Late - onset Bipolar Affective Disorde

A Review

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Abstract:
The diagnosis of bipolar affective disorder (BPD) in the geriatric population is uncommon. However it comprises a significant health care utilization and cost requirements, which are expected to increase in the geriatric population. The authors review the literature pertaining to late onset BPD 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 geriatric population. Clayton (1983) estimates that 90% of bipolar patients become ill by the age 50. This indicates that an estimated 10 % of population may develop bipolar disorder for the first time after the age 50, a substantially l number considering that the number of pe aged 65 and over is on the increase (Bl 1980).

This article will review the literature pertainin late - onset BPD and discuss the epidemiol psychopathology, neuropathology, differen diagnosis, latency, course and management.

EPIDEMIOLOGY

There are many caveats in the epidemiologi studies of late - onset BPD. To begin with, t cut off age of late - onset BPD is not define Some studies consider 30 as the cut off a (Ghadirian, 1986) where as other studies used higher cut off age as 40 (Rosen & Rosenth 1983). Yassa et al (1988) have proposed higher cut off age of 50 for the diagnosis of lat onset BPD. Another difficulty is that many of th studies were conducted before the dichotom into unipolar and bipolar disorders. As a resul

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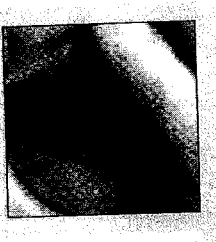
studies used Kraepelinian classification d to in the introduction. Thus these s are not helpful in assessing the ence of BPD arising later in life. Third ity is that many of the geriatric studies do not differentiate een the first admissions and missions with earlier onset. This s it difficult to estimate the lence of BPD in older patients. h difficulty is related to the type of up in which the study was ucted that is whether it was a te hospital, public hospital or a ng home. A final difficulty is that many of e studies were not retrospective.



ia affects approximately 1% of general ulation (Lyketsos et al, 1995), however e are no large scale community surveys of atric populations that would allow the elopment of age -adjusted incidence and valence rates for late - onset BPD. reover, there is much controversy whether incidence of BPD increases, decreases or mains the same as people age. Between 5% d 19% of all geriatric patients presenting for e treatment of an affective disorder are manic eorge, 1996; Mirchandani et al, 1993; Young Klerman, 1992; Young 1992). It has been timated that 50% of new - onset manic isodes occur in patients over 50 years of age aplan & Sadock, 1985). Studies have found equency of BPD in elderly patients to range om 0 - 0.1 % (Young, 1997; Berrios, 1991) ith prevalence rate from 0.1 to 0.4% in atients over 65 (Shulman et al, 1992; nowdon, 1991). Epidemiological Catchment rea (ECA) study found that 9.7% of all nursing ome patients had BPD (Greenwald et al, 992).

AGE AT ONSET

Currently there are no strict criteria for the categories of early - onset and late - onset BPD. n general early - onset refers to BPD that begins in patients 20s or 30s [illegible] BPD that begins after the age [illegible] consider BPD that begins after age 65 [illegible] 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

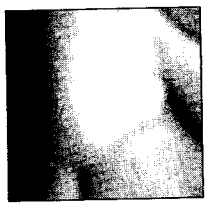
Geriatric BPD occurs in a 2 to 1 ratio of women to men (Liptizin, 1992), perhaps because there are more women in the geriatric population. In a study of BPD patients 50 years and older, men had mean age -at- onset of 53.2 years, which was significantly earlier than the female average of 61.9 years (Shulman & 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 lo incidence of family history of mood disord (Stone, 1989; Shulman & Post, 1980; Charro al, 1991) compared to early - onset ca Various studies have reported that bet 26.5% and 48% of elderly BPD patients h family history of mood disorder (Snowdon, Charron et al, 1991; Stone, 1989). Interes a number of studies have found that late - BPD patients with [illegible] 33% family history of mood [illegible] lower [illegible] or [illegible] neurological [illegible] (Shulman, 1992; Snowdon, [illegible] Mc Donald et al, 1991; Stone et al, 1989) 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 a 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 hyperactivity and expensiveness 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 review.



Cognitive dysfunction is more common in older BPD patients versus the same - age control subjects (Young, 1997; Young et al, 1992; Young, 1992) but is equally common in early - onset and late - onset BPD patients (Mirchandani 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 heterogenous demonstrable cerebral disorders including stroke, traumatic brain injury and space - occupying lesions (Young, 1992; Shulman et al 1992; Broadhead & Jacoby, 1990, stone, 198 Cumming & Mendez, 1984; Shulman Post, 1980). Of further interest is study finding that first episode manic patients over 60 years of age had clos temporal association between onset o BPD and evidence of cerebral organi disorder (Broadhead & Jacoby, 1990) Another study of new onset manic patients over the age 60 found that the are more likely to have cerebral organic disorders than patients with early onset and multiple episodes of mania.

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 brain parenchyma (Young, 1992). Cerbral atrophy has been described in the late - onset BPD patients, however it also occurs in normal geriatric population (Charon et al, 1991). Ventricular enlargement is not reported in late - onset BPD (Broadhead & Jacoby, 1990).

Neurotransmitter dysfunction

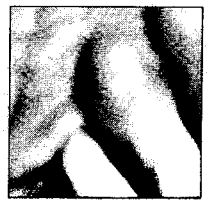
Neurotransmitters have been implicated in the pathophysiology of late - onset BPD . Ascending

ye that travel through the midbrain connect
blc region, basal ganglia and cerebral
heres may increase their functional output,
an excess of norepinephrine, dopamine
rotonin (Mc Daniel et al, 1996) and thus
mania. Deficiency of GABA may also play
In causing mania (Mc Daniel et al, 1996)
that mood stabilizers enhance the
lssion of GABA.

been suggested that the right hemisphere
ot the left, may be able to increase the
onin - receptor binding after injury, thereby
cting the damage inflicted and possibly in
ase of secondary mania overcorrecting it
kstein et al, 1990).

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

ent nosologic system do not separately
sify BPD with late - onset. It is a
rogenous group of patients with a
stantial proportion having major
ression that can change polarity with
eased age, type V BPD (personal
tory of only depression but a family
tory of BPD) or Type IV - BPD (mood
orders resulting from general medical
ndition or substance induced) and late
onset pure BPD. Manic states
curring for the first time in late life generally
eet DSM IV criteria for bipolar I disorder.



ecause of the frequent atypical presentation of
te - onset BPD, the condition may be
cognized with great difficulty. Elderly patients
re poor historians; often key family members
ave 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 the typical presentation,
several conditions should be differentiated form
late - onset BPD (Yassa et al, 1988).

Agitated depression - Here the mood is one of depression accompanied by all the vegetative symptoms and signs of depression. Delusions of self -depression 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, a 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. Localizing signs as well as appropriate tests, such as EEG and CT will help in the differential diagnosis of these space - occupying lesions.

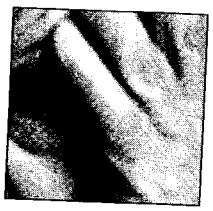
Different drugs can precipitate manic / depressive attacks, which at times may need special tests to detect the offending agent. Infections like neurosyphilis, HIV have also been reported to present as mania / depression.

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 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 the episode, length of rehospitilisation 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 found greater proportion of elderly manic patients suffered depressive episodes 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 precipit BPD.

Lithium

This is one of the most investigated drug wi established efficacy in uncomplicated BP (Shulman & Post, 1980). Because of the ag associated decline in renal clearance leading t higher plasma level, introduce the drug slowl with a dose of 150 mg per day. Usually half o the adult dose is sufficient for the elderly with plasma level of 0.3 - 0.6 mEq per liter. Plasm half life of lithium is 24 - 36 hours in patients wh are in their 70s. Therefore steady state plasm levels is achieved in 5 or more days after stabilization of daily dosage. The onse of action of lithium 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 function especially in patients with dementia. It can cause sinoatrial block either alone or in combination with digitalis or β 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 (Clabrese & 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

ose who are at high risk for lithium toxicity or responders to lithium.

siness and sedation are frequent dose ndent adverse effects of both sodium oate and carbamazepine to which patients e habituated with continued use. Slow duction of the drug can reduce the lem. In elderly, carbamazepine can e confusion and ataxia. The free lon of valproic acid can increase age (Young, 1995). This is related decreased albumin concentration. clinical significance of this is not wn. Clinicians must be alert to the t that valproate can inhibit hepatic zymes, decreasing the metabolism of me concomitantly administered drugs.

her drugs

ltated elderly manic patients may be treated h lorazepam because it undergoes only ase II metabolism (glucuronidation), a ocess minimally affected by ageing. Other ternative is haloperidol 0.5 - 5 mg daily. These ugs should be used only during the early hase and should be withdrawn when the gitation is controlled and therapeutic level of ood stabilizer is achieved. Newer atypical ntipsychotic drugs such as risperidone, lanzapine and clozapine either alone or in ombination with mood stabilizers also effective n late - onset BPD with lesser side effects Chengappa & Levin, 2000).

ELECTROCONVULSIVE THERAPY

Controlled studies have demonstrated that ECT s an established treatment in both geriatric lepression and mania because of its efficacy, apid onset of action and safety. ECT may be hosen in patients with mood syndromes who annot wait for the gradual effect of mood tabilizer, those with concurrent medical roblems, those with contraindication to drug herapy 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, arrythmias and in patients requiring anticoagulant therapy (Alexopoulose, 1989).

CONTINUATION AND MAINTENANCE THERAPY

The efficacy and toxicity of lithium and anticonvulsant in continuation and maintenance treatment have been described specifically in 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 (1982) 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 patents, may constitute 5% of admission

to 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 provision of better quality of life in this patient group.

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