Archives of Neurology and Neuro Disorders

Volume 1, Issue 1, 2018, PP: 17-20

Challenges in the Management of an Elderly Patient with Chronic Schizophrenia and Dementia

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Abstract

As the patients with Schizophrenia live longer, the emergence of cognitive complaints become challenging to the geriatricians and psychiatrists in terms of diagnosis, treatment and prognosis. This is taking into account the various drug combinations given to manage the psychotic symptoms of Schizophrenia, many of which contain anticholinergic properties which increase the risk of delirium among the elderly. In addition to drugs, the presence of depression and delirium are both common in the elderly and may have implications on diagnosis and long term outcome.

Keywords: *Schizophrenia, psychosis, dementia, delirium, depression*

Introduction

As the population ages, there will be more elderly living with schizophrenia surviving into their silver years. Individuals with schizophrenia have been shown to be more likely to develop dementia compared with healthy individuals, and this trend has been found in both Asian and European populations [1-3]. In Asian populations, this association also seems to be greater in women and in individuals aged greater than 65 years [1].

Over 25% (up to as much as 80%) of geriatric patients with schizophrenia have cognitive impairment[3]. Classically, Emil Kraepelin used the term 'dementia praecox' (or premature dementia) to describe schizophrenia in his Lehrbuch der Psychiatrie of 1893, where he emphasized that while there is progressive deterioration of cognition in schizophrenia,the domain of memory appears to remain spared. While most studies have shown that schizophrenia is a progressive dementing illness manifesting as a significant decline in cognition over time, some studies have found it to be a static encephalopathy with the cognitive impairment remaining relatively stable [3]. It is still unclear if the cognitive impairment in schizophrenia is a unique entity, or if it is a risk factor for the manifestation of a secondary dementia syndrome. Hence, there appears to be significant variability in cognitive outcomes for geriatric patients with schizophrenia. While it has been shown that schizophrenic patients who meet the criteria for dementia are most often diagnosed with a concomitant Alzheimer's disease [4], post-mortem studies of schizophrenics with cognitive decline have shown that Alzheimer's disease is diagnosable in less than 10%, and that there is no excess of vascular dementia, frontotemporal dementia or Lewy Body disease compared with non-schizophrenic controls, inevitably leading to the postulate that the cognitive decline seen in schizophrenia may be due to a unique neuropathological process [5,6].

Regardless of the etiology of cognitive decline, the management of concomitant schizophrenia and dementia remains challenging. In this case report, a patient with chronic schizophrenia who subsequently developed cognitive decline consistent with Alzheimer's dementia is discussed to highlight some of the difficulties faced in clinical evaluation, diagnosis and management.

Case Report

Madam T was a 75 year old lady with a long history of chronic schizophrenia under the care of psychiatrists for decades. She was labeled with treatment-resistant schizophrenia that had been managed with a combination of antipsychotics for her symptoms. She had been given electroconvulsive therapy (ECT), the last of which was administered 8 years prior to her current admission. She had also had a long history of

depression. Her family also noted a gradual decline in short term memory for a few years, and that she had been gradually becoming more dependent on her instrumental activities of daily living (IADL) as well as her basic activities of daily living (ADL). The attending psychiatrist diagnosed her as probable Alzheimer's disease from her clinical presentation. She had recently been put on long-term nasogastric tube feeding for worsening dysphagia with poor oral intake after an admission for pneumonia a couple of months prior.

Madam T was initially admitted to a general medical ward for a urinary tract infection with fever, where she was found to be more confused and agitated than usual, causing her to repeatedly pull out her nasogastric tube. She was catatonic, bed bound, uncommunicative, and drowsy. She was noted to have extrapyramidal side effects from chronic antipsychotic use with stiffness and dyskinesia. The dyskinesia manifested as a severe oral facial dyskinesia which interfered with her ability to feed orally, as well as dyskinesia affecting her trunk and limbs to the extent that she was constantly turning around in her bed. The nursing staff were fearful of her causing injuries to herself, thus she was put on physical restraints to control her movements. During the admission, in-house psychiatrist was consulted and she was diagnosed with delirium secondary to ongoing infection, electrolyte abnormalities, undernutrition and excessive psychotropic medications.

Her care was subsequently transferred to the dementia ward because of agitation and behavioural symptoms. Her medication list on transfer included olanzapine, benzhexol, mirtazapine, lorazepam (served when necessary for anxiety), and zopiclone (served when necessary for insomnia). In consult with psychiatry, it was noted that her schizophrenia was likely quiescent as patient no longer manifested positive symptoms, therefore a plan was formulated to gradually tail off her antipsychotic and antidyskinetic medications to minimize the number of psychotropic medications contributing to her delirium. Olanzapine and benzhexol were subsequently tailed off. As she remained persistently drowsy, lorazepam and zopiclone were also subsequently discontinued, and mirtazapine was changed to escitalopram to minimize sedation. She was also started on a trial of rivastigmine patch for probable Alzheimer's Disease after resolution of delirium. With the above medication adjustments, her behaviour, mood and appetite initially improved and she bagan to smile at us and communicated verbally with us. However, the oral and limb dyskinesia persisted which continued to affect her ability to feed properly and limited her quality of life. She was started on clonazepam with limited improvement in her dyskinesia, and the clonazepam dose had to be attenuated in view of its sedative side effects.

She continued to have inadequate oral intake on a background of moderate to severe dysphagia secondary to dementia and weight loss, exacerbated by underlying depression, profound deconditioning from recurrent infections, prolonged hospitalisation, chronic altered feeding habits, and intermittent oral dyskinesia. On oral feeding, she would only be able to take a few spoons of oral nutritional supplements a day. She persisted in oral refusal despite different strategies tried, including syringe feeding, feeding by spoon, and had previously rejected nasogastric tube on multiple occasions.Her altered feeding habits manifested as a tendency to opportunistically slurp her food, likely a compensatory mechanism to overcome her persistent oral dyskinesia. Decision was made not to reinsert a nasogastric tube as it was noted that tubes, lines and restraints worsened her agitation. Evaluation by gastroenterology also deemed her not a good candidate for a percutaneous endoscopic gastrostomy tube for feeding as she was at high risk of dislodging it. In the absence of tube feeding, we faced problems with medication compliance. Extensive discussion was held with her family, who noted that her poor feeding was secondary to multiple factors, many of which were not reversible. She was continued on comfort feeding to optimize her quality of life, but she gradually deteriorated and passed away shortly after discharge to hospice care.

Discussion

Schizophrenia is closely correlated with cognitive decline, and hence it can be difficult to differentiate schizophrenic patients in late-life from those with dementia, such as that resulting from Alzheimer's disease[7]. As a further confound, the treatments for intractable schizophrenia such as ECT have been known to worsen cognition [8], although the effect of antipsychotic use on cognition remain inconclusive [9]. It is hard to determine if the cognitive decline in our patient was a direct result of her schizophrenia, a consequence of her previous treatments, or the development of a comorbid dementia.

Many etiologies of dementia also present with psychotic symptoms which can be difficult to distinguish from the positive symptoms of schizophrenia. It is estimated that up to 70% of patients with Alzheimer's disease experience delusional symptoms, and up to 80% of patients with Lewy Body dementia experience visual hallucinations [10]. There is also expanding interest in the relationship between schizophrenia

and frontotemporal dementia in view of symptom similarity, familial co-morbidity and neuroanatomical changes, although the available literature does not definitively support a conclusive link between the two entities [11, 12]. It is noted that the nature of delusions in dementia tend to be based on memory impairment and misinterpretation of stimuli encountered in daily living, such as delusions of theft, phantom boarders, belief that a caregiver is an imposter (Capgras syndrome), belief that a caregiver will abandon the patient, or an unfaithful mate [7,10]. This differs from delusions in schizophrenia, which tend to be more paranoid or persecutory in nature [7]. In our patient, the positive symptoms of schizophrenia had become quiescent for several years, with no psychosis symptoms during her prolonged hospital stay and this is consistent with the finding that schizophrenics tend to have fewer hallucinations and delusions in late life [7].

In addition to her premorbid chronic schizophrenia and dementia, the management of Madam T was complicated by depression and superimposed delirium. Depression is prodromal feature for dementia and for schizophrenia, as well as a common complication or consequence of both [13]. While the positive symptoms of schizophrenia attenuate with age, the negative symptoms such as blunted affect, psychomotor retardation, mutism, social withdrawal and poverty of conversation tend to persist [7], and these symptoms are difficult to distinguish from those of unipolar depression.The diagnosis of dementia in a patient with schizophrenia is fraught with pitfalls, particularly when confounded with depression. This may be due to the fact that depression, schizophrenia and dementia have significant overlap in symptomology. It has even been postulated by Ha¨fner et al. that the three entities can be represented by a hierarchical model of preformed symptom patterns, with depression as a common response to stressors, schizophrenia (manifesting as depression and psychosis) as a result of brain dysfunction, and dementia (manifesting as depression, psychosis and cognitive decline) as a result of neurodegenerative brain disorders [13].

The combination of schizophrenia, dementia, depression and delirium in a single patient can easily lead to polypharmacy with multiple psychotropic medications [14]. Evidence of effective therapies in the context of such co-existing illnesses remains lacking [6]. While adult mental health services are more adept at managing schizophrenia, geriatricians and psychogeriatric services are more adept at managing dementia. To provide comprehensive care to such patients will require a multidisciplinary team, as it has been noted that memory clinics alone may not have the necessary expertise to adequately manage people with both schizophrenia and cognitive impairment [3].

Madam T was initially on a combination of antipsychotic, antidyskinetic, antidepressant and sedative medications. Many of these classes of drugs have anticholinergic properties with a negative impact on cognition and alertness. There is evidence to suggest that antipsychotics will improve cognition if the impairment is associated with psychotic symptoms [3], but our patient had a paucity of positive symptoms so it was questionable to persist with antipsychotic therapy, particularly when there is also evidence that antipsychotic medications are contraindicated in patients with primary dementia because of the increased risk of stroke and mortality [3,14]. There is limited evidence to suggest that acetylcholinesterase inhibitors (donepezil and rivastigmine) may be beneficial in the treatment of comorbid schizophrenia with dementia, and their effects are still being evaluated [3,6,15]. Mendelsohn et al. noted improvements in cognition, ADL and psychotic features after 12 weeks of rivastigmine use [15]. Madam T also initially improved cognitively when started on rivastigmine, but her persistent dyskinesia from protracted antipsychotic use continued to be difficult to overcome. Particular care should be taken when coming up with drug therapies for patients with comorbid schizophrenia and dementia, as a combination of cholinergic and anticholinergic medications may attenuate the effects of one or both drugs administered [16].

Conclusion

As life expectancy increases, the number of patients with comorbid schizophrenia and dementia will also increase. While the treatments for separate conditions are improving, therapeutic guidelines and effective therapies for co-existing conditions remain lacking. The recommended therapies for one condition may worsen the outcomes in other conditions, such as the risk to cognition from ECT or prolonged antipsychotic use. Furthermore, prolonged antipsychotic treatment may have a negative impact on physical function in the form of extrapyramidal side effects and tardive dyskinesia, exacerbating the already poor functional outcomes in dementia patients. Moving forward, it will be important to look carefully at the interaction between such comorbid presentations to optimize the treatment for such challenging patients.

References

[1] Ku, H, Lee EK, Lee KU et al. Higher prevalence of dementia in patients with schizophrenia: A nationwide population-based study. Asia-Pacific Psychiatry 2016(8); 145:153

[2] Ribe AR, Laursen TM, Charles M et al. Long-term Risk of Dementia in Persons With Schizophrenia: A Danish Population-Based Cohort Study. JAMA Psychiatry 2015(72:11); 1095:1101

[3] Radhakrishnan R, Butler R, Head L. Dementia in schizophrenia. Advances in psychiatric treatment 2012(18); 144:153

[4] Brodaty H, Sachdev P, Koschera A et al. Long-term outcome of late onset schizophrenia:5-year follow-up study. British Journal of Psychiatry 2003(183); 213:219

[5] de Vries PJ, Honer WG, Kemp PM et al. Dementia as a complication of schizophrenia. Journal of Neurology, Neurosurgery and Psychiatry 2001(70:5); 588

[6] Stryjer R, Strous RD, Faina B et al. Beneficial Effect of Donepezil Augmentation for the Management of Comorbid Schizophrenia and Dementia. Clinical Neuropharmacology 2003 (26:1); 12:17

[7] Song MH, Hamada H, Mimura M. Seniological Differences between Late-life Schizophrenia and Senile Dementia. The Keio Journal of Medicine 2014 (63:2); 34:38

[8] McClintock SM, Choi J, Deng ZD et al. Multifactorial determinants of the neurocognitive effects of electroconvulsive therapy. Journal of ECT 2014 (30); 165:176

[9] Wolf A, Leucht S, Pajonk FG. Do antipsychotics lead to cognitive impairment in dementia? A meta-analysis of randomized placebo-controlled trials. European Archives of Psychiatry and Clinical Neuroscience 2017 (267); 187:198

[10] Brendel RW, Stern TA. Psychotic Symptoms in the Elderly. Primary Care Companion Journal of Clinical Psychiatry 2005 (7:5); 238:241

[11] Harciarek ML, Malaspina D, Sun T et al. Schizophrenia and frontotemporal dementia: Shared causation? International Review of Psychiatry 2013(25:2); 168:177

[12] Cooper JJ, Ovsiew F. The Relationship Between Schizophrenia and Frontotemporal Dementia. Journal of Geriatric Psychiatry and Neurology 2013 (26:3); 131:137

[13] Ha¨fner H, Maurer K, Trendler G et al. Schizophrenia and depression: Challenging the paradigm of two separate diseases-A controlled study of schizophrenia, depression and healthy controls. Schizophrenia Research 2005 (77); 11:24

[14] Wu YH, Lai CY, Chang YS. Antipsychotic polypharmacy among elderly patients with schizophrenia and dementia during hospitalization at a Taiwanese psychiatric hospital. Psychogeriatrics 2015 (15); 7:13

[15] Mendelsohn E, Rosenthal M, Bohiri Y et al. Rivastigmine augmentation in the management of chronic schizophrenia with comorbid dementia: an open-label study investigating effects on cognition, behavior and activities of daily living. International Clinical Psychopharmacology 2004 (19): 319:324

[16] Carnahan RM, Lund BC, Perry PJ et al. The Concurrent Use of Anticholinergics and Cholinesterase Inhibitors: Rare Event or Common Practice? Journal of the American Geriatric Society 2004 (52); 2082:2087

Citation: **Dr. Jing Wei Lim, Dr. Si Ching Lim**. *Challenges in the Management of an Elderly Patient with Chronic Schizophrenia and Dementia. Archives of Neurology and Neuro Disorders. 2018; 1(1): 17-20.*