NEUROLOGY & NEUROSURGERY NEED SPECIFICATION DOCUMENTS

These unmet clinical needs were identified following an 8-week clinical immersion that was conducted by a team in Neurology and Neurosurgery. The clinical immersion was conducted in high volume tertiary care centers in South India. We observed what's done and how it affects the provider, the patient, and the system. This was followed by a peripheral immersion to numerous primary, secondary and tertiary health care centers across India. The clinical needs found in the tertiary care hospital were validated in other large centers and new primary and secondary specific unmet needs were added on. At the end of 2 months of clinical immersion, the team had over 30 detailed observations with significant negative outcomes and 40 unmet clinical needs. These needs were then filtered using objective parameters, detailed below:

THE FILTERING PROCESS: 3 STAGES OF FILTERING

Level 1: The level 1 filter eliminated those needs which are redundant, pharmaceutical related or process related.

Level 2: This level of filtering focused on the severity of the clinical condition (in the perception of observers and clinicians) as well as the epidemiology of the disease and the frequency of the negative outcome. This data was then validated by a comprehensive literature review of incidence and prevalence data. A scoring system of 1 – 3 – 5 was used through the process.

Epidemiology

- Frequency of problem as per clinician (number of cases per month)
  - < 5 patients per month = 1
  - 6-12 patients per month = 3
  - >13 patients per month = 5
- Frequency of problem as per observers (number of cases seen per month during the clinical immersion)
  - < 2 patients per month = 1
  - 2-5 patients per month = 3
  - >5 patients per month = 5

Criticality

- Short lasting, reversible: Not resulting in death, disability, hospitalization, or socioeconomic stress = 1
- Resulting in death, hospitalization >3 days, disability/ handicap (> 6 months), large financial burden to the patient/family = 5
- Needs in between 1 and 5 = 3

Observed Epidemiology and criticality score: 3 (Frequency of clinician) + Frequency of observer + 3 (Criticality score)

Target patient population in a given year: We used data wherever available for India. However, in many cases due to the dearth of validated health statistics, certain assumptions had to be made using a combination of data from India and global epidemiological data.

- <100,000 patients/year = 1
- 100,000 – 500,000 patients/year = 3
- >500,000 patients/year = 5

Secondary research-based epidemiology and criticality score: Target patient population * Criticality score

Filter 2 score: Subjective epidemiology and criticality score + secondary research-based epidemiology and criticality score (out of 10)

Level 3: The third level of filtering evaluated the technical complexity of the solutions available, the regulatory landscape and the buyer environment.

Number of predicates: This was made based on the solutions which currently exist as per guidelines and those being used in the Indian clinical setting. Both at prevailing practice as well as gold standards were considered.

- High number of predicates i.e. >5 = 1
- Medium number of predicates i.e. 1 to 5 = 3
- No predicates = 5

Technical complexity of predicates: This filter considered the technology behind the solution as well as the expertise needed to implement it in current clinical practice. A medium complexity solution is rated the highest, followed by low complexity and lastly by a highly complex solution.

- High =1
- Medium =5
- Low =3

Regulatory and clinical trial complexity: This filter was based on the regulatory hurdles and clinical trials one would have to conduct for a particular solution. It was a judgment call based on the current predicates in the system and the classification of devices as per the Global Harmonisation Task Force classification (Class A - Low Risk, Class B - Low to Moderate Risk, Class C - Moderate to High Risk, Class D - High Risk)

- High (Class D) =1
- Medium (Class C) =3
- Low (Class A & B) =5

Buyer environment: This filter was based on the eventual buyer of a particular medical solution. This, in turn, depended on which level in the healthcare system the particular

condition was treated. The peripheral immersion helped to understand, more thoroughly, the referral system in India which defined this filter.

- High (Tertiary Care Centre) = 5
- Medium (Secondary Centre) = 3
- Low (Individual/Primary centre) = 1

Filter 3 score = Number of predicates score + Technical complexity of predicates score + Regulatory and clinical trial complexity score + Buyer environment score

Final Score = Filter 2 score + (Filter 3)/4 (Out of 15)

NEED SPECIFICATION DOCUMENTS NEUROLOGY & NEUROSURGERY

1. VENTILATOR-ASSOCIATED PNEUMONIA

BACKGROUND

Ventilator Associated Pneumonia (VAP) is a lung infection caused due to a patient being ventilated on a mechanical ventilator [1]. Typically this occurs in cases of long-term ventilated patients. Long-term can be as short as 3-4 days or even after 3-4 weeks of being on a ventilator. There are several causes of VAP including poor immunity of the patient, oral secretions trickling down the trachea, poor patient hygiene etc. VAP is one of the most common Hospital Acquired Infections (HAI) and accounts for more than half of all Hospital-acquired Pneumonia infections. [2] This increases the patient's stay at the hospital and delays recovery and rehabilitation. Several patients die annually from VAP in India which is commonly caused by multidrug-resistant organisms.

OBSERVATION

Presentation

A 41-year-old male came into the Neuro-emergency ward of a tertiary care hospital in an ambulance after being involved in a road traffic accident. There was no external bleeding.

History

The patient had no history of Hypertension, Diabetes, or any other co-morbidity.

Treatment

An emergency CT scan revealed an epidural hematoma that was causing compression on the brain. There was a slight mid-line shift. The patient's Glasgow Coma Scale (GCS) score was 11/15. The patient was taken for a Decompression surgery via Craniotomy. The hematoma was successfully evacuated. Due to poor respiratory effort post-surgery, the patient had to be intubated and was put on mechanical ventilation. The neurosurgeon said that the poor respiratory effort was due to pulmonary edema secondary to Trauma Brain Injury (TBI). The patient was put on prophylactic antibiotics due to an External Ventricular Drain (EVD) catheter that was inserted to lower increased intra-cranial pressure.

On Post-op day 7 (POD_7), the patient contracted Pneumonia (VAP). Sputum cultures were sent to labs for culture sensitivity tests. Labs came positive for Pseudomonas infection. The patient had to be put on stronger antibiotics because the infection had developed antibiotic resistance.

The infection took 13 days to completely clear out. The patient was extremely weak and could barely move his hands or legs. The patient was put on Deep Vein Thrombosis (DVT) Prophylaxis.

On POD_{14}, the patient developed Urinary Tract Infection (UTI). Cultures came positive for E. coli infection. The patient was put on further antibiotics (Fosfomycin) for treating the UTI.

Until POD_{23}, the patient was on the ventilator. Patient's postoperative (post-op) GCS was 9/15 (with minor fluctuations) until POD_{21}. GCS was checked when the patient was vacated from sedation daily to assess for extubation. The patient's respiratory effort was gradually improving and the brain inflammation was slowing reducing.

Patient's GCS started declining on POD_{22} morning to 8/15. Although the patient could breathe by himself, the secondary brain damage had led to hypoxia in certain parts of the brain. MRI revealed hypoxia in several parts of the brain. The patient's GCS dropped to 6/15 by evening. Doctors had given up hope of reviving the patient due to severe secondary injury. The patient died on POD_{22} night.

THE PROBLEM

Hospital-acquired pneumonia (HAP) is pneumonia that develops 48 hours or longer after admission to a hospital. HAP is the second most common nosocomial infection. HAP increases a patient's hospital stay by approximately 7-9 days and can increase hospital costs by an average of $40,000 per patient. [1,2]

Ventilator associated pneumonia is one such HAP. The diagnostic triad for VAP consists of the following clinical criteria: Pulmonary infection, bacteriologic evidence of pulmonary infection and radiologic suggestion of pulmonary infection. When the combination of radiologic infiltrates and 2 clinical criteria are observed, the sensitivity of diagnosing VAP is 69% and the specificity is 75%. VAP is commonly associated with multidrug-resistant pathogens. The risk factors for developing a resistant infection include: Hospitalization admission of greater than 5 days, hospital admission more than 2 days in the preceding 90 days, antibiotic use in the previous 90 days, residence in a nursing home or extended-care facility, home infusion therapy and wound care, long-term dialysis within 30 days and immunocompromised. Ventilator Associated Pneumonia (VAP) is currently prevented by following the ventilator bundle [3]. The bundle involves elevation of the patient's head to 30-45 degrees, assessment of readiness to extubate, peptic ulcer prophylaxis and deep vein thrombosis (DVT) prophylaxis. In addition, the dedicated ICU nurse periodically suctions the subglottic and oropharynx region and also does an oral lavage with chlorhexidine (disinfectant). Studies have shown that the ventilator bundle has reduced the incidence of VAP cases. However, in spite of this prophylactic management, a large number of patient's still contract VAP due to an ineffective protocol.

NEED STATEMENT

A way to reduce the incidence of Ventilator Associated Pneumonia (VAP) in long-term intubated patients at THCs to prevent prolonged stay at hospital or death

FILTERING PROCESS

Final Score = 29

Rank = 1

MARKET POTENTIAL

India has an incidence of around 30 VAP infections per 1000 ventilator days. More than 3 lakh people die annually from VAP and related complications. [3,4] VAP occurs in 9-27% of all mechanically ventilated patients. Ventilator-associated pneumonia (VAP) is a complication in as many as 28% of patients who receive mechanical ventilation. The incidence of VAP increases with the duration of mechanical ventilation. Estimated rates are 3% per day for the first 5 days, 2% per day for days 6-10, and 1% per day after day 10. The crude mortality rate for VAP is 27-76%. [1]

COMPETITIVE LANDSCAPE

VAP is prevented by minimizing the patient's exposition to bacteria. This involves maintaining a sterile environment around the patient in the Intensive Care Unit (ICU). Adherence to the VAP bundle protocol, along with regular oral and larynx disinfection using chlorhexidine. [1]

AVAILABLE OPTIONS

- **Ventilator bundle –** This comprises of 4 main actions that are taken to prevent Pneumonia.
    1. *Elevation of the head to 30-45 degrees* – The top half of the body is inclined to this angle so that the patient's trachea and oesophagus are inclined and hence help the secretions flowing down the pharynx to enter the oesophagus, instead of entering the trachea.
    2. *Sedation vacation and assessment of readiness to extubate* – The patient is vacated from sedation and respiratory effort is checked. If the respiratory effort is adequate, the patient is extubated.
    3. *Peptic Ulcer Prophylaxis* – The patient is normally put on proton pump inhibitors to reduce the acid production in the stomach. The acid reflux can lead to pulmonary aspiration and can cause Pneumonia.
    4. *Deep Vein Thrombosis (DVT) Prophylaxis* – Long-term intubated patients are at high risk for developing DVT. This can lead to a Pulmonary Embolism (PE).

However, DVT prophylaxis has not shown to reduce the incidence of VAP. It still remains part of the bundle.

- **Subglottic, Supraglottic and oropharynx secretion suctioning** – The nurse periodically suctions the secretions from the subglottic and oro-pharynx space that reduces the risk of any secretions moving into the trachea. Oral washes with Chlorhexidine which is a disinfecting agent helps in reducing the chance of lung infection. [5]

IDEAL SOLUTION

The solution should reduce the incidence of Ventilator Associated Pneumonia (VAP) compared to the current standard of care.

NEED CRITERIA

MUST HAVES

- Reduce the incidence of VAP compared to current standard of care statistics (Less than 10 VAP infections per 1000 ventilator days)
- Must not increase the cost to patient in an ICU by more than 20%

NICE TO HAVE

- Completely prevent Ventilator-Associated Pneumonia

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2. INTRACRANIAL PRESSURE MONITORING

BACKGROUND

Elevated intracranial pressure (ICP) is seen in various conditions such as head trauma, hydrocephalus, intracranial hemorrhage, subarachnoid hemorrhage, intracranial tumors, hepatic encephalopathy, and cerebral edema. Elevated ICP can lead to death or devastating neurological damage either by reducing the perfusion of essential oxygen and nutrients to the brain also known as reduced cerebral perfusion pressure (CPP) or by compressing of vital structures in the brain and brainstem. Prompt recognition is crucial in order to intervene appropriately. Intractable high ICP is also the most common "terminal event" leading to death in neurosurgical patients. The association between the severity of intracranial hypertension and poor outcome after severe head injury is well recognized. Outcomes tend to be good in patients with normal ICP, whereas those with elevated ICP are much more likely to have an unfavorable outcome. Elevated ICP carries a mortality rate of around 20%. The rapid recognition of elevated ICP is therefore of obvious and paramount importance so that it can be monitored and so that therapies directed at lowering ICP can be initiated. A raised ICP is measurable both clinically and quantitatively. Continuous ICP monitoring is important both for assessing the efficacy of therapeutic measures and for evaluating the evolution of brain injury. [1]

OBSERVATION

Presentation

A 42-year-old male came into the Neuro-emergency ward of a government tertiary care hospital in an ambulance after being involved in a road traffic accident. There was no external bleeding.

History

The patient had a history of hypertension for 1 year and was on medication for the same.

Treatment

The patient's GCS was 10/15. A CT scan was immediately done which revealed a left-sided subdural hematoma with midline shift. The patient was rushed to the Operation Theatre (OT) and the hematoma was evacuated. There was severe Edema and the brain had swollen. An External Ventricular Drain (EVD) catheter was placed in the left ventricle and the patient was monitored in the post-op ICU. The EVD was connected to an ICP monitor which revealed that the patient's ICP was 45 mmHg. The patient was put on Mannitol to manage the edema.

The disposable ventricular catheter needed to be changed every 7 days due to the high risk of infection. Nearly 20% of all patients with ICP monitors contract infection that leads to ventriculitis or even encephalitis. The catheter with ICP monitoring capability costs INR.50,000. Due to the high cost, it takes the government time to procure large numbers of these catheters.

The hospital did not have a replacement catheter and had to remove the EVD after 7 days. The patient's GCS was still 11/15 and there was still cerebral edema as indicated by the CT scan

done every 2-3 days. The patient's ICP was now being estimated based on GCS only. On Post-op day 9 (POD9), the patient's GCS fell to 9/15. A CT scan revealed a fluid-filled compartment in the subdural space. Another emergency surgery had to be done to evacuate the fluid and find the source of the fluid. The surgeon said the fluid was a collection of Cerebrospinal fluid (CSF) and edematous fluid.

POD2 the patient's GCS was 11/15 and holding. The doctor said that GCS and vital parameters are the only ways of estimating ICP.

THE PROBLEM

External Ventricular Drains (EVD) are expensive, costing up to INR.50,000. In addition, this drain has to be changed by a neurosurgeon every three to seven days due to the high risk of infection [2]. Doctors have tried re-sterilizing the used EVD, however, there is a high risk of contracting Prions Disease. CT/MRI scans give an accurate indication of significantly increased ICP; however, this is not a reliable way to continuously monitor ICP. It is also expensive for many patients to afford a CT scan on a daily basis. An EVD is placed blindly using standard anatomical landmarks in the brain. There is no real-time feedback as to whether the catheter tip is inside the lateral ventricle. There can also be bleeding along the EVD insertion tract which can lead to subdural or epidural bleeds which may even require surgical management.

Lumbar Punctures (LP) also come with risks of brain herniation and infection if the needle is left in situ for several days. Patient mobility becomes almost impossible.

NEED STATEMENT

An affordable, safer and continuous way to monitor ICP in patients at THCs to prevent the high risk of infection, permanent brain damage and seizures.

FILTERING PROCESS

Final Score = 27.5

Rank = 2

MARKET POTENTIAL

More than 3 lakh cases of continuous monitoring of ICP are required annually in India [2]

COMPETITIVE LANDSCAPE

External Ventricular Drain (EVD) Catheter is the standard of care for monitoring ICP continuously. The Glasgow Coma Scale (GCS) is used as an estimate when EVD catheters are not available. However, GCS is lowered several hours or even days after the ICP has increased. Many times, it is too late to act after GCS values drop.

AVAILABLE OPTIONS FOR MONITORING INTRACRANIAL PRESSURE

- **Clinical examination:** This is the most important tool for diagnosing potential elevation of ICP and monitoring its progression is the clinical neurological examination. Despite medical advances, clinical observation has not lost its importance in the ongoing monitoring of a patient's condition. The patient should be evaluated for the following: Headache, nausea, and vomiting, Degree of alertness or consciousness (Glasgow coma score), language comprehension, repetition, fluency, articulation; Pupillary reactivity; Extraocular movements and visual; Vital signs (Cushing triad: respiratory depression, hypertension, bradycardia); Gag or cough reflex and response to noxious stimuli
- **Funduscopic examination:** This also remains the criterion standard in the evaluation of increased ICP. careful attention to the optic nerve can prove useful for evaluating the ICP. The optic nerve is surrounded by subarachnoid space and experiences pressure changes in the same way that the intracranial compartment does.
- **Ventriculostomy –** This is a surgery wherein a Ventricular Catheter is placed in the lateral ventricle via the Kocher's point. An EVD is connected to the Catheter to drain out excess CSF to control ICP within normal limits. An EVD needs to be changed every 7-10 days due to high risk of infection.
- **Lumbar Puncture (LP) / Spinal Tap** – A long needle is inserted typically in the L2-L3 space of the spine and a tap is established with the subarachnoid space. A manometer is then connected to the same to measure ICP. This is useful when only small quantities of Cerebrospinal Fluid (CSF) need to be drained.
- **Glasgow Coma Scale** – Although this is not the recommended standard of care, this is often used on government hospitals when EVD Catheters are not available. A score is given based on the patient's eye movement, verbal response, and motor response. A score of less than 9/15 requires emergency medical attention. [3]
- **MRI/CT Scan –** Imaging can help to determine whether the there is increased ICP or not. This can be done by observing the arteries, ventricles and brain tissue on T1 weighted images.

IDEAL SOLUTION STATEMENT

The solution should be able to accurately and continuously monitor ICP, without any significant risk factors such as infection.

NEED CRITERIA

MUST HAVES

- Accurately and continuously measure ICP

- No risk of infection or any equivalent morbidity
- Should not increase cost of management for an ICT patient by more than 20% in an Intensive care setting

NICE TO HAVEs

- Non-invasive and accurate measurement of ICP
- Should also allow for management through cerebrospinal fluid drainage

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3. PARKINSON'S DISEASE

BACKGROUND

Parkinson's Disease (PD) is a neurodegenerative disease which affects the motor system over a period of several years and even decades. Parkinson disease (PD) is one of the most common neurologic disorders, affecting approximately 1% of individuals older than 60 years and causing progressive disability that can be slowed but not halted, by treatment. The 2 major neuropathologic findings in Parkinson disease are a loss of pigmented dopaminergic neurons of the substantia nigra pars compacta and the presence of Lewy bodies and Lewy neurites. Common symptoms include shaking (tremors), rigidity, slowness of movement and difficulty walking. India has an annual incidence rate of at least 1 lakh new cases [1].

The incidence of Parkinson disease has been estimated to be 4.5-21 cases per 100,000 population per year, and estimates of prevalence range from 18 to 328 cases per 100,000 population, with most studies yielding a prevalence of approximately 120 cases per 100,000 population. The incidence and prevalence of Parkinson disease increase with age. Onset in persons younger than 40 years is relatively uncommon. Parkinson disease is about 1.5 times more common in men than in women. [1]

Before the introduction of levodopa, Parkinson disease caused severe disability or death in 25% of patients within 5 years of onset, 65% within 10 years, and 89% within 15 years. The mortality rate from Parkinson disease was 3 times that of the general population matched for age, sex, and racial origin. With the introduction of levodopa, the mortality rate dropped approximately 50%, and longevity was extended by many years. This is thought to be due to the symptomatic effects of levodopa, as no clear evidence suggests that levodopa stems from the progressive nature of the disease. [1]

OBSERVATION

Presentation

A 71-year-old female came into the Neurology OPD of a tertiary care hospital with complaints of progressive muscle rigidity, especially in the joints over the past 1-2 years, along with mild postural instability. The family also informed doctors that the patient had occasional episodes of mild shaking of the left wrist when kept at rest.

History

The patient had no history of stroke, seizures or brain tumor. The patient had no history of hypertension, Diabetes or high cholesterol. The patient had no other comorbidities.

Treatment

The neurologist asked for a brain and spine MRI which was completely normal. Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP) and autoimmune panel - all came normal. A complete neurological examination was done and doctors noticed significant slowness in

movement and walking compared to speech. The patient's Montreal Cognitive Assessment (MoCA) score was 23/30, which was indicative of Mild Cognitive Impairment (MCI). Doctors were unsure whether this was an early onset case of Alzheimer's Disease (AD), Parkinson's Disease (PD) or Progressive Supranuclear Palsy (PSP). Based on the occasional tremors in the left wrist, the doctors assumed a diagnosis of Parkinson's Disease and started the patient on Levodopa and Carbidopa. The patient was advised to go for frequent walks and solve puzzles and play games to improve physical movement as well as cognitive function.

The patient was regularly followed up every month for 1 year. During this period, the family reported periodic improvements and deterioration of the patient. The patient was often nauseous, vomited every alternate day and felt dizzy when getting up from the bed. The doctor informed the patient's family that these were the side-effects of the medication. Patient's family requested the doctor not to increase the dose of L-dopa due to the side-effects. By then, the doctors had a confirmed diagnosis of PD due to the increase in wrist shaking at rest. Doctor's offered surgery as an option wherein a Deep Brain Stimulation (DBS) device would be implanted into the brain of the patient and would reduce the patient's Parkinson's symptoms. However, the device and surgery were priced at INR.15 lakhs, which the patient's family could not afford.

After 3 years, the doctors are still managing the patient on medication (L-dopa and Dopamine agonists). The tremors have increased severely. The patient can barely stand for one minute due to severe pain caused by the rigidity in the waist and knee joints. The patient's family had to be with her at all times to prevent a fall.

THE PROBLEM

There is no affordable solution that stops or significantly reduces the rate of degeneration caused by PD. DBS is the only solution that manages the symptoms by sending electrical pulses to certain regions in the brain. However, this solution is expensive and unaffordable to most Indians. Most medications do not have significant improvement of the symptoms. L-dopa dose needs to be increased periodically to maintain the same the effect.

A common problem with all long-term medication, including those for treatment of Parkinson's Disease, is that the strength of the dosage needs to be progressively increased with time for the same or increased efficiency. Most common cause for this is the development of the body's immunity to the same. The side effects of these medications also increase progressively with increase in dosage. Beyond a certain point, the benefits of the medication are less than the degree of side effects and hence, the dosage has to be saturated. Heavy medication almost invariably puts a heavy load on the kidneys and liver. Liver Function Tests (LFTs) and Renal Function Tests (RFTs) need to be periodically done to ensure that medication is not taking its toll on the patient. The side effects of prolonged Levo-dopa treatment are well known in Parkinson's Disease. Dyskinesia is the key side effect seen in PD patients on L-dopa medication. Many times, the treatment needs to be tapered down to prevent the severe movement disabilities introduced in PD patients.

NEED STATEMENT

An affordable (as compared to surgery) and effective (no side-effects due to medications) way to manage Parkinson's disease in patients at THCs to prevent severe morbidity.

FILTERING PROCESS

Final score = 19.5

Rank = 3

MARKET POTENTIAL

More than 1 lakh annual incidence rate of Parkinson 's disease in India. Nearly 1 million people in India are currently living with PD. [2]

COMPETITIVE LANDSCAPE

Treatment of Parkinson's Disease primarily lies around management with medication and palliative care. Although L-dopa has it's side-effects and is not entirely effective, this is the most affordable and widely accepted solution. There is no cure for any neurodegenerative disease as of today. Most of the solutions are aim at delaying the progression of the disease. L-dopa has episodic positive and negative effects on the patients. [3]

AVAILABLE OPTIONS FOR PARKINSON'S DISEASE

- **Levodopa and Dopamine agonists –** Most of PD symptoms are due to deficiency of Dopamine in the Basal Ganglia region of the brain. Hence, medication is given to increase dopamine levels in the brain. Due to the blood-brain barrier, a significant dose of L-dopa needs to be taken to have a mild effect in the brain. High levels of L-dopa in the blood causes all the side-effects associated with L-dopa medication.
- **Other Medication** – COMT Inhibitors, MAO-B Inhibitors and anticholinergics do not have strong evidence supporting their use in the treatment of motor symptoms accompanying PD.
- **Deep Brain Stimulation (DBS)** – This is an implant is placed in the brain via surgery and stimulates the Thalamus, Globus Pallidus or subthalamic nucleus depending on the motor symptoms observed. The device is then calibrated wirelessly from outside. The device costs INR.15 lakhs including the surgery. Subsequent follow-ups and re-calibrations add to the cost.
- **Functional Stereotactic Lesioning Surgery –** This is a minimally invasive brain surgery which involves creating lesions using a 3D geometrical pin-pointing technology.

The exact location in the brain is lesioned using temperature. The patient has to be retrained after the surgery to regain fine control over the muscles.

IDEAL SOLUTION STATEMENT

The solution should be able to either cure, stop or significantly slow down the neuro-degeneration process, while also maintaining reasonable Quality of Life. The solution should be affordable to the rural people of India.

NEED CRITERIA

MUST HAVES

- Significantly and progressively slow down or stop rate of neuro-degeneration (Patient should have a life expectancy of at least 10 years post diagnosis, assuming no other comorbidities)
- No side-effects causing significant morbidity or discomfort (as currently available drugs do)
- It should be comparable with minimal access surgery costs in other surgical specialities

NICE TO HAVE

- Completely reverse and stop any degeneration until treatment (Patient should have a normal life expectancy, assuming no other comorbidities)
- Patient should completely regain full and normal control over motor system

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4. PRESSURE ULCERS IN STROKE PATIENTS

BACKGROUND

Stroke is a severe disabling disease affecting millions in India annually. Stroke is becoming an important cause of premature death and disability in low-income and middle-income countries like India, largely driven by demographic changes and enhanced by the increasing prevalence of the key modifiable risk factors. As a result developing countries are exposed to a double burden of both communicable and non-communicable diseases. The poor are increasingly affected by stroke, because of both the changing population exposures to risk factors and, most tragically, not being able to afford the high cost of stroke care. Majority of stroke survivors continue to live with disabilities, and the costs of on-going rehabilitation and long-term-care are largely undertaken by family members, which impoverish their families. Elderly patients affected with stroke takes significantly longer to recover from the paralysis. In the process, these patients develop several secondary disabilities such as Osteopenia/Osteoporosis, Deep Vein Thrombosis (DVT), Clinical Depression, Edema etc. [1]

Stroke is one of the leading causes of death and disability in India. The estimated adjusted prevalence rate of stroke range, 84-262/100,000 in rural and 334-424/100,000 in urban areas. The incidence rate is 119-145/100,000 based on the recent population-based studies. There is also a wide variation in case fatality rates with the highest being 42% in Kolkata. Stroke units are predominantly available in urban areas that too in private hospitals. Intravenous and intra-arterial thrombolysis is commonly used in India. [2]

Although the terms decubitus ulcer, pressure sore, and pressure ulcer have often been used interchangeably, the National Pressure Ulcer Advisory Panel (NPUAP) currently considers pressure injury the best term to use, given that open ulceration does not always occur. A pressure ulcer is localized damage to the skin and underlying soft tissue, usually over a bony prominence or related to a medical or other device. It can present as intact skin or an open ulcer and may be painful. It occurs as a result of intense or prolonged pressure or pressure in combination with shear.

OBSERVATION

Presentation

An 81-year-old female was brought into the emergency ward of a Tertiary care hospital with left-sided hemiparesis and complete paralysis of the right hand. The patient had developed aphasia with a deviation of the mouth to the right. She also had severe difficulty in breathing. The family reported that they noticed the gradual difficulty in speech 4 days before.

History

The patient was a known case of Diabetes Mellitus Type 2 for 15 years. She was on medication for the same and her Blood Sugar was under control.

Treatment

Immediate MRI-scan in the emergency ward revealed large areas of ischemic hypoxia in the left and right frontal-parietal regions. The patient was put on Levipil and rushed to the ICU, where she was put on a ventilator. She was in ICU for two weeks, during which she developed Ventilator Associated Pneumonia (VAP). She was put on antibiotics for the same and recovered from the same. Her respiratory effort improved and was extubated. She was still aphasic.

During her recovery over 6-7 weeks in the regular ward, the patient has several emotional outbreaks. Psychiatry was consulted and she was put on antidepressants. The patient developed bedsores and was treated with wound care and medication for the same.

The patient was put on calcium and Vit. D3 to prevent osteopenia. The patient was also put on DVT prophylaxis (antithrombotic drugs and stockings). Patient's physiotherapy sessions were very poor as she showed little to no effort. A full-time trained attender was employed to take care of the patient.

The nursing bundle was followed with turning every few hours but the patient developed a pressure ulcer at the coccyx. The patient was kept on her side and the mattress was exchanged with a water bed. The ulcer grew larger despite the daily dressing and debridement and the patient could not afford the vaccum or hydrogel dressings.

The daily dressing continued for the 10 weeks of admission in the hospital with minimal improvement in the ulcer bed after which the patient was discharged and advised to get daily dressing done at the local clinic

THE PROBLEM

Due to the aging process, recovery from stroke in the elderly is significantly slower. Doctors rely on the family, physiotherapists and speech therapists to help the patient in creating the enthusiasm and will to perform rehabilitation tasks. Patients frequently go into clinical depression. Doctors frequently say that an incentivized and quantifiable rehabilitation system which gives feedback to the patient about his/her progress can go a long way to speed up the rehabilitation process. Majority of stroke survivors continue to live with disabilities, and the costs of on-going rehabilitation and long-term-care are largely undertaken by family members, which impoverish their families.

Pressure is exerted on the skin, soft tissue, muscle, and bone by the weight of an individual against a surface beneath. These pressures often exceed capillary filling pressure (~32 mm Hg). In patients with normal sensitivity, mobility, and mental faculty, pressure injuries do not occur. Feedback, conscious and unconscious, from the areas of compression leads them to change their body position, and these changes shift the pressure before any irreversible tissue damage develops. It is often seen in bedridden patients can is very difficult to manage. It can result in complications like malignant transformation, autonomic dysreflexia, osteomyelitis and sepsis.

NEED STATEMENT

An effective way to prevent the onset of pressure ulcers in patients bedridden for >72 hours in any setting (hospital or home)

FILTERING PROCESS

Final score = 18

Rank = 4

MARKET POTENTIAL

More than 1 million cases of pressure ulcer annually [2]

COMPETITIVE LANDSCAPE

The current standard of care is primarily supportive and palliative care. A nursing bundle is used in all bedridden patients and includes the following

- Support surface Use an appropriate pressure redistribution support surface and reassess as the patient's needs change
- Skin inspection Check entire skin regularly, with particular emphasis over bony prominences, and document in patient's healthcare records
- Keep moving Implement a turn/reposition schedule and optimise/ encourage independent movement. Refer to occupational therapist/ physiotherapist when appropriate
- Incontinence and moisture Ensure appropriate management of incontinence (urinary and faecal), perspiration or exudate in conjunction with a structured skin care programme to maintain skin integrity
- Nutrition and hydration Encourage individuals to eat and drink regularly and assist patients when necessary. Refer to dietitian when appropriate

IDEAL SOLUTION

The solution needs to be able to prevent clinical depression, bed sores, DVT, UTI, Pneumonia while also recovering the patient faster than current methods. A faster rehabilitation solution will invariably reduce the risk for all of the above-mentioned co-morbidities.

NEED CRITERIA

MUST HAVES:

- Must be at least as effective as the nursing bundle and air/water beds

- Must cover at least 3 of 5 key risk factors: uneven distribution of pressure, humidity, raised temperature, contact with medical devices, sheering force
- Must not cause irritation or increase pressure on skin thereby leading to pressure ulcers
- Must not only detect high risk locations but also adjust the pressure accordingly
- Must be used to amenable to long term use
- Must be easy to use even by caregivers at home
- Must given an alert in case there is a risk for pressure ulcer development

NICE TO HAVE:

- Useful in prevention as well as management of pressure ulcer
- Re-useable

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5. DELAYED CEREBRAL ISCHEMIA (DCI)

BACKGROUND

The term subarachnoid hemorrhage (SAH) refers to extravasation of blood into the subarachnoid space between the pial and arachnoid membranes which surround the brain. Subarachnoid Hemorrhage (SAH) is a medical emergency in which there is bleeding into the subarachnoid space of the brain, often causing what is described as the most painful thumping headache in a person's life. t occurs in various clinical contexts, the most common being head trauma. However, the familiar use of the term SAH refers to nontraumatic (or spontaneous) hemorrhage, which usually occurs in the setting of a ruptured cerebral aneurysm or arteriovenous malformation (AVM). This is commonly followed by increased intracranial pressure leading to the patient losing consciousness gradually. One of the most common causes of Subarachnoid hemorrhage is a ruptured intracerebral artery aneurysm. An aneurysm is a blood-filled bulge in the lining of the arterial wall. Atherosclerosis, infection (most commonly Syphilis) and congenital etiology are the most common causes of an aneurysm. This occurs most commonly due to the weakening tone of the arterial wall. This requires emergency surgery to decompress the cranium and prevent further bleeding. Delayed Cerebral Vasospasm is a common complication post-surgery in which cerebral arteries constrict leading to severe cerebral ischemia and often death [1]. Vasospasm occurs in nearly 50% of all SAH patients post decompression. Delayed Cerebral Ischemia typically starts on Post-op Day 2-3 (POD_3) and peaks at POD_5 - POD_{14}. [1]

The frequency of ruptured and unruptured aneurysms has been estimated at 1-9% in different autopsy series, with a prevalence of unruptured aneurysms of 0.3-5%. Retrospective arteriographic studies show a prevalence of less than 1% with the limitation that some cases did not receive adequate evaluation and thus some aneurysms may have been missed. Annual incidence increases with age and probably is underestimated because death is attributed to other reasons that are not confirmed by autopsies. The annual incidence of aneurysmal SAH in the United States is 6-16 cases per 100,000 population, with approximately 30,000 episodes occurring each year. Unlike other subcategories of stroke, the incidence of SAH has not decreased over time. However, since 1970, population-based survival rates have improved. [1]

OBSERVATION

Presentation

A 55-year-old male came into the emergency department of a government tertiary care hospital with a sudden onset splitting headache. The patient was unconscious when he was brought into the emergency ward. Family informed that the patient had an unbearable headache, after which the patient started losing consciousness.

History

Neither the patient nor the family has had a history of headaches or blackouts. The patient had a history of Diabetes Mellitus Type 2 for 10 years and was on medication (insulin) for the same. According to the family, the patient's blood sugar was under control.

Treatment

CT scan showed a massive aneurysm rupture in the basilar artery leading to a subarachnoid hemorrhage. The patient was immediately rushed for surgery to stop the source of the bleed and decompress the cranium. An aneurysm located at the basilar artery was coiled to prevent blood from entering the cavity. The patient was given Nimodipine (Calcium channel blocker) prophylactically in view of possible cerebral vasospasm. Post-op patient's Glasgow Coma Scale (GCS) was 12/15. The patient was started slowly on triple-H (Hypertension, Hypervolemia, and Hemodilution) therapy prophylactically along with continuation on Nimodipine. Patient's GCS was 10-11/15 early on Post-op day 3 (POD_3).

The doctor said that there was no way to continuously monitor the cerebral arteries for vasospasm, so as to act only when vasospasm is observed. Prophylactically starting triple-H therapy along with Nimodipine without vasospasm has high risks of re-bleeding, hyponatremia, renal medullary washout, and cerebral edema. Hence, most of the time the doctors have to start triple-H therapy with Nimodipine only after vasospasm has begun. This requires continuous intensive monitoring.

Due to dropping GCS, contrast Magnetic Resonance Angiography (MRA) was done on POD_3 and revealed bilateral Anterior and Middle Carotid Artery (ACA and MCA) vasospasm. Triple-H therapy was made more aggressive at the risk of re-bleeding. However, by POD_4, patient's GCS had reduced to 9/15. MRI done on POD_4 evening revealed cerebral ischemia. The patient died on POD_5 early morning before any surgical intervention could be planned.

THE PROBLEM

There is no way to continuously and accurately monitor major to medium intracranial artery perfusion. This leaves doctors delivering medication prophylactically, not knowing whether they are required and in what dose. Most of the prophylactic medication such as Calcium channel blockers and triple-H therapy have various side-effects. Nimodipine and triple-H therapy are often contraindicated in severely hypertensive patients. There have been cases of cerebral re-bleeds, Gastro-intestinal (GI) bleeds, cerebral edema and renal medullary washout due to calcium channel blockers and triple-H therapy. Most of the times, it is too late to plan for surgical intervention such as angioplasty due to severe ischemia leading to irreversible neurological deficits. More importantly, triple-H and Calcium Channel blockers have not clearly shown to be effective in preventing secondary ischemia, especially in patients with moderate to severe Subarachnoid Hemorrhage. Management of severe SAH cases is almost entirely prophylactic since most of such patient progress to DCI. [1]

NEED STATEMENT

An affordable and continuous way (compared to MRI, CT [monitoring] Calcium channel blockers and triple-H therapy [preventing vasospasm]) to manage (monitor) cerebral arterial perfusion in patients with subarachnoid hemorrhage at THCs to prevent Delayed Cerebral Vasospasm and Delayed Ischemic Neurological Deficits (DIND)

FILTERING PROCESS

Final Score = 18

Rank = 5

MARKET POTENTIAL

More than 2 lakh annual cases of subarachnoid hemorrhage (SAH) in India. Nearly 50% of these cases develop severe Cerebral Vasospasm leading to Delayed Ischemic Neurological Deficits (DIND). [2,3,4,5] Hence, we have more than 1 lakh annual cases of SAH progressing to DCI of varying severity.

COMPETITIVE LANDSCAPE

Diagnosis of vasospasm is done via Contrast MR Angiography. Calcium channel blockers and Triple-H therapy are primarily used to prevent vasospasm. A meta-analysis by Cochrane reviews has shown that there is no clear benefit in using these therapies to prevent DIND. Due to lack of continuous monitoring, angioplasty cannot be planned in advance.

- **Calcium Channel Blocker –** Nimodipine is primarily used to induce vasodilation. This drug is given for up to 3 weeks post-op since vasospasms can occur even 2 weeks after the hemorrhage. Nimodipine is a short-acting drug and hence needs to administered 2-3 times a day under constant monitoring for re-bleeds [6]. Since these drugs induce systemic vasodilation, their dose needs to be closely monitored, especially in patients on antithrombotics and vasodilators.
- **Triple-H Therapy** – Hypertension, Hypervolemia, and Hemodilution are induced through intravenous (IV) fluids to counter vasospasm. However, the hypertension is systemic and not local. There is a high risk of renal medullary washout. Hence, IV fluids need to need to be administered after closely monitoring kidney function [7].
- **Angioplasty surgery** – If the vasospasm is detected early and is localized to a small length of the artery, angioplasty can be planned to allow for normal perfusion. However, most of the times vasospasm is caught late due to lack of continuous monitoring.

- **Glasgow Coma Scale (GCS)** - This scale is the most commonly used method in the assessment of consciousness. This indirectly gives an indication of Intracranial Pressure (ICP) and cerebral perfusion. However, it is poor in distinguishing the precise cause of decreased consciousness.
- **Intraventricular Catheter for ICP monitoring** - This uses a pressure transducer inserted via a catheter in the lateral ventricles or third ventricle. When ICP increases, there is
- **Transcranial Doppler Ultrasound** - This monitoring method uses ultrasound waves to monitor cerebral perfusion in arteries close to the cranium. However, deep-seated major arteries cannot be monitored effectively and accurately using this method. This method is, however, extremely useful due to its non-invasive nature and hence can be done 2-3 times a day, if required.
- **CT / MRI scan** - Scans are normally asked for, only when symptoms start showing in the patient. Decreased GCS and increased ICP are common reasons for getting a CT/MRI scan. These scans are expensive, require the patient to be mobilized and also require the patient to be still during scans. This causes MRI scans to be a last resort. MRI scans are used only when other easily usable monitoring options are either unavailable, contraindicated or do not give the required diagnostic information.

IDEAL SOLUTION STATEMENT

The solution should help in the monitoring of cerebral perfusion pressure.

NEED CRITERIA

MUST HAVES

- Should allow for the accurate, continuous monitoring of cerebral perfusion pressure
- Should be able to assess perfusion in different parts of the cerebrum, midbrain and brain stem
- Cost to patient should not exceed 20% of the current ICU costs
- Should alert the physician in case of a drop in pressure

NICE TO HAVE

- Should be non-invasive
- Should be easy to use (any trained ICU nurse should be able to use the solution)

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6. CHILDHOOD EPILEPSY

BACKGROUND

Epilepsy in children is a common condition affecting millions in India. Childhood epilepsy is defined as epileptic seizures of any type in children up to the age of 18 years of age. There are various causes of epilepsy in children such as preterm birth, uneventful and prolonged labor, maternal exposure to toxins, genetic and congenital causes, etc. More than 3 million children under the age of 18 years are epileptic. Several of these epileptic conditions do not have clear and definite treatment options. Most of the children respond to Antiepileptic Drugs (AEDs). However, around 5-10% of childhood epilepsy patients are refractory to AEDs. This leads to the underlying disease to worsen, leading to cognitive decline, increase in frequency and severity of seizures and severe developmental retardation. Early and accurate diagnosis of the cause of epilepsy can significantly help doctors is pursuing the right course of treatment. [1]

OBSERVATION

A 12-year-old boy comes to the Neurology OPD of a tertiary hospital with a history of increased frequency of seizures over the past 72 hours. Seizures involve clenching of the teeth, tonic posturing of the head and drooling of saliva from the mouth.

History

The boy is the youngest of four siblings. He was born of a non-consanguineous marriage. He was born at Full-term via normal vaginal delivery**.** Antenatal period of pregnancy was uneventful with infrequent checkups. The boy had bluish discoloration at birth. The baby was kept in intensive care for close monitoring. The baby had seizures from the 2nd day of birth. He was discharged 10 days after a period of no seizures. He had generalized yellowish discoloration which worsened over the next 15 days. His stools were white in color. The boy had decreased feeding and reduced cry. The patient was taken to a government tertiary care hospital where he was treated and followed up for one year. Seizure frequency was under control.

The patient had delayed and abnormal development over the course of the first 2 years. At 18 months of age, the patient was brought to the hospital with several episodes of seizures over the previous 48 hours. Seizure Semiology: Up rolling of the eyes, frothing at the mouth and generalized tonic-clonic movements. The patient was a given a dose of steroids over 3 months which stopped the seizures temporarily.

At 4 years of age, the patient presented to the hospital with left hemiparesis, deviation of angle of the mouth. The patient recovered with treatment gradually over 4-5 months. The patient had decreased use of upper and lower limbs.

Patient has been on valproate, Topiramate, and Clobazam since birth. Patient has responded well to corticosteroids previously when the frequency of seizures had increased to many episodes per day.

The frequency of seizures has increased over the past 10 years. Initially (first 2 years), the boy had 1 seizure per month. For the next 5-6 years, the boy had 4 seizures per month. Currently,

the patient has several episodes of seizures per day. Patient experiences different types of seizures such as generalized tonic-clonic seizures, generalized atonic seizures and atypical absence seizures.

Treatment

On examination: Patient is conscious and oriented in person. Not co-operative in speech. Patient has right eye ptosis, left lower limb hypertonia along with brisk Deep Tendon Reflexes (DTR) in the lower left limb with ankle jerks. The patient had Plantar extensor withdrawal with ataxic gait. The patient had multifocal myoclonic jerks in limbs.

MRI revealed mild multi-focal cortical atrophy involving insular and peri-insular regions. There was a presence of ipsilateral ventricular enlargement. Presence of T2 hyperintensity or atrophy of the head of caudate nucleus. Unilateral cortical and caudate atrophy progressively worsening (when compared to previous MRI scans).

Current short-term plan of treatment: 2-3 months of corticosteroids

Long-term plan: Functional MRI imaging along with brain biopsy. Multiple subpial transection/corpus callostomy / Hemispherectomy may need to be done.

THE PROBLEM

A large number of children with Rasmussen's Encephalitis (RE), Lennox-Gastaut Syndrome (LGS) and some form of Hypoxic-Ischemic Encephalitis (HIE) sequelae are refractory to antiepileptic drugs (AEDs). Most commonly used AEDs include valproate, lamotrigine, topiramate, benzodiazepines and rufinamide. Patients with RE progressively worsen with a severe reduction in cognitive function. MRI generally indicates hemispheric atrophy with inflammation. There is no way to definitive way to cure RE, LGS or HIE sequelae. Early management with AEDs, corticosteroids and immuno-active agents does not always significantly improve neurological deficits.

Neurologists' precise diagnosis and cause of childhood epilepsy is based on MRI scans, blood workup, and neurological examination. Various conclusions can be drawn from these, leading to a diagnosis which does not point to a concrete etiology.

NEED STATEMENT

A long-term and definitive (compared to antiepileptic drugs and corticosteroids) way to treat epilepsy (Hypoxic Ischemic Encephalitis sequelae, Lennox Gastaut Syndrome and Rasmussen Encephalitis) in children at Tertiary care centers to prevent hemispheric atrophy, severe cognitive decline, and severe generalized morbidity

FILTERING PROCESS

Final score = 18

Rank = 6

MARKET POTENTIAL

More than 3 million children in India are epileptic. More than 3 lakhs of these children are refractory to medication and often require drastic surgical intervention, introducing severe morbidity. [2,3]

COMPETITIVE LANDSCAPE

Various types of Anti-Epileptic Drugs (AEDs) are the standard of care for children with epilepsy. Several devices are available which alert the family and relatives of the child during a seizure so that the child can be attended to immediately. In patients who are refractory to standard AEDs, stronger anticonvulsant medications are used. These medicines have moderate to severe temporary side-effects. In cases where medication has no effect at all, surgery is the last resort. Hippocampal resection, Corpus Callostomy and cerebral hemispherectomy are most common surgeries resorted to.

- **Anti-Epileptic Drugs (AED) –** Various AEDs are given depending on the type and severity of the epileptic seizure to prevent further episodes. Several patients have seizures in spite of being on AEDs. A fraction of them go into status epilepticus and need to be intubated after sedating them.
- **Steroids –** Steroids are often used as a first-line treatment in children with seizures accompanied by encephalitis. This is done to suppress the inflammatory response. Tacrolimus is also occasionally used although there is no evidence supporting its use in childhood epileptic encephalitis.
- **Intravenous Immunoglobulin (IVIG)** - Some studies have shown IVIG to reduce the frequency and severity of seizures. However, a Cochrane review classified these studies as highly biased and still claims that there is no clear unbiased Randomised Control Trial (RCT) evidence for IVIG in the treatment of refractory epilepsy [5].
- **Sedation and Intubation –** In case the patients progress to Status Epilepticus (SE), the patients are sedated and intubated till the electrical activity in the brain stops (as seen on EEG).
- **Neurostimulation** – In patients where surgery is contraindicated, neurostimulation may be used. Vagus nerve stimulation, anterior thalamic stimulation and closed-loop responsive stimulation are the common neurostimulation procedures. [4]
- **Surgery –** Brain resection surgery is a last resort when drugs do not show any improvement. Surgery introduces permanent disability due to removal or disconnection of parts of the brain. Hemispherectomy or corpus callostomy are the two common surgery options used in cases of refractory epileptic seizures.

IDEAL SOLUTION STATEMENT

The solution should be able to prevent seizures. In addition, the solution should prevent brain atrophy and cognitive decline. The child's overall development should be unhampered and progress at a normal rate.

NEED CRITERIA

MUST HAVES

- Prevent seizures
- Prevent cognitive decline
- Allow normal growth and development

NICE TO HAVEs

- Completely reverse any damage done to the brain

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7. TRAUMATIC BRAIN INJURY

BACKGROUND

Traumatic brain injury (TBI) is a nondegenerative, noncongenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness. TBI is a major cause of death and severe morbidity in all countries. Fall from heights and Road Traffic Accidents (RTA) are the leading causes of TBI. [1]

The definition of TBI has not been consistent and tends to vary according to specialties and circumstances. Often, the term brain injury is used synonymously with head injury, which may not be associated with neurologic deficits. The definition also has been problematic with variations in inclusion criteria. Inconsistency in the definition and classification of traumatic brain injury (TBI), along with discrepancies in data collection, has made the epidemiology of TBI difficult to describe accurately. Problems with TBI data collection include the fact many patients with mild TBI may not present to the hospital, and the ones who do present may be discharged at the emergency department (ED) without adequate documentation. Severe TBI with associated death at the scene of the accident or during transport to a hospital also may not be accounted for completely in data collection for TBI epidemiologic studies.

Differences in diagnostic tools and admission criteria also may affect the above-defined severity classifications. In the past, the use of roentgenograms to help diagnose skull fractures after head injury did not show much of any concurrent intracranial lesions. These lesions were difficult to diagnose until the advent of CT scanning, which is now the diagnostic imaging of choice in TBI cases. [1]

OBSERVATION

53 year-old-male was brought into the emergency ward of a tertiary government hospital after encountering a Road Traffic Accident. Patient's GCS was 8 ($E_2V_2M_4$) with no visible open injury or bleeding. Patient's eyes were barely reactive to light.

History

The patient had a history of Diabetes Mellitus Type 2 for the past 6 years and was on medication for the same. No history of smoking or alcohol consumption. No other comorbidities.

Treatment

Immediate CT-scan in the emergency ward revealed a midline shift with a severe subdural hemorrhage. The patient was started on Mannitol and immediately rushed to emergency OT for Decompressive Craniectomy.

Patient's GCS post-op was 11/15. Post-op CRASH prognostic tool for TBI patients indicated that there was more than a 40% chance that the patient would not survive post op due to petechial bleeds, midline shift, and low GCS.

Patient's GCS progressively dropped from 11 to 7 over the next 4 days. There was increased Intracranial Pressure (ICP) as measured from the intraventricular catheter. There was severe brain inflammation. The doctor said GCS dropped due to secondary hypoxic insult caused by extensive inflammation. All the doctor could do was administer mannitol and nimodipine to manage edema and prevent vasospasm respectively. The patient died Post-op day 6 due to severe brain hypoxia.

THE PROBLEM

There is very little that doctors can do in the acute stage (days immediately post-op) after Decompressive Craniectomy in moderate to severe TBI patients. The brain inflammation needs to subside by itself as part of the healing process. ICP monitoring is an expensive affair and the intraventricular catheter needs to be changed frequently due to high risk of infection.

NEED STATEMENT

A way to improve post decompressive craniectomy prognosis in patients with moderate to severe TBI (intracranial hematomas) at THCs to prevent persistent vegetative state or death

FILTERING PROCESS

Final score = 17

Rank = 7

MARKET POTENTIAL

More than 1 million cases of TBI annually

More than 40% of all severe TBI cases do not survive past 14 weeks of admission to a Tertiary Care Hospital (THC). [2, 3]

COMPETITIVE LANDSCAPE

TBI cases are always an emergency and early management can lead to better outcomes. As soon as the patient is taken to a THC, a GCS score is evaluated and CT-scan is done to look for intracranial hematomas. If there is significant intracranial bleeding, the patient is rushed for Decompression surgery. The patient is administered Mannitol to help reduce brain edema and hence ICP. Intraventricular catheters are expensive (around INR.50,000 per use and are

frequently not available in government THCs) and need to be changed frequently (every 2 weeks) due to the risk of infection. Most of the damage to the brain is done due to secondary injury. This happens due to hypoxia induced by increased ICP. Eventually, doctors have to wait and hope that the brain can withstand the ICP and heal in time. CT-scans may be required every 2 days to monitor for any uncontrolled bleeding or fluid collection, which may be expensive or impossible (unavailability of CT-scan) in a government THC.

- **CT/MRI scan** – This used to gauge the extent of damage done by the TBI. It helps the doctors plan a further course of action in terms of surgery and medication.
- **Decompressive Craniectomy (DC)–** This is done to evacuate the hematoma, reduce ICP and allow the brain to expand due to inflammation. The cranium flap is stored in a sterile container at very low temperatures. DC does not always lower ICP to normal levels. If the inflammation and edema are too much, then ICP still remains elevated to a level that poses a risk for hypoxia. Continuous ICP monitoring is essential. [4]
- **ICP Monitoring –** Continuous ICP monitoring along with periodic GCS scores is the state of the art when it comes to the quick assessment of the patient's progress in the case of TBI. Higher ICP normally correlates with low GCS and vice-versa. When ICP is above threshold values, doctors initiate an action such as a CT/MRI for details as to why the ICP is high. This may be followed by a change of medication or surgery, as the case may be.
- **Medication –** Mannitol is the standard drug used to treat cerebral edema and inflammation. Anti-Epileptic Drugs (AEDs) are used when patients have seizures or are at high risk for the same.
- **Palliative Care –** If the patient has managed to regain a GCS above 13, the patient is discharged, even though the patient is far from regaining full control of his/her body. Physiotherapy and speech therapy is an active part of helping the patient taking back control of his/her daily activities. A healthy diet and certain drugs can help this process
- **Therapeutic hypothermia**: Also termed target temperature management (TTM), is the cornerstone of neuroprotective strategy. Dating to the pioneer works of Fay, nearly 75 years of basic and clinical evidence support its therapeutic value. Although hypothermia decreases the metabolic rate to restore the supply and demand of O_2, it has other tissue-specific effects, such as decreasing excitotoxicity, limiting inflammation, preventing ATP depletion, reducing free radical production and also intracellular calcium overload to avoid apoptosis. [4]
- **Functional Electrical Stimulation:** Promising results are coming from the use of functional electrical stimulation (FES). FES uses a computer and electrodes to give small bursts of electricity to paralyzed muscles. This is done to generate muscle contractions. Currently, FES is being used to try and restore function that was lost after a spinal cord injury. By stimulating paralyzed muscles, patients are experiencing improvement in hand movements, bladder and bowel control, and even potential for respiration without the use of a ventilator. Researchers are trying to advance this current system, which is showing promising results not only in spinal cord injuries, but for stroke patients as well. [5]
- **Brain-Computer Interfaces (BCI):** Brain-computer interfaces bypass nerve cells that have been damaged from a spinal cord injury. These devices, when implanted, may restore control of voluntary muscle movement to paralyzed muscles. Because patients

with spinal cord injuries usually have normal brain function, researchers have been able to use the signals sent off by the brain to control a computer-assisted device. Hand and arm orthotics have been used with this device as a way to give hand function back to patients with paralysis. The ability to restore any amount of function in a paralyzed patient is a huge step in the patient having some normalcy in their life after spinal cord injury. [5]

IDEAL SOLUTION STATEMENT

The ideal solution should be able to prevent a persistent vegetative state or death due to secondary injury caused by TBI.

NEED CRITERIA

MUST HAVES

- Prevent Persistent vegetative state or death
- Non-invasive and continuous monitoring of ICP
- Affordable (daily cost should not exceed 10% of post-op ICU cost)
- Post-op GCS should not decline up to POD_{14}

NICE TO HAVE

- Easy-to-use (by an ICU nurse)
- Affordable (daily cost should not exceed 5% of post-op ICU cost)
- GCS should not go below 10/15 up to POD_{14}

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8. ALZHEIMER'S DISEASE

BACKGROUND

Alzheimer's Disease (AD) is a chronic neurodegenerative disease that progressively worsens with time. AD is the most common form of dementia and begins after the age of 65 years. The disease is characterized by progressive memory loss, inability to recognize people and places, cognitive decline, and mood swings. In 2015, AD resulted in nearly 2 million deaths worldwide. AD invariably affects the family and caregivers, since the patient entirely depends on them for his/her day to day needs. There is no early and accurate way to diagnose or effective way to treat AD [1]. It is an incurable disease with a long preclinical period and progressive course. In AD, plaques develop in the hippocampus, a structure deep in the brain that helps to encode memories, and in other areas of the cerebral cortex that are involved in thinking and making decisions.

AD is the sixth leading cause of death in the United States, accounting for 3.6% of all deaths in 2014. In the United States alone, approximately 6.08 million Americans had either a clinical AD or mild cognitive impairment due to AD in 2017. That number is expected to grow to 15 million by 2060. Economically, AD is a major public health problem. Total payments in 2017 for health care and long-term care for all individuals with AD or other dementias are estimated at $259 billion. By 2050, these costs could rise as high as $1.1 trillion.[1]

OBSERVATION

Presentation

77 year-old-male was brought into OPD due to the slowness of movement (bradykinesia), and short-term memory loss that had been developing over the past 2 years.

History

The patient had no history of trauma or any other related comorbidity

Treatment

MRI scan was done which showed atrophy near the hippocampus and lateral ventricular enlargement, which are both classic signs of AD. The patient was diagnosed with mild to moderate AD. The patient was started on memantine (NMDA receptor antagonist) daily dose.

There was little to no improvement over 2-3 years after which the patient started to deteriorate further. The patient was also put on Acetylcholinesterase inhibitors. There was mild retardation in the rate of deterioration over one year, after which the patient started to deteriorate even faster. Over 7 years (84 years old), patient progressed to severe Alzheimer's Disease. The doctor wanted to start glutamate, but the disease had progressed to a point where no treatment would work. The patient failed to recognize people or places. He progressed from only being able to use phrases to being able to use words only. Most communication was through facial

expression. A full-time caregiver was employed to take care of the patient. The patient died at the age of 85 years due to Pneumonia.

THE PROBLEM

There is no solution to cure or manage (significantly delay the progression of the disease) of AD. The disease mechanism has not been understood completely, which hampers the way for effective solutions. The current standard of care is largely palliative with full-time trained people looking after the patient, especially when the disease has progressed to severe stages.

A common problem with all long-term medication, including those for the treatment of Alzheimer's Disease, is that the strength of the dosage needs to be progressively increased with time for the same or increased efficiency. Most common cause for this is the development of the body's immunity to the same. The side effects of these medications also increase progressively with increase in dosage. Beyond a certain point, the benefits of the medication are less than the degree of side effects and hence, the dosage has to be saturated. Heavy medication almost invariably puts a heavy load on the kidneys and liver. Liver Function Tests (LFTs) and Renal Function Tests (RFTs) need to be periodically done to ensure that medication is not taking its toll on the patient.

NEED STATEMENT

A better way to treat impaired cognitive function, memory loss, disorientation and aphasia in patients with Alzheimer's Disease (AD)

FILTERING PROCESS

Final Score = 13.5

Rank = 9

MARKET POTENTIAL

More than 2 lakh annual incidence rate

More than 2 million people living with AD

Nearly 3 lakh annual deaths due to AD [2]

COMPETITIVE LANDSCAPE

NMDA receptor antagonists and Acetylcholinesterase inhibitors are the two classes of drugs which have shown mild improvements for some period of time (1-5 years) when the disease in mild to moderate stages. There are no drugs that can slow down the disease when it has progressed to the severe stage. Psychosocial intervention has shown no measurable improvements in retardation of disease progression.

AVAILABLE OPTIONS

Drugs have been the mainstay treatment for managing Alzheimer's Disease. NMDA receptor antagonists and Acetylcholinesterase inhibitors are the commonly used drugs. However, their effect is limited to when the disease is in the early to moderate stages. Even then, the effect is not often not significant to improve quality of life. Glutamate has been used to improve neurotransmitter function, but high risks of excitotoxicity leading to brain cell death exist. The side-effects of long-term use of these drugs means that there is still no clear and proven treatment for AD. [3, 4]

Recent advances:

Deep brain stimulation: Scientists placed hair-thin electrodes into the brain to deliver electricity. The electricity delivery is designed to increase the activity of the nucleus basalis of Meynert, which is the area of the forebrain that is degenerated in Parkinson's and Alzheimer's diseases. [5]

Rehabilitation Technology: Numerous devices are software are now available and being developed to help in the rehabilitation of patients with AD. So far they have had moderate success but do not help in halting/reversing the disease progression. [5]

Non-invasive brain stimulation: Using an MRI, scientists were able to determine where memory-related brain networks were located and stimulated them with non-invasive electromagnetic stimulation. The brain stimulation lasted 24 hours and correlated with brain activity changes. This non-invasive stimulation gives scientists a better understanding of memory improvement. Previous brain stimulation only lasted a short amount of time and have short-lived effects on thinking abilities. [5]

Brainwave oscillations: These technique are used to induce brainwave oscillations to treat Alzheimer's disease symptoms. It is presently in the early stages of research and development and plans to start using the techniques that it has used in mouse models in humans. [5]

IDEAL SOLUTION STATEMENT

The solution should be able to quantifiably improve Cognitive function over time as indicated by the Montreal Cognitive Assessment (MoCA) or Mini-Mental State Examination (MMSE) tests. The solution should not have any side-effects and should take effect before the disease progresses to moderate to severe stages.

NEED CRITERIA

MUST HAVES

- Quantifiably improve Cognitive function
- Prevent progress of short-term memory loss
- Prevent aphasia
- No side-effects of treatment (such as those caused by current medication)

NICE TO HAVE:

- Easy-to-use (by any trained doctor)
- Affordable to most patients (comparable to existing cost of management)
- Solution should be portable

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9. BRAIN TUMOR

BACKGROUND

A brain tumor is formed when abnormal cells grow rapidly inside the brain. When the tumor grows large enough in size, it leads to increased Intracranial Pressure (ICP)e. A decompressive craniotomy with tumor resection is the first line of treatment in reducing the ICP. However, most of the Indian population cannot afford a brain surgery in a private hospital, whereas government hospitals are overburdened with surgeries. India has around 1500 neurosurgeons, which does not meet India's demand for neurosurgeons performing surgeries at economical prices for the vast majority of the country [1].

OBSERVATION

Presentation

A 46-year-old male came into the OPD of a government tertiary care hospital after being referred from a Primary Healthcare Centre (PHC). His family brought him with mild impairment of speech and occasional inability to recognize people.

History

The patient had no other medical history or related comorbidity.

Treatment

The neurologist asked for an MRI which revealed a tumor in the parieto-occipital region of the brain. Surgery was planned for a month later due to unavailability of Neuro Operation theatre (OT) before a month.

During the surgery, the surgeon could not resect all of the tumor due to its contacts with major arteries. Hence, the doctor resected as much as possible and said that the patient will have to be monitored for any re-growth of the tumor. Histopathology of the tumor was done and the tumor was reported to be a high-grade Glioblastoma Multiforme (GBM). The patient was followed up with IV chemotherapy for 6 months at the District Hospital which was 300 kilometers from his home. Government hospitals do not have newer non-invasive radiation surgery tools such as stereotactic radiation surgery, which helps in tumor resection in difficult to access places.

The patient was advised to do an MRI every 4 months for the next 2 years to confirm that the tumor is not re-growing. The patient lived 800 kilometers from the THC but was willing to travel the distance for the free treatment. After 1 year of follow-up, the tumor was confirmed to be re-growing and hence required another surgery. However, the Neurosurgery OT was booked for the next several months with equally urgent surgeries. The Neurosurgery department maintains a priority list of which patients require surgery urgently. This patient was put down on the list for a surgery which was scheduled 6 months later. When the department called the patient 6 months later, the family informed that the patient had died.

PROBLEM

A tumor resection surgery via craniotomy costs up to 5 lakhs in a private hospital. Most of the rural population cannot afford such a surgery. Hence, they resort to government hospitals. Government hospitals are burdened with surgeries for 6 days in a week. Decompressive Craniotomy with tumor resection typically takes at least 5-6 hours and can go up to 24 hours depending on the complexity of the tumor. Hence, neurosurgery departments generally handle one such case per day. Most of the patients have to wait until it's their turn for surgery. Many patients die during the wait. Chemotherapy is relatively ineffective in completely killing malignant primary tumors.

NEED STATEMENT

An effective and reliable method to allow for visualisation and complete clearance of brain tumors in patients at tertiary care centres to avoid recurrences

FILTERING PROCESS

Final Score = 13.5

Rank = 9

MARKET POTENTIAL

More than 1 lakh annual incidence rate of a malignant brain tumor. [2,3]

COMPETITIVE LANDSCAPE

Most of the brain tumors present with neurological symptoms once the tumor has grown to a large enough size that chemotherapy and radiation therapy in themselves cannot completely kill all the tumor cells. Hence, surgery via craniotomy is the most common line of treatment. Chemotherapy and Radiation therapy is often used to get rid of tumor tissue that is unreachable in the brain. However, radiation therapy and chemotherapy have their own respective side-effects.

CURRENT TREATMENT

- **Surgery via Craniotomy –** This involves removing a piece of the cranium by creating burr holes. The tumor is then resected. [4]
- **Radiation surgery** – Stereotactic radiosurgery involves sending high energy radiation from various directions to meet at a common point (location of the tumor). The joint radiation created high energy which is converted to heat and hence burns the tumor tissue. [5]
- **Chemotherapy** – Chemotherapeutic agents are delivered either orally or through an intravenous cannula. However, chemotherapy is rather ineffective in killing primary malignant tumors, which accounts for most of the high-grade brain tumors.

- **Radiation Therapy –** Radiation therapy involves dispersing ionizing radiation to the target location. Radiation therapy has its own set of side-effects.

IDEAL SOLUTION STATEMENT

The solution should be able to resect all of the tumor within one attempt, especially if the solution is invasive. The solution should be affordable to the rural population of India.

NEED CRITERIA

MUST HAVES

- Remove the entire tumor (with margin) in a single attempt
- Should allow for confirmation of clearance (visual or otherwise)
- Cost to patient should not be more than 20% of current neurosurgery

NICE TO HAVE

- No side-effects such as those caused by chemo or radiation therapy
- Should be easy to use (any trained neurosurgeon should be able to use the solution)

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10. SWALLOWING FUNCTION ASSESSMENT

BACKGROUND

Aspiration is defined as the inhalation of either oropharyngeal or gastric contents into the lower airways, that is, the act of taking foreign material into the lungs. This can cause a number of syndromes determined by the quantity and nature of the aspirated material, the frequency of aspiration, and the host factors that predispose the patient to aspiration and modify the response. Almost all patients who develop aspiration pneumonia have one or more of the predisposing conditions such as alcoholism, drug overdose, seizures, stroke, head trauma, general anesthesia or intracranial mass lesion. [1] Several stroke patients lose control over swallowing function due to hypoxia in certain regions of the brain. During rehabilitation in the hospital, the patient's swallowing function has to be repeatedly evaluated, until which, the patient is fed via a nasogastric tube. There is a high risk of the patient developing Aspiration Pneumonia (AP) when swallow tests are done using water fed through a tablespoon [1]. Nearly 30% of all stroke patients require swallow function tests. [1] Several studies suggest that 5-15% of the 4.5 million cases of community-acquired pneumonia (CAP) result from aspiration pneumonia. A retrospective review found that the 30-day mortality rate from aspiration pneumonia is 21% overall and slightly higher in healthcare-associated aspiration pneumonia (29.7%).

OBSERVATION

Presentation

A 67-year-old female was admitted to emergency with sudden onset weakness in left upper and lower limbs, along with deviation of mouth to the right.

History

The patient was a regular consumer of tobacco until 3 years ago. The patient had a history of a minor ischemic Cerebro-Vascular Accident (CVA) (acute pre-central gyrus infarct due to right middle cerebral artery block) 3 years ago. The patient was on anti-stroke medication and had an almost complete recovery in 6 months. She stopped her medication by herself after she had completely regained control over the affected skeletal muscles.

Days of admission

On the day of admission, the MRI revealed a frontal-parietal infarct along with mild compression of the right lateral ventricle. The patient was put on anti-stroke and anti-seizure medication. An Echocardiogram showed a sclerosed aortic valve along with aortic regurgitation. The left atrium was dilated. Doctors concluded that this was a cardio-embolic stroke. The patient was kept in the hospital to recover from the stroke.

Three days later, she had a Generalized Tonic-Clonic Seizure (GTCS) and patient progressed to status epilepticus. The patient was sedated and shifted to ICU, where she was intubated. A few

days later the patient was shifted to a regular ward since there were no more episodes of seizures. The patient was on Nasogastric Tube for feeding. Doctors were unable to assess any improvement in the swallowing action of the patient. Doctors tried to give a teaspoon of water to assess swallowing. There was a high risk of aspirating the water into the lungs. Doctors said that many patients in the past have contracted pneumonia both before and after evaluation of swallowing via this crude method.

THE PROBLEM

Doctors do not have an affordable, portable and easy-to-use solution to assess swallowing functionality. A Functional Endoscopic Evaluation of Swallowing (FEES) is an expensive and skilled solution that cannot be used on a daily basis. Hence, neurologists resort to giving a little water to drinking in a teaspoon. If the patient is able to receive the water well repeatedly, they proceed with soft food items. Patients who have not regained full control over swallowing end up having the water enter the lungs, which leads to infected lungs (Pneumonia/Pneumonitis). Currently available solutions to assess swallowing function are both expensive and non-portable.

NEED STATEMENT

A quantifiable, portable and affordable way to assess swallowing function in post-stroke patients at Tertiary Healthcare Centres to avoid the high risk of aspiration pneumonitis / Pneumonia.

FILTERING PROCESS

Final Score = 13.5

Rank = 10

MARKET POTENTIAL

There are 15 lakh new reported cases of stroke every year.

Dysphagia occurs in 55% of all acute stroke patients.

At least 20% of these cases are at high risk of developing Aspiration Pneumonia (AP): At least 3 lakh cases of AP every year. [2]

COMPETITIVE LANDSCAPE

There is no portable and affordable technology that can accurately assess swallowing function. Due to the lack of technology, neurologists often resort to risky methods such as providing a measured quantity of water to drink. The doctor then manually observes the throat along with any cough or gag reflex that may be present.

X-ray and endoscopy-based methods are available. However, they are not preferred to be used on a daily basis due to the cost of the scan and the immobility of the technology. Additionally, these technologies also have their own side-effects.

AVAILABLE OPTIONS

- **Fibreoptic Endoscopic Evaluation of Swallowing (FEES)** - This is an endoscope which helps to visualize the tongue and epiglottis and judge their functionality. Based on this, doctors decide on whether the patient is ready to start receiving food from the mouth. This requires a skilled doctor to both use the equipment as well as evaluate the swallowing function. Endoscopes are immobile devices and are expensive to the patient for daily usage on the same patient. [3]
- **Barium Swallow Test** – The patient is made to drink a Barium Sulphate solution while an x-ray video is captured. The Barium Sulphate absorbs x-ray radiation and hence shows up on an x-ray. The video clearly shows the movement of the liquid from the mouth to the stomach. Rear-tongue and epiglottis function is judged from this video. Patients may be allergic to Barium Sulphate and go into anaphylactic shock. Patients may also develop constipation. Long-term effects from the x-ray radiation include the high risk of cancer. [4]
- **Toronto Bedside Swallowing Screening Test –** This involves delivering 10 teaspoons of water to judge whether the patient has dysphagia or not. However, there is a high risk of the water aspirating into the lungs, hence inducing pneumonia. [5]

IDEAL SOLUTION STATEMENT

The solution must be designed such that it can be used daily on the same patient bedside, without incurring much cost to the patient. The solution must be easy-to-use and should give a quantified assessment of swallowing function.

NEED CRITERIA

MUST HAVES

- Quantified assessment of swallowing (scale of 1 to 10)
- Easy-to-use (ideally usable by interns and trained nurses)
- Portability (bed-side usability)
- Cost to the patient should not cross 20% of current management costs for stroke

NICE TO HAVE

- Non-invasive method
- Any nurse should be able to use (with simple instructions)
- Should directly indicate whether doctor swallowing of food should be tried or not
- Handheld device

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