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Original Article

Integrated case-based clinical approach in understanding pathways, complexities, pitfalls and challenges in neurodegenerative disorders

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Abstract: Introduction: This paper presents 5 cases of neurodegenerative disorders from our tertiary care rural hospital in south India. The purpose of this paper is to generate an emerging common theme by thematic analysis of clinical data from each of these patients. A theme emerged, we identified that there was a common clinical ground in patients with movement disorders and psychiatric symptoms. From this common theme, these patients eventually went on to develop different courses of illnesses. Methodology: Clinical analysis of a case series of 5 patients with neurodegenerative disorders attending the Medicine or Psychiatry services of our hospital. Conclusion: A clear & consistent association between movement disorders and psychiatric symptoms was found. Although our data is limited, we conclude that movement disorders can be early clinical markers of organic psychopathology. However, we are aware that this association can be confounded by substance abuse, stress, sleep disruption and even therapeutic interventions, and thus these factors were accounted for and yet we conclude that movement disorders can be early clinical indictors of organic psychopathology.

Keywords: Neurodegenerative disorders, organic psychopathology, case-based blended learning ecosystem, movement disorders, Parkinson's disease

Introduction

In recent times, the distinction between organic and functional psychoses has become blurred and these are better described as primary and secondary psychoses, where "secondary" refers to an identifiable pathogenic substrate [1]. Neurodegenerative disorders are known to cause delusions and psychosis by extensive loss of acetylcholine and dopamine releasing projection neurons in the basal forebrain [2]. Due to disrupted brain circuitry, with time these brain areas atrophy and psychosis can occur. In addition to structural alterations, faulty dopamine signals and the loss of receptor modulation can provoke delusions in both primary and secondary psychosis. This hypothesis merits further research [2].

Our daily clinical practice employs the use of case-based blended learning ecosystem (CBBLE) [3] in psychiatry and neurology and aims at reducing misdiagnosis and mistreatment. In this system, concerted team communication and collaborative learning between participants provide evidence-based, patient-centred inputs across an online platform so the learning outcome of participants is coupled to improved patient outcomes [3].

In this paper, we evaluated the similarities and differences among 5 cases of neurodegenerative disorders who presented to our hospital. The detailed case histories, the multidisciplinary team involvement, and the rapid uptake of reviewing the literature to solve diagnostic and therapeutic uncertainties through our CB-

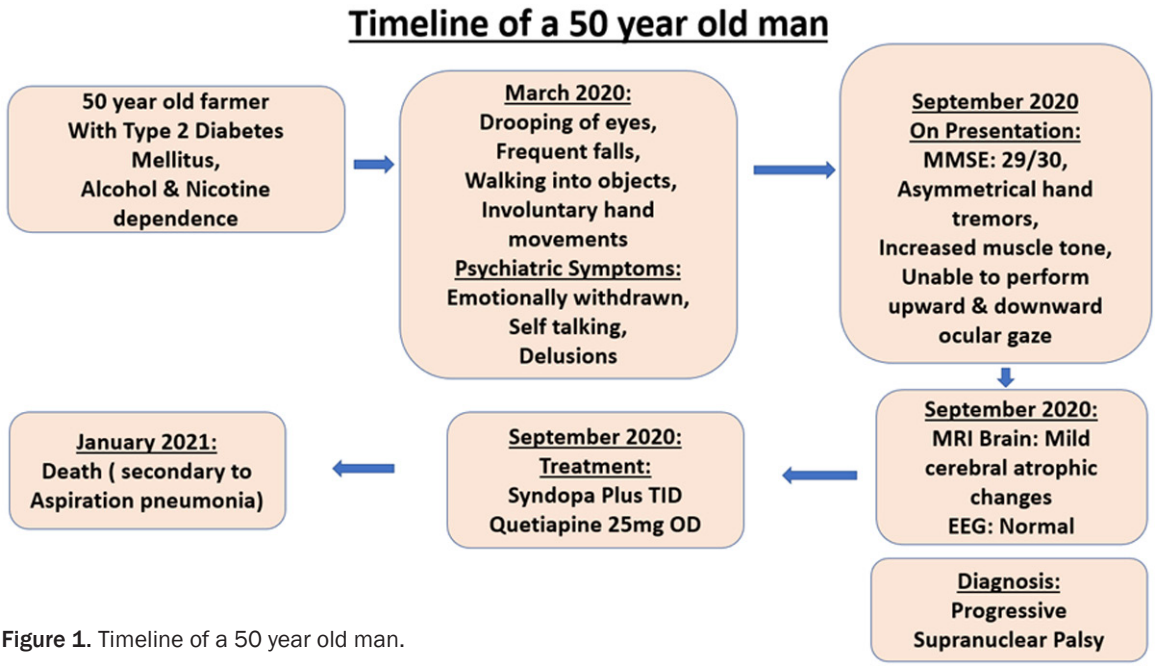

Figure 1. Timeline of a 50 year old man.

BLE system is also highlighted through these cases.

Case 1

A 50-year-old farmer with a history of alcohol and nicotine dependence and type 2 diabetes mellitus (DM), presented with frequent falls, drooping of eyes, walking into objects, and involuntary coarse hand movements. He also reported progressive difficulty in keeping his eyes open, worsened by blinking. His wife reported that since a few months, he was emotionally withdrawn and stopped conversing with others, which was his usual self. She also reported that he had been talking to himself for the past 6 months. She also reported him developing delusions of persecution. While walking, he would hardly look around or sideways and there was minimal arm swing. He noticed intermittent involuntary bilateral hand movements, while doing his fieldwork, particularly noting that these abated with activity. At presentation, the patient was noted to have a mask like facies and was also noted to be emotionally withdrawn. His mini-mental state examination (MMSE) score was normal (29/30). The MMSE is a widely used test of cognitive function among the elderly; it includes tests of orientation, attention, memory, language, and visual-spatial skills [4]. He had asymmetrical, small, rhythmic movements of his hands and fingers. Central nervous system (CNS) examination was significant for, increased muscle tone in all four limbs and brisk deep tendon reflexes (biceps, triceps, and knee). His plantar reflex was normal. He was unable to perform upward and downward gaze movements pointing towards oculomotor nerve involvement. His routine blood work was normal, neuroimaging showed mild cerebral atrophy.

He was diagnosed with a Parkinson-plus syndrome with progressive supranuclear palsy and discharged with a prescription for a thrice-daily combination of levodopa and carbidopa along with once-daily quetiapine. A brief timeline of events for case 1 is illustrated in **Figure 1**.

Case 2

A 66-year-old farmer with no significant past or family history of mental illness presented with gross rigidity in his body and asymmetric tremors for 4 to 5 years. He also reported visual hallucinations, memory deterioration, disinhibited behaviour, fearfulness, insomnias, and episodes of aimless wandering for the past 2 years. According to his family members, his voice became muffled and lower in intensity, and he would answer only in one or two words, unlike his previous self. His family members noted that the pace of his gait and normal arm swings were remarkably reduced in the last 3

years. He was started on a twice daily combination of 110 mg levodopa/carbidopa 3 years ago by his local district neurologist. However, there was minimal response and shaky, pill-rolling movements of distal hands gradually increased in frequency, intensity and duration with a typical static pattern leaving him incapacitated and unable to hold any objects for the last 6 months.

For disinhibited behaviour, he was prescribed 5 mg Haloperidol for a week by a psychiatrist after which his rigidity and bradykinesia worsened. He further developed confusion when he was put on 6 mg/day of trihexyphenidyl. He stopped identifying people visiting his home with increasing episodes of disinhibited behaviour, talking to himself, visual hallucinations, fearfulness, delusions of persecution, and aimless wandering. He reported seeing unfamiliar people standing in front and observing him, or running in his veranda, at other times he would behave as if his relatives were sitting beside him and on several occasions, he screamed as if snakes were crawling around his bed/house. His self-care, social interactions with his friends and his usual activities like going to the vegetable market and attending social functions decreased. There was no history of headache, seizures, head injury, or vomiting in the morning nor any history of hyperorality and hypersexuality.

On examination, he did not have any signs of meningeal irritation, spinal, or cranial deformity. He was conscious, oriented to time and place but was confused about the people around him. His mood was depressed and labile. His cognition was impaired (MMSE 11/30), and memory registration and retrieval were poor and there were gross deficits in recent, remote, and working memory. His speech was muffled, low volume, curtailed and monotonous without inflection or prosody. His judgement, praxis, simple calculations, object naming, logical thinking and insight were impaired. His posture was stooped and had a festinating gait. He had symmetrical tremulousness of his distal upper limbs with typical 6 Hz frequency, coarse, pill-rolling tremors. He also had increased muscle tone with 'cog-wheel' rigidity of forearms and wrists. Examination of cranial nerves was normal.

An axial T2 weighted magnetic resonance imaging (MRI) brain showed asymmetric cerebral atrophy with mild, generalized enlargement of fronto-temporo-parietal sulci and mild decrease signal intensity of basal ganglia with apparently normal width of pars compacta in substantia nigra without any clear depigmentation or blurring or thinning in contrast to what is typically seen in Parkinson's Disease. There was also evidence of mild thinning of the postcentral gyrus (left > right side) possibly indicating early cortico-basal degeneration (CBD). Midbrain tegmentum areas appeared smaller in size than normal; however, pons and cerebellar volumes were normal and there were no T2 hypointensities in either putamen or caudate nucleus suggestive of multiple system atrophy.

A diagnosis of Parkinson's plus syndrome was made (Parkinson's disease with dementia and cortico-basal degeneration). He was started on quetiapine 50 mg at night, his levodopa/carbidopa combination (110 mg/day) dose was increased to thrice a day, 10 mg morning memantine was started for his dementia which was gradually increased to 20 mg/day and 60 mg propranolol was added for his tremors. 2 mg of lorazepam was given in the initial few days for his sleep disturbances along with quetiapine. A serial MMSE was implemented weekly showing an improvement of 3 to 4 points over 12 to 16 weeks. His Alzheimer's disease assessment cognitive score improved from baseline 26 to 36. He showed marked improvement in hallucinations, delusions, disinhibitory and wandering behaviour and in his sleep and personal care. He responded positively to rehabilitation for routine and basic needs and use of directions, in addition, specific symbols and behavioural principles were taught to the family. His bradykinesia & tremors were mildly reduced but his rigidity persisted. After 6 months, he deteriorated markedly in cognitive functions (MMSE score in October 2019 was 10), developed apathy and worsened in his parkinsonian symptoms, especially rigidity and akinesia and he was not responsive to even four times daily levodopa (110 mg). Eventually, he developed difficulty in identifying relatives, upper limb myoclonic jerks and dystonia in the neck and upper limbs leading to dysphagia. His dose of memantine was increased to 30 mg/day and he was started on rivastigmine 3 mg/day, with lorazepam 6 mg/day. Lorazepam was withdrawn while he remained on the other prescribed interventions, however,

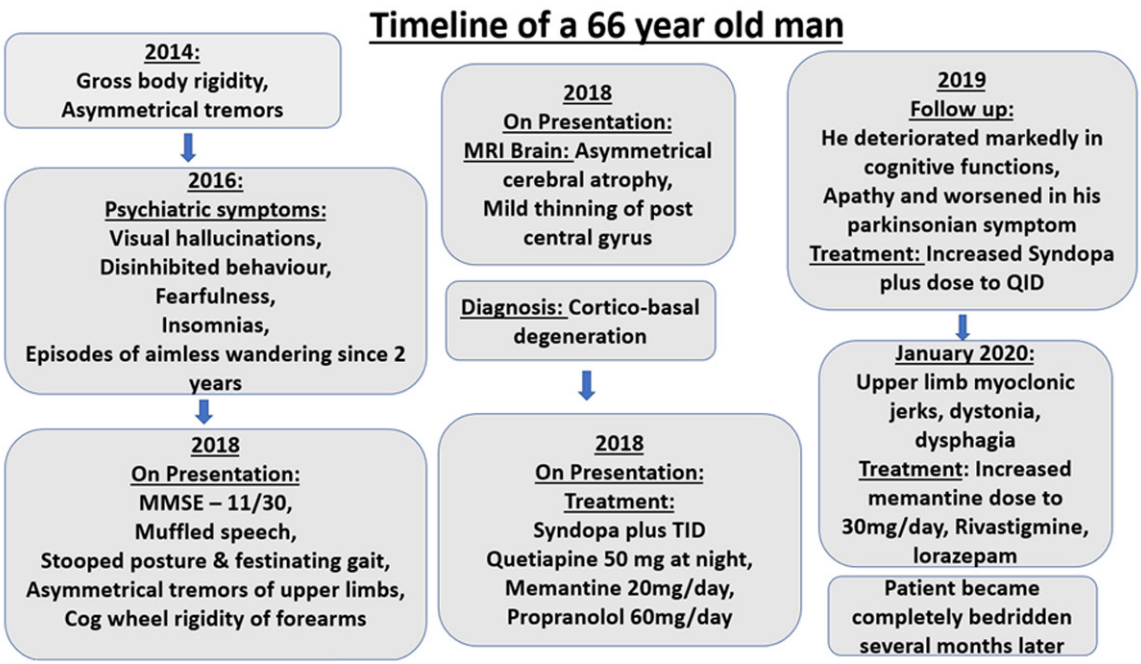

Figure 2. Timeline of a 66 year old man.

this produced no specific improvements, and the patient became bedridden with deteriorating quality of life. A brief timeline of events for case 2 is illustrated in **Figure 2**.

Case 3

A 67-year-old, right-handed woman presented with rhythmic, involuntary movements of her right forearm and her left index finger and thumb and intermittent stiffness for 4 to 5 months. Her husband started noticing these involuntary movements insidiously while she was taking her food plate and eating. These movements worsened with rest and were present intermittently when she went to sleep. She reported having no voluntary control on these movements and attributed them to aging. Gradually the tremors worsened, and intermittent stiffness commenced in her left hand as she noticed difficulty in initiating movements when trying to put on her clothes. In addition, she also reported difficulty in holding things with her left hand too, since a few weeks.

For the past 3 weeks, her husband reported that she did not involve herself in family conversations like her previous self and she stopped responding to people or would give only a delayed and curtailed response.

On examination, she was conscious, oriented to time, place, and person. She had a mask-like face. She presented with asymmetrical rhythmic, oscillatory, small amplitude movements of distal muscles of her right forearm and pill-rolling tremors were observed involving the left thumb and index finger. Her tremors were present at rest but were absent during motor activity. She had slow initiation of motor activity and a delayed verbal response during interviews. Her limbs had lead pipe rigidity and all her reflexes were exaggerated. Her MMSE score was 25/30. On cardiovascular system examination, a systolic murmur was heard in the aortic area till apex. Her laboratory investigations were within the normal range. Her 2D Echo revealed severe aortic stenosis, concentric left ventricular hypertrophy (LVH) with an ejection fraction of 55%. A magnetic resonance imaging of her brain showed mild cerebral atrophy, tiny calcifications without any oedema in the right lentiform nucleus and left temporal lobe, and a few small vessel ischaemic changes. She was diagnosed with Parkinson's disease and was discharged on levodopa and carbidopa combination 110 mg with vitamins B1, B6 and B12 combination thrice a day. A month later, she began having occasional, second person auditory hallucinations on follow-up at our hospital,

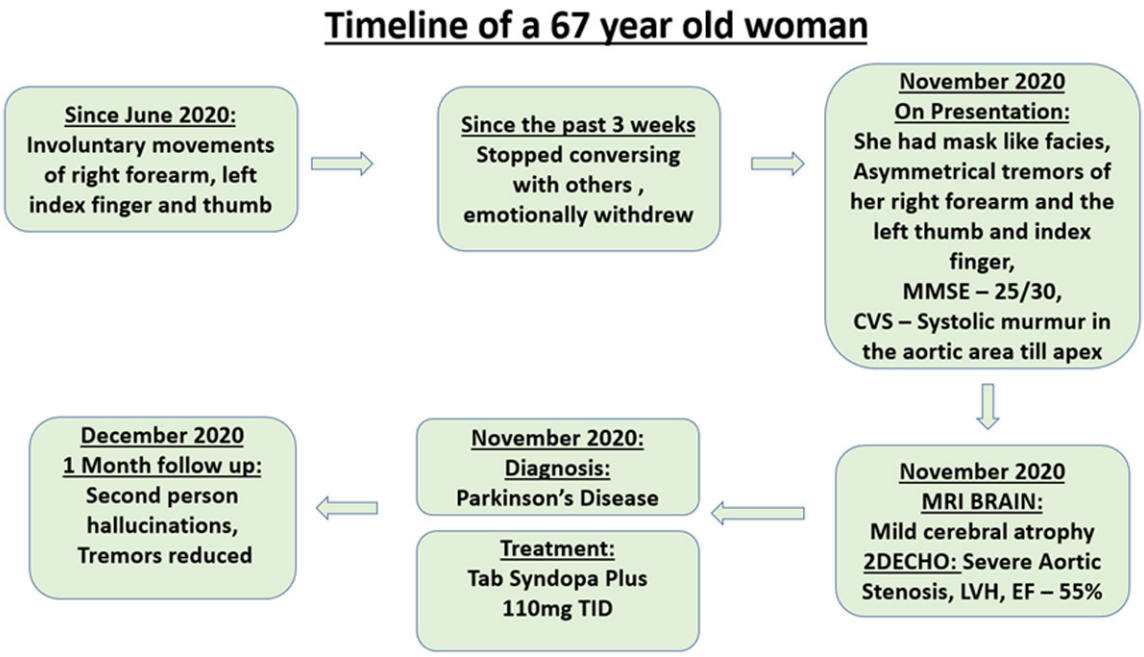

Figure 3. Timeline of a 67 year old woman.

however, her tremors were abating. A brief timeline of events for case 3 is illustrated in **Figure 3**.

Case 4

A 49-year-old right-handed man, working as a lecturer in Sanskrit and English without any significant past medical or psychiatric history, presented to us with an insidious onset of progressive, asymmetric involuntary movements of the index and middle fingers of his right hand. The patient reported that his movements worsened with rest and abated with voluntary activity. For the past 2 months, he had been unable to correct answer sheets or write properly because of to-and-fro oscillatory rhythmic movements of right thumb, resulting in an inability to maintain his hand stability. His handwriting was getting crowded and distorted with very small letters which at times were difficult to decipher. He started feeling stiffness in both wrists (right > left) throughout the range of motion while lifting or wiping movements and could feel its accentuation to his elbows at times. His brother and wife reported that he had been speaking in a monotonous tone for the past 2 months or so. For one month, these involuntary movements started appearing in his left hand as well and his gait had become slow, difficult to begin spontaneously, rather had to start with small, short steps and a forward stoop typical of Parkinson's disease. He also reported difficulty taking stairs and experienced episodic loss of balance at times. He further admitted that he had difficulty walking in the dark and felt he would fall without support. On further inquiry, he reported that for a few weeks he had no morning erections and a loss of sexual desire, however, his sleep and appetite were normal. He did not report difficulty in his activities of daily life. There was no history of swaying movement of the trunk while walking, overshooting his hand while picking up objects or difficulty in descending stairs. He denied having light headedness when waking up, stiffness in his lower limbs, cotton wool sensation of the floor, burning pain or difficulty in discriminating hot or cold perceptual stimuli. Further, there was no history suggestive of mood or psychotic disorder, substance use disorders, head injury, epilepsy, urinary incontinence, or memory deficits.

On examination, his baseline MMSE score was 29/30. Cogwheel rigidity was noted in his right wrist, coarse tongue tremors and micrographia were present. On a foot-tapping examination, movements of his right lower limb were found to be slower than his left lower limb. He also had a postural blood pressure drop of about

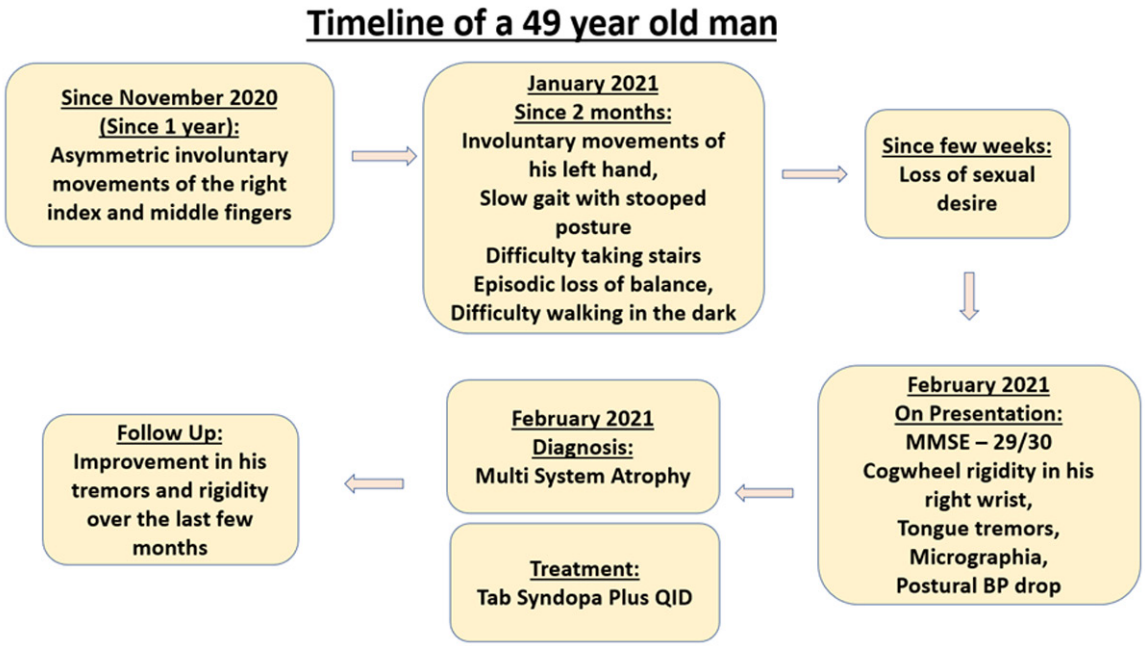

Figure 4. Timeline of a 49 year old man.

20/10 mm of Hg. The patient was diagnosed with Parkinson's disease and was started on oral syndopa (Levodopa plus carbidopa) 110 mg 4 times a day. He showed significant improvement in his tremors and rigidity over the last few months. A brief timeline of events for case 4 is illustrated in **Figure 4**.

Case 5

A 58-year-old, married man with a history of alcohol dependence, currently in remission for 3 years, presented to our hospital with acute onset of slurring of speech, deviation of angle of mouth to the right, urinary incontinence and significant cognitive decline for 6 months leading to termination from employment. He was found to carry temple belongings without permission when visiting temples for which he incurred the wrath of priests and family members and was further forbidden from attending nearby temples. He had significant forgetfulness for daily tasks, people he met or happenings around and he failed to recall names of objects asked. He became reluctant to shave, shower or bathe independently and would need assistance from his son for these tasks. Soon after, he began having difficulty recognizing friends and relatives but would recognize his son and wife. He would smile while naming his family members as he could not get their names properly. He sometimes had transient visual and/or auditory hallucinations. He was unable to button or unbutton his shirt or fold clothes, unlike his usual self. He repeated the same answer for every question asked (echolalia/perseverations) and would get impulsive or aggressive about petty issues but also calmed down within 5 to 10 minutes on his own as if nothing had happened. He also stopped laughing at jokes. For the last few weeks, he had difficulty in swallowing solids and liquids, with drooling from the left side of his mouth. He showed a remarkable delay in responding to commands, persistent urinary incontinence accompanied by intermittent wandering spells and disinhibited behaviour.

At presentation, the patient was conscious and cooperative, with an MMSE score of 9/30. His speech was slurred and incomprehensible, fluency was impaired, but repetitions were intact. Examination for parietal lobe functions revealed impaired right-left discrimination, visuospatial orientation, and construction abilities. Persistent prosopagnosia was noted on examination for functions of occipital lobe. On cranial nerves examination, there was clear evidence of left facial nerve palsy as we observed the loss of wrinkles over the left forehead and deviation of his mouth to the right. His biceps, triceps and supinator and knee reflexes were

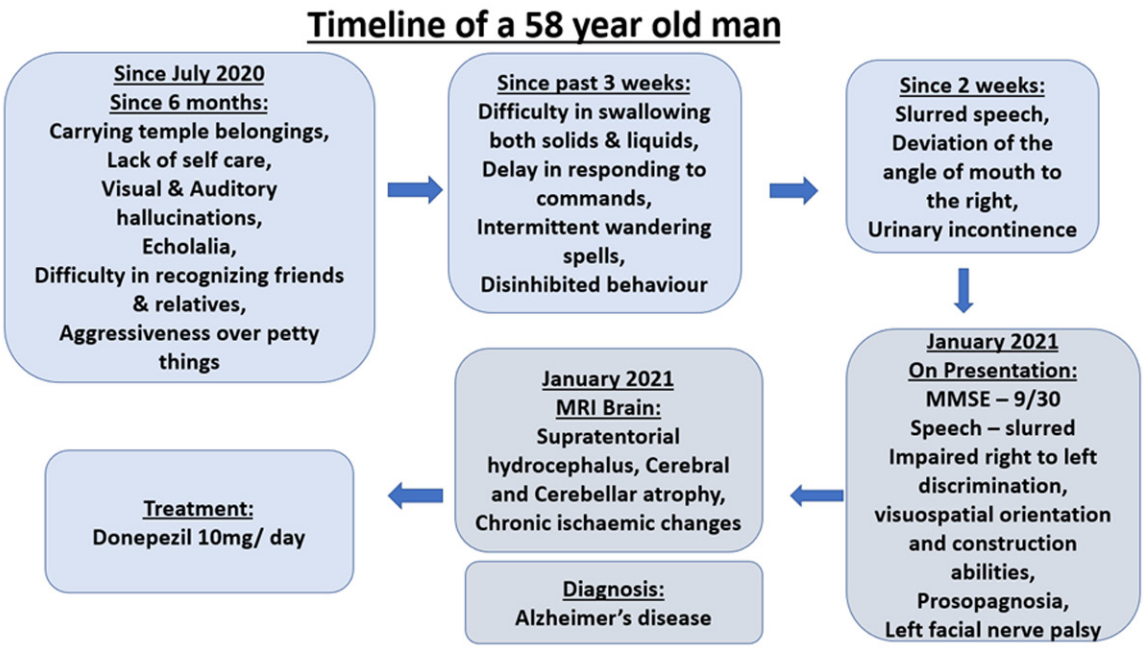

Figure 5. Timeline of a 58 year old man.

exaggerated. His metabolic profile was normal. MRI Brain showed supratentorial hydrocephalus, cerebral and cerebellar atrophy along with chronic ischaemic changes. He was diagnosed with probable Alzheimer's dementia and was started on donepezil 10 mg/day which was increased to 20 mg/day after a week. His MMSE score improved from 9/30 to 11/30 by week 6, however, he was lost to follow-up. A brief timeline of events for case 5 is illustrated in **Figure 5**.

Discussion

We performed this study within our existing framework of a CBBLE [3] when we received an invitation from the American Journal of Neurodegenerative Disorders (AJND). Psychoses can develop in patients with brain disorders secondary to neurodegenerative disorders, tumours, or cerebrovascular accidents. We explored the organic psychopathology of psychosis in neurodegenerative disorders. We postulate that movement disorder is an obvious, logical bio-clinical marker towards organic psychopathology of psychiatric symptoms found in neurodegenerative disorders. A detailed thematic analysis of the accessible events in these patients offered newer insights into the organic nature of psychiatry.

Localization of psychoses

Cummings was one of the first to correlate psychotic symptoms with the underlying pathobiology by focussing on delusions. He proposed that dysfunction of subcortical basal ganglia - limbic system interactions including abnormal dopamine neurotransmission leads to the formation of delusions [5]. Acute onset of delayed psychosis was observed after right temporo-parieto-occipital stroke or trauma in a study, in patients with no prior history of psychiatric illness [6-8]. Kumral and Ozturk prospectively observed mental status of patients with acute stroke and found 5% of them with right hemispheric lesions developed delusions and were more common with right posterior temporoparietal lesions [7]. A meta-analysis revealed that there was a greater grey matter volume reduction in the right inferior frontal gyrus and superior temporal gyrus among subjects at high risk of psychosis. These studies conclude that dysregulations in the right prefrontal cortex including the basal ganglia and the limbic system are associated with the development of delusions [8, 9].

Case discussion

Case 1 - a middle-aged man with progressive supranuclear palsy (PSP): This case clearly highlights the organic psychopathology that

underlies the comorbidity of movement disorder, and motor and psychiatric manifestations in a neurodegenerative disorder.

Our patient was diagnosed with progressive supranuclear palsy (PSP). PSP is an atypical variant of Parkinson's disease, also known as the Steele-Richardson-Olszewski syndrome [10]. This condition is characterized by supranuclear gaze palsy, progressive axial rigidity, pseudobulbar palsy, and mild dementia and dysphagia. Most patients with PSP present with moderate to severe apathy followed by disinhibition, depression, and sleep disorders [10]. Approximately 20% of PSP patients are noted to have impaired interpersonal functioning, communication problems, poor relationships, and difficulty in active engagement with family and friends. Emotion expression in PSP is more impaired than in Parkinson's disease [11].

This neurodegenerative disorder is histopathologically recognized by a neuronal loss with globose neurofibrillary tangles, and the presence of tau-positive inclusions in tufted astrocytes, and gliosis in the basal ganglia, cerebellum, and brain stem [12].

Case 2 - corticobasal degeneration: Our second patient was diagnosed with corticobasal degeneration (CBD). CBD is usually characterized by akinetic-rigid parkinsonism, dystonic and myoclonic movements, associated with cortical symptoms such as ideomotor apraxia, alien limb phenomena, aphasia, or sensory neglect [13]. Our case has typical symptoms of Parkinson's disease with gradually progressive cognitive decline and complex amalgamation of neuropsychiatric symptoms with clear functional deterioration. This patient received undocumented antipsychotics which worsened his parkinsonian features. The usual confusion of onset of independent Lewy body dementia (DLB) in Parkinson's disease always showed a great deal of controversy as few expert consensuses believe that it should occur within the first year after the onset of Parkinson's to call it as an independent entity since Alzheimer's Dementia (AD) like dementia can also occur in the progressive brain degeneration [14]. McKeith et al described the spectrum of clinical features in comparison between 21 cases of DLB and 37 cases of AD proven histologically [15]. The DLB patients tended to show mild cognitive impairment at presentation and more often showed depression as well as marked fluctuation. Almost half of the patients with DLB had falls or transient and unexplained loss of consciousness, which were rare in those with AD.

Case 3 - parkinson's disease: Our third patient's clinical presentation was consistent with Parkinson's disease. She had no symptoms suggestive of dementia but developed drug-induced auditory hallucinations. The presence of psychosis or dementia-like presentation is not uncommon in Parkinson's disease, since the nigrostriatal pathway is commonly involved in Parkinson's disease, with gradual impairment in mesolimbic and mesocortical pathways which may lead to some of the psychiatric manifestations [16]. Studies have reported common presentation of hallucinations (15%-40%) and delusions (10%) in patients on therapy for Parkinson's disease [16, 17].

Case 4 - multisystem atrophy: Our fourth patient's clinical presentation was consistent with multisystem atrophy. Multi-system atrophy (MSA) is a rare neurodegenerative disorder characterized by autonomic dysfunction, tremors, slow movements, muscle rigidity, postural instability [18]. In a study conducted with 30 MSA patients it was found that in 73% of patients with MSA, the first symptom was autonomic followed by motor symptoms in 10% [18]. Erectile dysfunction was found to be the most common symptom among these patients followed by postural instability, urinary hesitancy, and sleep disorders [19].

Case 5 - Alzheimer's dementia: Our patient presented with slurred speech, mouth deviation to the right, smacking of lips, urinary incontinence, visual hallucinations for 6 months and progressive dementia for 5 months. Alzheimer's disease (AD) is characterized by progressive dementia and is the most common cause of dementia. In patients with probable AD, higher incidence (51.3%) of hallucinations and delusions were reported at 4 years [20]. The accelerated cognitive decline may also be a predictive marker for their occurrence [20]. Studies have found that psychosis in AD was associated with more rapid cognitive decline and increased mortality rate [21, 22].

Diagnostic uncertainty & Role of clinical autopsy

Most of our cases had neuroimaging scans without dopaminergic deficit, the definitive diagnosis can be achieved in these patients only after a clinical brain autopsy. This report of a 50-year-old man, a doctor who was initially diagnosed with Parkinson's disease and later with multi-system atrophy but after his death, the brain autopsy revealed parkinsonism with Lewy body disease with brainstem predominance, further highlights how diagnostic uncertainty is a common challenge and needs to be addressed with certainty [23]. Accuracy of the aetiology of diagnosis is paramount for the family as well as for the population at-large for healthcare planning and research.

Clinical autopsy, also known as a pathological autopsy, is regarded as the gold standard for confirmation of the cause of death, which may be unknown to treating clinicians and relatives of the deceased. Studies have found significant discrepancies between premortal clinical diagnoses and autopsy findings in over 30% of patients [24]. For example, 16% of the cancers diagnosed at autopsy were unknown earlier. Similarly, a significant portion of deaths with uncertainty were found to have a myocardial infarction at autopsy, which can be presumed as the prevalence of atherosclerotic heart disease is also higher [25]. Despite its ominous importance, it remains underutilized in most medical institutions in India coupled with a declining trend even in most developed countries [25]. Several factors might be contributory to the barriers to this global health thanatology implementation problem in India such as overlooking the potential benefits of clinical autopsy among clinicians and the general public, religious views and stigma attached to body dissection of close ones [26]. Clinicians assume that the cause of death is already known or will not yield any additional information or that the relatives will oppose autopsy. In addition, financial reasons and advancing diagnostics may also undervalue the need for a clinical autopsy.

In this case series with neurodegenerative disorders, clinical autopsy could have shed light into the pathophysiology of, but this does not reveal the dynamic functional information needed to understand their daily living. Clinical autopsy procedures can increase knowledge of the disease systems, biological underpinnings and frailties that exacerbate the disease.

To increase the utilization of clinical autopsy procedures in India, facilities need to be expanded and all the stakeholders will need to get involved including the clinicians, policymakers, and lay public. Clinicians and policymakers need to understand the values of clinical autopsy in education, research, healthcare quality control and declare it as a useful investigative tool. The lay public, being an essential decision-maker, should be made aware of the potential benefits of the procedure. The medical institutions, particularly in rural areas, should build the infrastructure including forensic-pathology laboratories, equipment, and forensic experts within a well-defined policy framework. Autopsies are also one of the best ways to train pathologists and therefore, postgraduate institutions can also make an effort to implement a clinical autopsy training program involving a multidisciplinary clinical team. Undergraduate medical students should be well-taught about clinical autopsy.

Therapeutic uncertainty

In our patients with a neurodegenerative brain disorder, we found common ground in their presentation with a movement disorder along with psychiatric symptoms and eventual development of different courses of illness. In a visual representation of event timeline in neurodegenerative disorders, one notices progression in a downward spiral while with other brain injuries such as stroke it is much more rewarding as it is more about recovery. However, such therapeutic uncertainty in our cases could be minimized if regularly supported by a CBBLE that can not only offer evidence-based decision tools, but also a regular connection with global experts to improve documentation, transparency, and newer learning insights as well as innovation toward optimizing solutions [3]. Once the neurodegeneration begins, like in dementia, there is no cure for it nor does any curative treatment exist to modify or reverse its eventual progression. Available therapeutic interventions, therefore, target to improve cognitive functions.

Neurocognitive rehabilitation

Cognitive impairment is a very prevalent non-motor symptom in Parkinson's disease and

may present in the early stages of the disease [27]. Cognitive impairment deteriorates with the progression of the disease until dementia develops after 10-20 years [27, 28]. Studies show that mild cognitive impairment (MCI) is contributory to developing dementia and may be a prodromal stage for dementia in Parkinson's disease [28, 29]. Both cognitive decline and dementia can reduce the quality of life and functional disability of patients with Parkinson's disease [29]. Therefore, patients with Parkinson's disease require therapeutic strategies that treat cognitive impairment.

Cognitive rehabilitation programs have been found efficacious in improving cognitive declines and may improve functional disability and make brain changes in patients with Parkinson's disease [30]. Cognitive rehabilitation of fewer than 3 months has demonstrated brain connectivity and activation improvements [30]. In addition, the involvement of caregivers in cognitive rehabilitation is important [31]. However, the data on long-term maintenance of cognitive changes post-rehabilitation is still scant. In our series of cases, neurocognitive rehabilitation could not be performed, which is again a barrier to implementation in Indian rural medical institutions where hospitals often do not have a good neurorehabilitation centre with enough cognitive rehabilitation experts. Chronic, progressive, incurable disorders like Parkinson's disease may also become a barrier to offering these non-pharmacological therapeutics. Patients with neurological disorders are mostly concentrated in rural India [32]. A study conducted in rural areas in Uttar Pradesh found that economical constraints, lack of awareness, family negligence and transportation problems are majors for neurocognitive rehabilitation [33]. The policymakers should help institute cost-effective, accessible rehabilitation centers for under-served populations and take appropriate measures to remove those barriers. The public should be well-educated about the potential benefits of neurocognitive rehabilitation. Facilities should build a good infrastructure for neurorehabilitation.

Conclusion

A rural medical college receives few neurodegenerative disorder patients. The diagnostic and therapeutic challenges identified in the study need to be addressed toward the improvement of diagnostic and therapeutic outcomes in this population. Scholarly integration of medical education and research through a global CBBLE model in psychiatry and neurology may help address those challenges in the missing Indian setting as reflected in the discussion. A current functioning prototype is available and accessible at our institution although it needs better active participation toward scaling and sustainability.

Given the composite nature of neuropsychiatric disorders that have a vigorous and complementary base, the organic and functional discrimination of these disorders appears to be a valuable tool that aids in deciding and prioritizing the treatment plan. The functional-organic discrimination strives to distinguish clinical features which could be possibly elucidated by well remarkable biological changes, still prevailing as one of the pivotal diagnostic bases in present-day neuroscience.

Disclosure of conflict of interest

None.

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