical about these concepts. The population prevalence of thyroid auto antibodies is pretty high. Around 11% of healthy adult population harbour anti TPO antibodies and this number increases with age. To date there is no conclusive evidence that any of the three thyroid autoantibodies (anti TPO, anti Thyroglobulin or anti TSH receptor antibodies) bind neurons. One study could demonstrate presence of these antibodies and circulating immune complexes in CSF. Auto antibodies against amino terminal of alpha enolase were proposed as a more specific marker of Hashimoto encephalopathy by some Japanese investigators but was not consistently reproduced in practice. The righteousness of using steroid response as a diagnostic marker was also challenged. Unless a drug corrects a specific metabolic defect or acts at a very specific target the response to treatment has got very little specificity of course. Case reports of isolated chorea cerebellar ataxia or myelopathy due to Hashimoto encephalopathy can be found in the literature but cautious interpretation is advisable in view of the points already discussed. Autoimmune neurology has under gone a revolution in past two decades and is making constant progress.Previously immune mediated encephalitis was considered synonymous with a paraneoplastic process associated with various malignancies. These disorders were not very responsive to immunotherapy. The antibody markers, typically targeting intracellular antigens were not considered to have a direct pathogenic role. In contrast the newly described disorders with antibodies predominantly against cell surface or synaptic targets were less associate with cancer and demonstrated excellent treatment response oftentimes. In current parlance these these disorders are considered two disease categories the former being called paraneoplastic encephalitis and the latter, simply autoimmune. The initial focus was identifying the specific clinical syndromes based on typical clinical features followed by antibody testing and if positive proceeding to immunotherapy. However as science advanced the myriad ways these disorders can present became known. The access to antibody testing and the inherent delay in getting the results were also concerning. The response to immunotherapy was also not very fast in many cases making it a less reliable criterion to be useful in early diagnosis. So in 2016 an expert panel suggested a practice guideline where they made a generic criteria to identify autoimmune encephalitis on the basis of initial clinical evaluation.4 1. Sub acute onset (rapid progression less than three months) of working memory deficits, altered mental status or psychiatric symptoms. 2. At least one of the following a. New focal CNS findings. b. Seizures not explained by a previously known seizure disorder. c. CSF pleocytosis. d. MRI features suggestive of encephalitis. 3. Reasonable exclusion of alternative causes. Once a generic diagnosis was made an effort is made to identify the specific syndrome. Significant number of cases, syndrome specific features will be lacking no antibody will be positive and a diagnosis of antibody negative autoimmune encephalitis will be made. In such cases if the thyroid antibody is positive a diagnosis of Hashimoto encephalopathy will be made. (A specific set of criteria is available to guide this decision.) This approach also has inherent problems. Many cases of suspected Hashimoto encephalopathy do not have surrogate markers of CNS inflammation in CSF or MRI. The pattern of steroid response if it occurs, is usually faster than that is observed in antibody mediated encephalitis.

A recent article published in 2020 reported poor predictive value of current diagnostic criteria for steroid responsiveness, the poor disease specificity of thyroid auto antibodies and emphasized the need for meticulous exclusion of other conditions.

The 5 criteria used in this study may be considered the current diagnostic criteria for this disorder. (All criteria need to be satisfied.) 1. Sub acute onset of cognitive impairment, psychiatric symptoms or seizures. 2. Euthyroid status or mild hypothyroidism. 3. Serum TPO antibodies >200 IU/mL 4. Absent neural antibodies inn serum and CSF 5. No evidence of infectious, toxic, metabolic, vascular, or tumoral causes that could explain the symptoms.

.(The cut off value for the antibody titre is arbitrary and no specific normal limits have been established.)

So in conclusion, the the diagnosis is still valid in the current era, provided a thorough workup has been done. Calling any neurologic syndrome without an apparent cause and positive Thyroid autoantibodies as Hashimoto encephalopathy should be discouraged. As autoimmune neurology advances further, we may understand the nuances of this mysterious entity further. Whether what we call Hashimoto encephalopathy is a heterogenous mixture of various disorders is a very likely possibility and answer to that question may be provided by future studies.

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BEST PAPER AWARD ESSENCE 2020
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SHOULD NEUROSURGEONS BE AGGRESSIVE IN TREATING SPONTANEOUS INTRA PARENCHYMAL HEMATOMAS?

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Abstract

Introduction

Management of intra parenchymal hematoma is always a controversial topic, still a life threatening entity which needs multidisciplinary approach.13% of all strokes constitutes hemorrhagic. Mortality and morbidity of patients depends on the status of initial presentation. This study deals with effectiveness of decompressive craniectomy(DC) with hematoma evacuation in reducing the mortality.

Materials and methods

Patients with spontaneous intra parenchymal hematomas were selected and those having herniation syndrome were considered for surgery. Patients were evaluated after 30 days and 6 months and mortality was calculated.

Results

420 patients were selected and after excluding other pathologies like, aneurysm bleed, tumor bleed, and 322 patients were studied. 128 patients were selected for surgery, in that 106 patients underwent DC with hematoma evacuation and 22 patients who were not willing for surgery were considered as control group. Mortality of patients were calculated at 30 days and 6 months and compared. Patients were divided into 3 groups according to ICH-GS score. Group with high scores had both high mortality in both surgical and control group, while in other 2 groups surgical intervention could significantly reduce the mortality.

Conclusion

Surgical intervention can effectively reduce the mortality in spontaneous intra parenchymal hematomas.

Introduction

Decompressive craniectomy (DC) is considered as a satisfactory treatment modality in space occupying lesions, acute subdural hematomas, ischemic stroke, cavernous sinus venous thrombosis. Management of hypertensive intra parenchymal hematoma is always a controversial topic, still a life threatening entity which needs multidisciplinary approach. DC with hematoma evacuation reported in studies. The goal of DC with hematoma evacuation is to prevent the mass effect resulting in mechanical complications and the toxic degradation of hematoma. Mortality and morbidity of patients depends on the status of initial presentation. Mortality is considered high in the first 72hours reporting up to 40%. DC is considered as an effective measure to reduce the early mortality. The aim of this study is to evaluate the feasibility of DC with hematoma evacuation for spontaneous intra parenchymal hematoma (ICH).

ICH –GS score was calculated and considered surgical evacuation for patients with herniation syndrome. Those patients who were not willing for surgical evacuation were considered control

Materials and methods

We performed prospective study of all patients presented to Govt. medical college Kottayam from August 2018 to January 2020. A total 420 patients of spontaneous ICH were presented to hospital were studied. Initial presentations, Presenting GCS score, pathology, size of hematoma and ICH –GS score was calculated and considered surgical evacuation for patients with herniation syndrome. Those patients who were not willing for surgical evacuation were considered control group. Patients were evaluated after2 weeks and after6 months.

Results

A total of 420 cases who presented in our department with spontaneous ICH were evaluated. 42 patients were presented with SAH secondary to intracranial arterial aneurysms, 35 patients were found to have lobar hematoma and 11 cases were tumor bleeds. Our study considered 322 cases of spontaneous capsulo-ganglionic bleeds, evaluated preoperatively with ICH-GS score which constitutes, age, size and location of bleed, intra-ventricular hemorrhage and GCS score.

According to the signs of herniation syndrome, 128 patients were selected for surgery and other patients were managed conservatively with anti-oedema measures. 22 patients were not willing for surgery. According to ICH scores 322 patients were divided into 3 groups and their scores were recorded after 2 weeks, 1 month and 6 months. All nonsurgical patients were monitored for 1 month and surgically selected patients were monitored for 6 months. In the surgical group 22 patients who were not willing for surgery were considered control group.

Out of 128 patients in the surgical group, 30 day mortality was 34 nos, in which 20 patients were post operative (group A). Six months mortality was 51 nos including 32 of group A and 19 of group B(not willing for surgery group). Fifteen out of sixty five patients in group A in ICH-GS score 7-10, died in 6 months (p value=0.024 – significant). That was about 23 percentage compared to 53 percent in group B, while 96 percentage of patients of ICH-GS >10 in both group A and B died in 6 months (Table 1).

		ICH- GS <7	ICH-GS 7-10	ICH-GS >10	total
No.of cases		193	98	31	322
Surgical group	Surgery done (A)	**15**	**65**	**18**	**106**
	Not Willing for surgery(B)	2	13	9	22
	Total	17	78	27	128
30 day mortality	A	**0**	**8**	**12**	**20**
	B	1	5	7	14
	Total	1	14	19	34
30 day mortality (non surgical group C)		0	4	4	8
6 months mortality	A	**0**	**15/65 (23.07%)**	**17**	**32/106 (30.18%)**
	B	2	**7/13 (53.8%)**	9	19/22 (86.36%)
	Total	2	22	26	51/128(39.8%)

Table 1. Mortality of patients.

Mortality of post operative patients with supra-tentorial bleeding in 6 months was about 26 percentage while 50 percent for infra-tentorial bleeding. 56 percent of patients with hematoma size more than 70 ml died in 6 months. 61 percent of patients with IVH died in 6 months (Table2).

		No of cases	30 day mortality	6 month mortality
Location	Supra-tentorial	90	14	24 (26.6%)
	Infratentorial	16	6	8 (50%)
Size of Hematoma	40-70 ml	81	10	18(22.22%)
	>70 ml	25	10	14 (56%)
Age	<45 years	16	2	3 (18.75%)
	45-65	54	10	15 (27.78%)
	>65	36	8	14 (38.8%)
IVH	Absent	80	2	8 (10%)
	Present	36	18	22 (61.12%)
GCS score	3-8	46	14	18 (39.13%)
	9-12	48	6	10 (20.83%)
	>12	12	0	2 (16.67%)

Table 2. Distribution of patients in Group A

Discussion

Management of spontaneous intra-cerebral hematoma include identification of pathology, timely intervention, ICU care, post operative care, physiotherapy and treatment of the pathology. This study deals with the mortality of patients of spontaneous ICH after 30 days and 6 months and how timely surgical intervention can reduce the mortality. In this study patients were grouped into three according ICH-GS score, <7, 7-10 and >10. In first group score less than seven, it was found that surgical intervention can reduce the mortality significantly. In group 7-10 score, surgical intervention can reduce the mortality from 53.8% to 23.07% which is statistically significant, while mortality in group >10, ICH-GS score is more than 95% irrespective of intervention.

On comparing the mortality with recent studies where only medical management were done, 30 days mortality ranges from 23.3% to 50% and after surgery mortality ranges from 31 to 40%. In our study, 30 days mortality is 10.55%, (mortality among surgical group is 18.86%), while 6 months mortality among surgical group is 39.8% (Table.3).

Conclusion

Spontaneous ICH can be managed both medically and surgical. Patients with herniation syndrome needed surgical intervention. In patients with low ICH-GS score, surgical intervention can reduce the mortality significantly in 30 days and 6 months. Patients with high ICH-GS score have more than 90% mortality whether managed surgically or medically.

Table. 3 Comparison of recent studies

Studies	Cases	Surgery group	30 days mortality	6 months mortality
THIS STUDY	**322**	**106**	**34 (10.55%)**	**51 (surgical group)**
BHATIA ET AL	212	NIL	70 (33.9%)	
DANIEL ET AL	153	NIL	53 (34.6%)	
SAFTALI ET AL	342	NIL	80 (23.3%)	
FOGELHOLM ET AL	411	NIL	50%	
DOUGLAS ET AL	70	NIL	28 (40%)	40 (57%)
FLOHARTY ET AL	MEDICAL-MX- 1041		34%	59%
	Surgical mx-	183	31%	53%

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Dr.K.Ajax John Receiving Best paper Award from Dr.Gnanadas .K . President KNS

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Dr. K M NAZMUL ISLAM JOY is an eminent Neurologist & Internist in Dhaka .He has done lot of research work in the field of Covid 19 in Neurology practice . Our editorial team is grateful to him for sharing his article and knowledge in this present scenario of the pandemic which would be useful for most our clinicians in these difficult situations.

Do's and Don'ts in Neurological Practice: COVID 19 Pandemic Perspective

Introduction:

The current novel corona virus pandemic, after starting as an outbreak in Wuhan, China in December 2019, has shattered the present world in 2020 with its high infectivity, diverse and mysterious clinical presentations and alarming mortality among the severe cases. The pandemic is likely to be sustained until an effective vaccine develops or herd immunity appears, both of which will cost the world an indefinite time period.

The pandemic situation is worse in south-east Asia, particularly in India & Bangladesh, where dense population burden, lack of motivation for wearing masks and sanitization among the population and lack of adequate testing facilities for proper isolation is causing increased number of infections day by day. There is also lack of adequate and ideal personal protective devices, particularly masks of medical grade (like N95) for the caregivers in these regions, like the physicians, nurses and paramedics. The virus is extremely contagious, and persons in close contact with suspected/confirmed cases within 6 feet/2 meter for 15 minutes are supposed to get infected, whatever the viral load may be. Neurologist practitioners are at high risk of getting infections as many of neurological examinations e.g. cranial nerve examinations including fundoscopy as well as investigative (e.g. lumber puncture, Nerve conduction studies & Electromyography) & therapeutic (e.g.botox injection) procedures need close contact with the patients. Neurologists also need to attend referral patients, and Intensive Care Units have a very high viral load because of aerosol generating, rendering them to a very high risk of getting infection.

However, life cannot remain for a total shutdown for an indefinite period; people have to engage in activities as physicians need to provide their services to them who are sick. Considering given scenarios, there has to be some basic principles for the general practitioners as well as neurologists for patient management .The problem is that there is not enough guidelines in this regard and some principles or guidelines vary from country to country. For example, coded telemedicine services are

enlisted for practicing physicians in the United States[1] which may not be applicable for regions like India or Bangladesh. In this article, we would try to focus briefly on the technical aspects which neurologist practitioners should be aware of.

Telemedicine in Neurology:

Telemedicine can be defined as medical care provided to a patient by a physician or other health care provider at a different location.[2,3] The application of telemedicine had been slowly growing for decades before the advent of COVID-19, mainly in primary or mental health care.[2]By the use of electronic communications technologies like video whatsapp, messenger, zoom,e-mails etc, it by-passed geographical, physical, and biological barriers to health care access. No universal consensus yet exists that high-quality care for neurology patients can be achieved via virtual methods, but the evidence base is increasing. For instance, virtual stroke care is now well established.[2,3,4] Legal and regulatory barriers impeding Telestroke adoption or delivery are being identified. It has been established that Telestroke is providing an effective solution for small and under resourced hospitals, even in India. With the onset of COVID-19, telestroke services are being further expanded to reduce or avoid physical visits. Outcomes after intravenous tissue plasminogen activator treatment via telemedicine are similar to those achieved with in-person evaluations.[3] Apart from stroke, teleneurology has been successfully deployed in the initial management of acute minor head trauma or in follow-up of post-operative cases of head injury, headache medicine and movement disorders.[3,4]

Some centers, e.g., New York University Langone Health (NYULH), use the Epic electronic medical record (EMR) ecosystem with interconnected apps on desktop and laptop computers, including Haiku on the iPhone and Canto on the iPad.[2] Mobile devices are used by physicians for the virtual visits. Their front- and rear-facing device cameras are technologically superior to the single built-in cameras on desktop or laptop computers. The screen size on an iPhone is adequate for many clinical encounters, and the iPad screen is sufficient for detailed neurologic examinations.

There is no currently validated examination for general teleneurology.[2]Some studies found the telemedicine examination to be more sensitive in detecting abnormalities than a face-to-face examination for all components; & was more specific for all but one clinical sign (plantar response).[2,3] However, most studies included trained presenters at the time of virtual examination. Mobile devices are preferred because their cameras are superior to desktop and laptop cameras, and the patient can also use the front-facing or rear-facing cameras of the device with moving it as required.[2]

Teleneurology consultations may take place at patient's own residence, with help of relatives, and some may take place at a medical/chamber facility with help of trained assistants.

Much of the cranial nerve (CN) examination can be performed reliably via telemedicine through a combination of direct maneuvers and application-based supplements. Ophthalmoscopy of the optic discs is currently a major limitation of the virtual examination and cannot be performed in a typical virtual visit. Table I

Preferred clinical examination	Scope	Comment
General appearance of the patient	On inspection via the video/audio connection.	
Vital signs	measurement of any 3 of : sitting or standing blood pressure, supine blood pressure, pulse rate and regularity, respiration, temperature, height, and weight	Cannot be measured and recorded without ancillary staff
Attention span/concentration, Orientation to person, place, and time, Language (e.g., naming objects, repeating phrases/ spontaneous speech)	Via the video/audio connection.	
Recent and remote memory	Via the video/audio connection.	MOCA appropriate for telemedicine (mocatest.org/remote-moca-testing/) or MMSE
2nd cranial nerve (e.g., visual acuity, visual fields) & Ophthalmoscopic examination	Confrontation visual fields performed by the examiner on the computer screen for gross assessment. Ask the patient to report any blurring or absence of the examiner's facial features.	When appropriate: *Eye Handbook App* for acuity and color vision. The download is free. Fundoscopy not possible unless there is a tele-ophthalmoscope
3rd, 4th, and 6th cranial nerves (e.g., pupils and eye movements)	Open and close eyes to look for pupillary reaction to ambient light Ask the patient to look left, right, up, and down with head still. Sustained upgaze for 30–60 seconds.	Patient or home assistant can use penlight/flashlight, may be able to do swinging flashlight test.
7th cranial nerve (e.g., facial symmetry and strength)	Inspection via the video/audio connection for facial symmetry, smiling, and nasolabial fold flattening	Specific movements: raising eyebrows, closing eyes can easily be performed.
8th cranial nerve	Via the video/audio connection—can they hear the examiner?	
9th cranial nerve (e.g., spontaneous or reflex palate movement)	Holding the camera very close to opened mouth to inspect palatal movement.	Probably best with assistant and penlight/flashlight
11th cranial nerve (e.g., shoulder shrug strength)	Inspect movement via the video/audio connection.	

Preferred clinical examination	Scope	Comment
12th cranial nerve (e.g., tongue protrusion)	Movements like ability to protrude the tongue, move the tongue side by side is easily assessed.	Atrophy/ fasciculations can be visible
Muscle strength in upper and lower extremities	Inspection via the video/audio connection for gross movements, posturing, movements against gravity, symmetry of movement, and drift of the arms or legs. Exercises—push-ups, stand up from the floor, stand up from the chair without using arms, going up and down steps, walk on heels, and walk on toes.	
Specific upper limb testing:	Ability to raise the arms above one's head Sustained abduction of the arms for up to 120 seconds. Ability to spread rubber band using one's fingers (tests finger abduction). Holding a paper clip or pen between the fingers (tests finger adduction)..	Ability to blanch the knuckles when making a fist Handwriting can easily be tested.
Specific lower limb testing:	Repeated arising from a chair without using arms, up to 20 reps. Ability to arise from a squat or from the floor Ability to stand on &hop on each leg. Ability to spread & flex toes.	
Muscle tone in upper and lower extremities	Shaking hands/arms while standing to observe for flail arms. Bradykinesia, tremors, and involuntary movements via the video/audio connection.	Notation of any atrophy or abnormal movements (e.g., fasciculation and tardive dyskinesia) via the video/audio connection is possible

Preferred clinical examination	Scope	Comment
Coordination	Finger-nose/Heel-knee-shin. Alternately slap the thigh with the front and back of the hand. Cycling with both legs while lying flat.	Inspection via the video/audio connection
Deep tendon reflexes in upper and lower extremities with notation of pathologic reflexes (e.g., Babinski)	Deep tendon reflexes may be self-tested or by the assistant by using finger tapping, kitchen spatula, and selfie stick handle.	However, cannot be properly assessed without an experienced assistance
Sensation (e.g., by touch, pin, vibration, and proprioception)	May be assessed with an assistance with putting cotton/sharp(tooth pick) or cold objects(metallic surface)	
Gait and station	Primary gait/Heel and toe walking/ Tandem gait.	Truncal stability assessed by observing the patient's balance when sitting with arms and legs held out in front of them (seated Romberg).

*MoCA=Montreal Cognitive Assessment, MMSE=Mini Mental Scale Examination

Limitations of teleneurology:

Not all patients are suitable for teleneurology. Patients having visual/auditory/cognitive impairment cannot take advantage of it unless assisted by an expert attendant. The technical procedure requires motivation, cooperation and mutual awareness, with specific directions to patients and family members. Operating the mobile device & software effectively, on either side, needs some sort of training. There is no version of forms for previous medications, allergies, past medical, family, and social history, and review of systems. for virtual visits. Some crucial examinations like ophthalmoscopic examination & plantar response are not possible unless high-tech equipments or skilled assistant is not avaible, respectively, which if very true for countries like Bangladesh or India. The most important challenge of implementing teleneurology in these regions is that socioeconomic determinants of health adversely affects access to access to appropriate devices or network connections and may not have a location for proper neurological examination. Interventions like botox injection, nerve block etc is clearly out of the scope of teleneurology.

Table I: Brief overview of neurological examinations which can be adopted virtually[2]

*__Neurological face to face practice where tele-neurology is not possible:__

Neurologists have to think and perform diversely during a patient consultation. Even though neurological examinations comprises of a number of elements, during this pandemic, very sharp clinical observation with minimum examinations is able to aid a diagnosis, as it was always taught in medical school. Minimum exposure time with high clinical skill along with a brief, technical counseling to the patient will help to achieve patient satisfaction also. Even non-touch methods like only taking history may help. There are no clean cut guidelines on neurological practice In general during time of COVID 19 pandemic. However, some general as well as neurological guidance can be made.

__Awareness of neurological symptoms of COVID-19 & decisions for referral:__

In addition to the conventional features of fever, cough and/or respiratory distress, a practicing neurologist should know the neurological manifestations of COVID 19 [5] and the time to referral to higher center.

Neurological Symptoms (in suspected or confirmed COVID 19 case /in any patient in a hotspot area)	**Neurologist's action**
Mild Headaches or Headaches suggestive of primary headache(Migraine/TTH), Anosmia, Myalgia, Fatigue	Symptomatic/specific (e.g. for migraine) outpatient management along with advice for follow up in case of severe neurological or respiratory symptoms.
Stroke	>Patients with other co-morbidities must be hospitalized, whether the patient is stable or not. >Cases where interventions like thrombolysis/ embolectomy/ clipping or coiling of aneurysms are needed should be referred to structured COVID dedicated stroke units. >Young patients with on risk factors and stable to go home need follow up.
Severe Neurological features: Headaches suggestive of secondary headache/raised ICP Features of meningitis(Headache with meningism/Neck rigidity) Recent Drowsiness/disorientation/ /Convulsion (Features suggestive of encephalitis) Cloudiness of consciousness, limb weakness(ADEM) Weakness of limbs, respiratory difficulty or bladder retention (GBS or Myelopathy)	Urgent referral to a facility with neurology department as well as a structured COVID unit, with central oxygen supply and ICU, as these cases need meticulous treatment and might need advanced support.
Other neurological features like cerebellar syndrome ,cranial nerve palsies etc	Neurologist's/GP's judgment of individual patient condition and if needed, referral or further consultation.

General rules of face to face neurology practice:

Personal protection: There must not be any compromization in personal protection at any cost. A consultant neurologist/GP must have the followings before starting his patient consultation.

1. Mask:

Should be worn throughout the patient service and must not be removed unless soiled or straps are torn. In case a mask must be doffed it better not to be in front of patient or assistants and a second clean mask is needed to be worn. There should be available/extra masks in physician's closet for any emergency situations.

A N95 mask of recognized category, like 3M's 8210 model/1860 model/Macrite or a KN95 or FFP2 or equivalent etc is the best mask for personal protection.[4,6] Although recommended for whom who will directly examine a confirmed covid-19 case, However, considering the facts that it is not possible to test every patient in south-east Asia setting, and even if patient is free of COVID like symptoms, he/she may be an asymptomatic /pre-symptomatic carrier (besides the patient attendant issues) and you need to attend the patient very closely at any time of his/her stay in the chamber, it is better not to use a low grade or only cloth masks for a physician.

Physician's own choice of masks also matters, as does the infection rate in the area where the chamber is located.However.in any situations, a doctor must wear a N95 mask or equivalent, or at least 02 surgical masks. It should be kept in mind that any amount of protection is not hilarious against COVOD-19.

2. Eye protector:

A face shield or eye goggles must be worn in chamber time and during examining the patient, better if disposable. These must be discarded at the end of chamber. In case of reusable ones, it must be kept in a covered place, which can be reused after 5-7 days later, or in case of early reuse, it should be cleaned with sanitizer or detergents. The external surface of shield is contaminated, as it for face masks, and must not be touched before sanitization. Re-placement/extra face shields should be kept in practice place.

3. Sanitization:

Proper sanitization materials, at least 70% isopropyl alcohol in form of hexiscrub or hexisol, must be available at chamber and used after examining every patient or touching fomites used by patient like reports, CT scan plate etc.

4. Surgical gloves:

Can be worn, if the physician feels to touch the patient, and must be disposed of before examining the next patient. Double gloves can also be worn to avoid rupture and cross infection.

5.Sanitization of instruments:

Sanitization of neurological instrument like tendon hammers, tuning forks, monofilaments, stethoscope etc need to be disinfected after each use, with sanitizations containing 70% isopropyl alcohol.

6. Coverall/Gown/Dress:

Hair covers & shoe covers sterile standard gown along with long-sleeved water-resistant gown can be worn during patient consultation. Inner dress should be comfortable (e.g. made of cotton) and easily washable.[6]

Chamber Infrastructure: There must be some form of adaptive changes in the infrastructure of chamber, to minimize infection.

Outside chamber:

1. Sitting arrangement of patients must be changed in the waiting room; chairs should be marked with at least 3 feet distance between. Not more than one attendant per patient should be allowed. A timed schedule for patient arrival should be made so that no overcrowding occurs in the waiting place.[6]

2. Instructions for wearing a face mask in a proper way for all patients and their attendants should be displayed on visible areas. No patient should be entertained without face mask during waiting period and inside doctor's chamber. Only the physician will decide to ask for its removal in need of examination of face or other purpose.

3. Adequate supply of sanitizers and disinfectant spray, for door handles, chairs, switchboards etc.

4. Chamber attendant should also be properly equipped with at least face mask, shield, gloves and aprons.

Inside chamber:

Sitting arrangement should be reshuffled for allowing patient to sit for at least 6 feet distance, with an extra seat for the attendant. Examination bed should be isolated.

Creating a glass or plastic barrier between patient sitting area and the doctor sitting area may be appropriate, provided the surfaces of the barrier be cleaned with disinfectants daily at end of patient consultation.[6]

Ideally the room should be properly ventilated. Should an AC run, the direction of flow should not be from patient to physician. Central AC system without proper filtration is discouraged.

Table 2:

Categorization of neurological conditions by level of importance of close examination in face to face visits:

Level of importance	Description	Possible action	Examples (not exhaustive)	Clinical assumptions
Must be closely attended	The clinical presentation is as such that for proper evaluation, diagnosis or referral, close examination believed to be necessary, whether acute or chronic.	**Appropriate precautions/personal protection is a must**. Risks and benefits/impact of the examination should be balanced.	>Sudden visual loss /visual obscuration/ Headaches indicating raised ICP requiring fundus examination provided ocular causes excluded and the physician cannot readily have a neuroimaging. >Examination of pupils/eyeball movements /palates in cases of differentiating NMJ disorders from cranial nerve palsies >Speech difficulties ,requiring bulbar muscles and tongue examination muscle and tongue examination & jaw jerks >Weakness of limbs causing difficulty in differentiating between peripheral nerves or spinal cord or muscle diseases, requiring minimal examination for deep reflexes, tones and plantar responses.	>Retinal artery occlusion/CRVO//Papillodema of ICSOL or IIH >MG or 3rd nerve palsy or polyneuritis cranialis. *Ice on eyes test* may be appropriate for ocular MG. >Bulbar or pseudobulbar palsy >GBS or Myelopathy or myopathy

Triage for neurology practitioner:

Depending on the neurological presentation, a physician can categorize patients to whom he or she needs to pay more attention. I structured a table which may be helpful in this regard(Table 2).Many of these are derived from practical experiences of the author and other senior or junior colleagues.

Conclusion:

COVID-19 is a rapidly evolving pandemic; we see each day information is being rectified. However it is the fact that life would never be normal as before. So practitioners engaged in either GP or neurology specialty should try to adapt to the teleneurology modalities, particularly the elderly physicians with co-morbidities. Prescription software technologies & billing methods need to be improvised in teleneurology sector with appropriate government legislature as well as corporate assistance. Face to face practice or direct interventions is not discouraged, but with a strict maintenance of personal safety measures and keeping in mind the basic neurological tips mentioned here, which surely would be modified in coming future.

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Life time Achievement Awards

Dr. K. A Salam

Former Professor of Neurology ,Eminent Faculty and Past President of NSI Kerala Chapter receiving Life time achievement award from Dr. P.Sreekumar Patron KNS,with Dr.Mohanlal D., Dr.A.S.Girija, Dr.Madhusudhanan M.and Dr.Jayakrishnan at NSI Kerala meeting Perinthalmanna—Essence2020

Dr. Madhusudanan M.

Former Professor of Neurology and Beloved Teacher of the State receiving Life time achievement award from Dr. P.Sreekumar, Patron KNS, with Dr.Mohanlal D., Dr.A.S.Girija, Dr.K.A.Salam and Dr.Jayakrishnan at NSI Kerala meeting Perinthalmanna—Essence2020

Essence 2020 - NSI Annual meeting Perinthalmanna

Inauguration By Prof.(Dr.) Mohanan Kunnummal,Vice-Chancellor,KUHS

Past President Handing over the Charge to the new President

Prof . A.S.Girija

Dr.Gnanadas .K.

Release of First Edition of Newsletter and e-journal ``BODHI"

BODHI , Volume I , Issue 2 ,November 2020

Essence 2020 - NSI Annual meeting Perinthalmanna

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From the editorial key board .....!

The newsletter and e journal BODHI' second edition is result of the enormous effort from the Senior and Junior members of the Society as the unprecedented pandemic calamity of COVID –9 has taken its toll on the Neuroscientists from all the specialties working in our state .We had to make timely changes in the format of academic as well as official meetings which could be made possible with launch of our own DIGITAL PLATFORM with the support of all Senior Members and Patrons of the Society. This was Officially inaugurated by Dr .P. Sreekumar, Patron of the Kairaly Neurosciences Society in the first Virtual Official meeting invited by Dr.Gnanadas, President of the Society in July 2020.

The DIGITAL PLATFORM is encrypted with most of the live webinar and video transmission options and could be used by the members of the Society all over the state for Academic meetings , teaching programs, Telemedicine, Conferences , PG case discussions etc. We request all the members to make use of it and so that learning process for the younger generations will propagate even in this difficult times .

We also request the members to contribute your scientific manuscripts and interesting cases to the e-journal so that everyone gets know about your dedicated work which is a major uplift for the science in our state. We are looking forward for the enrichment of scientific materials our future editions with your whole hearted co operation.

We thank all the Specialists who contributed for this edition and all the team members who extended their support in releasing this edition. We are grateful to Dr. K M NAZMUL ISLAM JOY from Dhaka for sending us his work on guidelines for Neurology practice in Covid –19 which is of great importance to withstand the test of these difficult times. We also thank Dr.Ramnarayan for sending us his unique work with late Dr Simon Hercules .

"Coming together is a beginning, staying together is progress, and working together is success." – Henry Ford

With Best Regards from Bodhi editorial team

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