Review Article

LATE-ONSET BIPOLAR AFFECTIVE DISORDER : A REVIEW

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Abstract

The diagnosis of bipolar affective disorder in the geriatric population is uncommon. However it comprises a significant health care utilization and cost requirements, which are expected to increase in the near future with the projected increase in the geriatric population. The authors review the literature pertaining to late-onset BPAD and discuss the epidemiology, psychopathology, neuropathology, differential diagnosis, evaluation, treatment, and outcomes.

Key Words:

Bipolar affective disorder, geriatric, late onset, mania Introduction

The occurrence of bipolar affective disorder (BPD) in geriatric population is uncommon. However, it comprises a significant health care utilization, which is expected to increase in the near future with the projected increase in the geriatric population. Clayton (1983) estimates that 90% of bipolar patients become ill by age 50. This indicates that an estimated 10% of population may develop bipolar disorder for the first time after the age 50, a substantially large number considering that the number of people aged 65 and over is on the increase (Blazer, 1980).

This article will review the literature pertaining to late-onset BPD and discuss the epidemiology, psychopathology, neuropathology, differential diagnosis, latency, course and management.

Epidemiology

There are many caveats in the epidemiological studies of late-onset BPD. To begin with, the cut-off age of late-onset BPD is not defined. Some studies consider 30 as the cut off age (Ghadirian, 1986) where as other studies used a higher cut off age as 40 (Rosen & Rosenthal, 1983). Yassa et al (1988) have proposed a higher cut off age of 50 for the diagnosis of late-onset BPD. Another difficulty is that many of the studies were conducted before the dichotomy into unipolar and bipolar disorders. As a result many studies used Kraepelinian classification alluded to in the introduction. Thus these studies are not helpful in assessing the prevalence of BPD arising later in life. Third difficulty is that many of the geriatric BPD studies do not differentiate between first admissions and readmissions with earlier onset. This makes it difficult to estimate the prevalence of BPD in older patients. Fourth difficulty is related to the type of set-up in which the study was conducted that is whether it was a private hospital, public hospital or a nursing home. A final difficulty is that many of these studies were retrospective.

Mania affects approximately 1% of general population (Lyketsos et al, 1995), however there are no large scale community surveys of geriatric populations that would allow the development of age-adjusted incidence and prevalence rates for late-onset BPD. Moreover, there is much controversy whether the incidence of BPD increases, decreases or remains same as people age. Between 5% and 19% of all geriatric patients presenting

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for the treatment of an affective disorder are manic (George, 1996; Mirchandini et al, 1993; Young & Klerman, 1992; Young, 1992). It has been estimated that 50% of new-onset manic episodes occur in patients over 50 years of age (Kaplan & Sadock, 1985). Studies have found frequency of BPD in elderly patients to range from 0-0.1%(Young, 1997; Berrios, 1991) with prevalence rate from 0.1 to 0.4% in patients over 65 (Shulman et al, 1992; Snowdon, 1991). Epidemiological Catchment Area (ECA) study found that 9.7% of all nursing home patients had BPD (Greenwald et al, 1992).

Age at onset

Currently there are no strict criteria for the categories of early-onset and late-onset BPD. In general early-onset refers to BPD that begins in patients' 20s or 30s and late-onset to BPD that begins after the age 50. Some consider BPD that begins after age the 65 as very late onset (Van Gerpen et al, 1999). One study of manic patients aged 65 or older found that 25% had their first manic attack after the age of 65 (Stone, 1989). Broadhead & Jacoby's sentinel study (1990) found no difference between early-onset and late-onset BPD with regard to the course, severity or treatment.

Sex differences

Geriatric BPD occurs in a 2 to 1 ratio of women to men (Liptizin, 1992), perhaps because there are more women in the geriatric population. In a study of BPD patients 50 years and older, men had mean age-at onset of 53.2 years, which was significantly earlier than the female average of 61.9 years (Shulman & Post, 1980). However, age-at-onset studies disagree regarding which sex may develop BPD earlier.

Family history of mood disorders

Patients with late-onset BPD have a lower incidence of family history of mood disorders (Stone, 1989; Shulman & Post, 1980; Charron et al, 1991) compared to early-onset cases. Various studies have reported that between 26.5% and 48% of elderly BPD patients have a family history of mood disorder (Snowdon, 1991; Charron et al, 1991; Stone, 1989). Interestingly, a number of studies have found that late-onset BPD patients with neurological disorders have 9-33% family history of mood disorders, which is lower than the rate of those without history of neurological disorders (Shulman, 1992; Snowdon, 1991; Mc Donald et al, 1991; Stone et al, 1989). It is reported that BPD patients over the age of 65 with family history of mood disorder had significantly earlier age-at-onset than those without family history (Stone, 1989).

Symptom differences

There has been considerable research, debate, and controversy surrounding the question of whether late-onset BPD differs significantly with regard to symptom profile from early-onset patients. Depressive episodes in patients with BPD have received little investigation in the elderly. Broadhead & Jacoby (1990) noted no difference in depressive features in late-onset compared with early-onset BPD. Traditionally, geriatric mania has been considered to have an atypical presentation with increased confusion, paranoia, dysphoria or irritability and negativity (George, 1996; Khouzam et al, 1994; Liptizin, 1992; Yassa et al, 1988). Paranoid delusions that are mood-incongruent (persecutory/referential), irritability and anger (replace the hyperactivity and expansiveness of younger patients), circumstantiality (replace the flight of younger patients), agitation and

negativity and depressive thoughts interspersed with manic symptoms (probably known as miserable mania or mixed mania or agitated depression) are more frequently reported in late-onset BPD (Yassa et al, 1988). Recent studies however have begun to revise this view.

Cognitive dysfunction is more common in older BPD patients versus same-age control subjects (Young, 1997; Young et al, 1992; Young, 1992) but is equally common in early-onset and late-onset BPD patients (Mirchandini et al, 1993; Young, 1992; Young et al 1992). Cognitive dysfunction may be partially or totally reversible with treatment. Recent studies do not support the view that late-onset BPD is associated with higher rates of dementia.

Association with cerebral organic disorders/Neurological comorbidity Several studies have documented that 17-43% of geriatric BPD patients have heterogeneous demonstrable cerebral disorders including stroke, traumatic brain injury and space-occupying lesions (Young, 1992; Shulman et al, 1992; Brodhead & Jacoby, 1990; Stone, 1989, Cumming & Mendez, 1984; Shulman & Post, 1980). Of further interest is a study finding that first episode manic patients over 60 years of age had close temporal association between onset of BPD and evidence of cerebral organic disorder (Broadhead & Jacoby, 1990). Another study of new onset manic patients over the age of 60 found them more likely to have cerebral organic disorders than patients with early onset and multiple episodes of mania (Tohen et al, 1984).

Pathology, Neuroradiology

Much of the information regarding the neuroradiological changes in the brain of late-onset BPD patients comes from the literature on secondary mania. It has been proved that cerebral insult to areas like basal ganglia, thalamic nuclei, midbrain nuclei and limbic areas in the orbito-frontal and baso-temporal cortices that control emotions and neurovegetative functions can result in mania (Wilson & Mc Laughlin, 1990; Cumming & Mendez, 1984). Mania is also highly associated with right hemispheric lesions predominantly in the right baso-temporal or the right thalamic or caudate nuclei (Robinson et al, 1988; Cumming & Mendez, 1984). A retrospective analysis of late-onset mania showed that 65% developed BPD after a silent cerebral infarction (Fujikawa et al, 1995). One MRI study of late-onset manic patients has found subcortical hyperintensities especially in the middle third of brain parenchyma (Young, 1992). Cerebral atrophy has been described in late-onset BPD patients; however it also occurs in normal geriatric population (Charron et al, 1991). Ventricular enlargement is not reported in late-onset BPD (Broadhead & Jacoby, 1990).

Neurotransmitter dysfunction

Neurotransmitters have been implicated in the pathophysiology of late-onset BPD. Ascending pathways that travel through the midbrain and connect the limbic region, basal ganglia and cerebral hemispheres may increase their functional output, causing an excess of norepinephrine, dopamine and serotonin (Mc Daniel et al, 1996) and thus cause mania. Deficiency of GABA-aminobutyric acid may also play a role in causing mania (Mc Daniel et al, 1996) given that mood stabilizers enhance the transmission of GABA.

It has been suggested that the right hemisphere but not the left, may be able to increase the serotonin-receptor binding after injury, there by cor-

recting the damage inflicted and possibly in the case of secondary mania overcorrecting it (Starkstein et al, 1990).

Diagnosis and differential diagnosis

Current nosologic system do not separately classify BPD with late-onset. It is a heterogeneous group of patients with a substantial proportion having major depression that can change polarity with increased age, Type V-BPD (personal history of only depression but a family history of BPD) or Type IV-BPD (mood disorders resulting from a general medical condition or substance induced) and late-onset pure BPD. Manic states occurring for the first time in late life generally meet DSM IV Criteria for bipolar I disorder.

Because of the frequent atypical presentation of late-onset BPD, the condition may be recognized with great difficulty. Elderly patients are poor historians; often key family members have left home or died or past events may be distorted. All these may lead to difficulties in obtaining a full historical background in such patients (Spar et al, 1979). With typical symptoms the diagnosis is relatively easy to make. However, with atypical presentation, several conditions should be differentiated from late-onset-BPD (Yassa et al, 1988).

Agitated depression- Here the mood is one of depression accompanied by the all vegetative symptoms and signs of depression. Delusions of self-depreciation or guilt may be present.

Schizophrenia- Differentiating this condition from late-onset BPD may be difficult sometime. According to Clayton (1986) exhibiting a triad of manic mood, rapid or pressured speech and hyperactivity should be considered as manic regardless of the presence of other symptoms.

Paranoid disorder- It may be difficult to differentiate this condition from the manic patient with mood-incongruent psychotic features. However, manic will have other symptoms such as flight of ideas, grandiose delusions and overspending side by side with mood-incongruent psychotic features.

Dementia- Manic pseudodementia may be misdiagnosed for Alzheimer's disease when they present in combination of manic and dementia symptoms. The latter usually clears with treatment, in contrast to the progressive deterioration noted in organic dementias.

Tumors in different areas of brain can cause manic/depressive symptoms. Localising signs as well as appropriate tests, such as EEG and CT will help in the differential diagnosis of these space-occupying lesions.

Different drugs can precipitate manic/depressive attacks, which at times may need special tests to detect the offending agent. Infections like neurosyphilis, HIV have also been reported to present as mania/depression.

Thus, a multitude of conditions can cause BPD in elderly patients. Systematic investigations should first be carried out in such cases before the diagnosis of a primary affective disorder. The tests include thyroid function tests, B12 and folate levels, serologic tests, renal function tests, skull radiograph, EEG and CT scans.

Latency and course

Studies of late-onset mania over the age of 65 have found 14.9-16 years latency between first depressive episode and first manic episode (Shulman et al, 1992; Snowdon, 1991; Stone, 1989). One study (Shulman, 1992) of late-onset

BPD has found shorter remission period between the index episode and first rehospitalisation. Another study when compared the course of BPD in elderly versus early-onset patients found no difference in the length of episode, length of hospitalization or in the length of various stages associated with mania (onset of mania to hospitalization, hospitalization to resolution of mania or resolution of mania to discharge from the hospital) (Broadhead & Jacoby, 1990). Same group have found greater proportion of elderly manic patients suffered depressive episode after the resolution of mania.

Some studies have reported increased vulnerability to relapse and decreased inter-episode interval in late-onset BPD (Swift, 1997; Angst et al, 1973). Dhingra & Rabins (1991), however, reported no difference in relapse over 5 to 7 years between early-onset and late-onset geriatric manic patients. Shulman & Tohen (1994) have reported that a "Type VI" course in geriatric mania (i.e. unipolar mania) is less common late- compared with early-onset BPD. The study by Dhingra & Rabins (1991) did not detect difference in mortality rates in late-onset versus same-age-early-onset BPD patients. Whether greater mortality rate is associated with late-onset cases needs further study because in such cases there may be increased medical/neurological comorbidity. Similarly the risk of dementia is not high in late-onset BPD compared to early-onset BPD (Dhingra & Rabins, 1991).

Management

Management of late-onset BPD follows the same principle as that for young adults (Young, 1997). However these patients should be examined for drugs, medical or neurological diseases that can predispose or precipitate BPD.

Lithium

This is the most investigated drug with established efficacy in uncomplicated BPD (Shulman & Post, 1980). Because of the age associated decline in renal clearance leading to higher plasma level, introduce the drug slowly with a dose of 150mg per day. Usually half of the adult dose is sufficient for the elderly with a plasma level of 0.3-0.6mEq per liter. Plasma half life of lithium is 24-36 hours in patients who are in their 70s. There for steady state plasma level is achieved 5 or more days after the stabilization of daily dosage. The onset of lithium action is slow in elderly requiring several days or weeks.

The adverse effects of lithium are pronounced in geriatric patients. Even at a non-toxic level they can develop tremor or myoclonus or delirium (Murray et al, 1983; Smith & Helms, 1982). It can also produce or worsen pre-existing parkinsonism. It may worsen cognitive functions especially in patients with dementia. It can cause sinoatrial block either alone or in combination with digitalis or (blockers. Combination with psychotropic drugs increases the risk of delirium in elderly. Salt restriction, thiazide diuretics may raise plasma lithium concentration resulting in toxicity.

Anticonvulsants

Anticonvulsants such as sodium valproate (Bowden, 1996), carbamazepine (Calabrese & Bowden, 2000) and oxcarbazepine are effective in both lithium responders and non-responders. Sporadic reports are available regarding the efficacy of gabapentin, lamotrigine and topiramate in late-onset BPD (Chengappa & Levin, 2000).

They are more effective in rapid cyclers, dysphoric mania and mania with neurological dysfunction. Although there is limited research on the use of these drugs in elderly population, they should be considered for those who are at high risk for lithium toxicity or poor responders to lithium.

Drowsiness and sedation are frequent dose dependent adverse effects with both sodium valproate and carbamazepine to which patients will be habituated with continued use. Slow introduction of the drug can reduce this problem. In elderly, valproate can cause gastrointestinal distress or ataxia; with carbamazepine are confusion and ataxia. The free fraction of valproic acid can increase with age (Young, 1995). This is related to decreased albumin concentration. The clinical significance of this not known. Clinicians must be alert to the fact valproate can inhibit hepatic enzymes, decreasing the metabolism of some concomitantly administered drugs. Approximately 0.4% of those receiving valproate and 2% of those receiving carbamazepine develop leucopoenia with a white cell count below 4000/mm3, mostly within 2 weeks after starting the drugs. For this reason periodic monitoring of white cell count is indicated, especially during the early phase of treatment.

Other drugs

Agitated elderly manic patients may be treated with lorazepam because it undergoes only phase II metabolism (glucoronidation), a process minimally affected by ageing. Other alternative is haloperidol 0.5-5mg daily. These drugs should be used only during the early phase and should be withdrawn when the agitation is controlled and therapeutic level of mood stabilizer is achieved.

Newer atypical antipsychotic drugs such as risperidone, olanzapine and clozapine either alone or in combination with mood stabilizers are also effective in late-onset BPD with lesser side effects (Chengappa & Levin, 2000).

Electro convulsive therapy

Controlled studies have demonstrated that ECT is an established treatment in both geriatric depression and mania because of its efficacy, rapid onset of action and safety. ECT may be chosen in patients with mood syndromes who cannot wait for the gradual effect of mood stabilizer, those with concurrent medical problems, those with contraindication to drug therapy and in those at risk for suicide, debilitation, dehydration and electrolyte disturbances (Alexopoulose, 1995).

Geriatric patients require more time to recover their memory function especially after bilateral ECT. Some time they develop prolonged confusion after ECT and falls have been reported. Compared to younger adults, elderly patients are more prone to develop cardiovascular events, occurring in the context of ECT or within few hours of treatment. However with adequate medical evaluation and monitoring after ECT and appropriate intervention, ECT has a benign outcome. Anecdotal literature suggest that ECT is an effective continuation or maintenance antidepressant treatment but high recurrence-relapse rate in ECT responders are reported who are previously medication resistant. There are reports that ECT was used uneventfully in patients over 100 years and in elderly with aortic aneurisms, cardiac pacemakers, myocardial infarction, strokes, severe hypertension, arrhythmias and in patients requiring anticoagulant therapy (Alexopoulose, 1989).

Continuation and maintenance therapy

The efficacy and toxicity of lithium and anticonvulsants in continuation and maintenance treatment have been described specifically in elderly BPD patients. One recent negative report (Abu-Saleh & Coppen, 1983) concerning affective morbidity and age at initiation of lithium prophylaxis reported poor efficacy on long-term treatment. Murray et al (1983) noted some increase in manic psychopathology, but not hospitalizations, at equivalent, moderate lithium levels in older compared with younger patients in a mixed-age sample that was followed prospectively.

Conclusion

BPD in old age, though not as common in younger patients, may constitute 5% of admission to a psychiatric unit. Because they may present with atypical symptoms diagnosis may be difficult and may need skilful examination of the patient and family members supplemented with investigations. The prognosis and treatment of BPD follow somewhat similar principles to those of younger patients. Time immemorial lithium stands to be the treatment of choice in this condition. Introduction of newer drugs with lesser side-effect profile will definitely help in the provision of better quality of life in this patient group.

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