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Original Research Article

A cross-sectional study to find out the prevalence, pattern, risk factors, comorbidities and severity of vascular depression in patients attending psychiatric outpatient department in a tertiary care centre

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ABSTRACT

Background: Depression was declared as the second major cause of disability adjusted life years (DALY) in 2020 and the economic burden experienced by those with depression is next to coronary artery disease. Vascular depression has late age at onset 60 years or older, nonpsychotic subtype, with no family history of mental disorders, presenting with loss of pleasure and functional disability. Depression is accompanied by cerebrovascular lesions as determined by MRI and not accompanied by neurological signs. This has a spiral correlation with various comorbidities leading to diagnostic enigma at one hand and worsened prognosis on the other. This emphasized the early diagnosis and treatment to improve the quality of life.
Methods: Patients with age more than 60 years, of either sex, with a mini GDS score ≥5, having the first episode of depression and who gave consent were included.
Results: Hypertension thus emerged as a significant risk factor and was positively associated with increased severity of depression.
Conclusions: Subclinical cognitive impairment and mild cognitive impairment (MCI) may precede years before the onset of vascular depression. Prompt detection and management of these entities and associated risk factors could prevent or postpone impending vascular depression.

Keywords: Depression, Vascular depression, Mini GDS score, Cognitive impairment, MCI

INTRODUCTION

As a common cold of mental illness, depression is currently the 4th most prevalent disorder. It is a matter of crucial concern that various neuropsychiatric signs and symptoms of depression like memory impairment, psychomotor retardation, apathy and loss of interest in work and surroundings are merely attributed to ageing, and thus ignoring and losing precious time for the management of the reversible disorder.

Late age onset depression is the second most common cause of DALYs loss by the year 2020.[1] Co-morbidity of geriatric depression with various physical disorders and its spiral interrelationship with regard to the course and outcome of each disease process has always been a challenging enigma. The presence of depression not only results in amplification of various somatic symptoms but also deteriorates the overall prognosis. It may at one hand increase the progression of the disease due to various internal biological alterations and on the other hand, be a hurdle for treatment strategies viz poor drug compliance,

pathological lifestyle, poor adherence to dietary regimens, vulnerability towards addiction.

Geriatric depression is frequently encountered with concomitant physical disorders of diverse nature, which invariably mask the psychiatric manifestations per se, leading to diagnostic and management dilemma. It is eventually missed and remains unmanaged as a separate entity. Depression may be found in patients of cardiovascular disorders, diabetes mellitus, renal failure or Parkinson's disease. It is often visible in post myocardial infarction state, CCF or in COPD patients.[2] In females and developing countries, unipolar major depression is projected to become the leading cause of disease burden.

Vascular depression constitutes a subgroup of late age onset depression, usually associated with neuroimaging abnormalities in the basal ganglia and white matter on MRI.[3] The cause of the structural brain changes is thought to be sclerosis in the small arterioles.[4] These end-artery vessels may be particularly susceptible to pulse-wave changes caused by arterial rigidity or hypertension.

The clinical characteristics of this depression includes more pronounced psychomotor retardation, anhedonia, greater overall cognitive impairment and physical disability, fewer feelings of guilt and greater lack of insight. Furthermore, non-psychotic symptoms prevailed whereas a family history of mental illness and especially affective disorder was less common.[5,6] The most robust findings from structural imaging studies of mood disorders has been the abnormally high prevalence of MR signal hyperintensities in the deep and periventricular white matter in elderly major depressive subjects and are predominantly seen in geriatric patients experiencing their first major depressive episode (MDE) of unipolar depression.[7] There are hyperintense foci of the MR signal, in the deep white matter, periventricular and basal ganglia region. These lesions are often associated with cerebrovascular disease.[8] Age, prior history of brain ischemia and hypertension are the most significant predictors of the incidence and severity of such lesions. Brainstem hyper-intensities are also common and are primarily seen in the pons. Although the correlates of such findings are occasionally evident in CT scans too; referred to as leuko-araiosis, their anatomical extent and frequency are best visualized in MR images. In MRI, white matter hyper intensities (WMH) were initially referred to as unidentified bright objects and later as leukoencephalopathy.[9,10] These findings are now descriptively classified as either WMH or lacunae and are graded by their size and location in T2 weighted MR scans. The periventricular hyper intensities (PVH) grading was mentioned by Fazekas et al in 1987 as 0-absent, 1-cap-pencil thin lining, 2-smooth halo, 3-irregular PVH extending into the deep white matter and deep white matter hyperintensities (DWMH); while by Sullivan in 1990, as 0-Absent, 1-punctate foci, 2-beginning confluence of foci, 3-large confluent areas lacunae, which reflect areas where infarcted tissue has been replaced by cerebrospinal fluid, are most commonly evident within the gray matter as irregularly shaped lesions >5 mm in size that appear hyper intense on T2-weighted images and hypo intense on Tl weighted images.[11,12] In contrast, smaller punctate foci of signal hyper intensity in T2 images generally reflect dilated perivascular spaces (Virchow-Robin spaces) that may be associated with perivascular gliosis presumably due to tissue loss around small blood vessels. The pathogenesis of these lesions is not well known, though deposition of lipids within arteriolar walls (lipohyalinosis) has been linked to hyper intensities. The location of the lesions was secondary to arterial occlusion of perforating vessels. The penetrating arteries affected by this process include lenticulostriate, thalamoperforate and medullary artery system. The location of the lacunae is primarily in the water-shed zone of the distribution of the perforating vessels.[13] These vessels are not only susceptible to hypertension but are also sensitive to hypotension. Hypoperfusion secondary to hypotension often results in infarction in these watershed areas.[14] Hypertension in its severe form can lead to infarction and chronic ischemia secondary to narrowing of the arterioles. This can result in loss of vasoregulation and subsequent cerebral oedema, perivascular demyelination, myelin pallor and gliosis. Steffens et al in the cardiovascular health study (CHS), have noticed association of depression with small lesions in basal ganglia, large cortical white matter lesions and severe subcortical white matter hyper intensities, suggesting that cerebrovascular disease at baseline is related to depressive symptoms over time.[15] Studies of MRI neuropathological correlates reveal that areas of increased signal are associated with a number of pathophysiological processes related to cerebrovascular disease, including arteriosclerosis, perivascular demyelination, dilated perivascular spaces, vascular ectasia, ischemia and incomplete infarction (partial myelin loss, axonal degeneration, gliosis, macrophage infiltration), complete infarction and necrosis.[7] Krishnan and associates in 19976 proposed the term subcortical ischemic vascular depression (SIVD) as a more accurate representation of the disease process. In one of his studies, out of 139 depressed elderly subjects, 75 (54.0%) met neuroimaging criteria for SIVD.

The objective of our study was to find out the prevalence, pattern, risk factors, comorbidities and severity of vascular depression in patients attending our psychiatric outpatient department.

METHODS

This cross-sectional study was conducted at both inpatient and outpatient sections of psychiatric, medical and geriatric OPD of RD Gardi Medical College, Ujjain (MP) between June 2018 to September 2019. Patients above 60 years of age were screened for underlying depression. The screening for depression was done by mini geriatric depressive scale (GDS) consisting of 15

questions to be answered in affirmative or negative and a minimum score of ≥5 as a cut-off point was considered for study.[16]

Patients with age more than 60 years, of either sex, with a mini GDS score ≥5, having the first episode of depression, and who gave the consent were included.

Exclusion criteria for the study was patients not fulfilling the inclusion criteria, patients who did not give consent, and all follow up patients.

A total of 116 patients were selected and were then evaluated with the help of semi-structured proforma consisting of detailed sociodemographic and clinical variables, after approval from the ethical committee. Thorough general and systemic examination was rendered and expert opinion regarding physical morbidities was taken from various specialties whenever indicated. The patients were subjected to battery of investigations which included completed blood picture, ESR, fasting and postprandial blood glucose, liver function tests, renal function test, lipid profile, urine routine and microscopic, 12-lead echocardiography, optic fundus, X-ray chest and CT scan and MRI, wherever possible.

The quantification of depression was then made on the basis of scores on Montberry Ausberg depression rating scores (MADRS). The cognitive dysfunction was assessed using mini mental status examination (MMSE).[16] The severity of the cognitive impairment was graded according to scores as minimal (>24), mild (20-24) and moderate to severe (<20). Various sociodemographic and clinical variables of late age onset depression were compared with control group and the observations were subjected to statistical analysis using SPSS software.

RESULTS

There was higher percentage of late age onset depressive symptoms (age of >60 onset years) (56.31%) as compared to control, with most being in-patients (66.11%) as compared to out-patients (51.02%).

Table 1: Addiction.

Addiction	LOD (n=116)*		Control (n=90)*	
	No.	%	No.	%
Tobacco	61	52.59	30	33.33
Smoking	38	32.76	32	35.56
Alcohol	13	11.21	26	28.89
Others	7	6.03	0	0.00
No addiction	32	27.59	28	31.11

* Patients had multiple addictions. χ^2=18.403, p=0.001, significant.

The younger old-age group (60-69 years) outnumbered the old late onset depressives (81.9% v/s 25.6%). The male female ratio was around 62:54 with mean age of male patients as 66.13±6 and females as 66.11±6. The majority of the LOD patients (59.50%) were married; and rest (40.5%) were widow/widower and most belonged to the lower socioeconomic strata (65.52%).

Table 2: Psychological stressors.

Nature of Stress	LOD (n=116)		Control (n=90)	
	No.	%	No.	%
Family history	0	0.00	0	0.00
Family relation	39	33.62	10	11.11
Loss of spouse	46	39.66	34	37.78
Adverse life events	18	15.52	5	5.56
No stressor	13	11.21	41	45.56

χ^2=38.15, p≤0.0001, significant.

Table 3: Clinical variables.

Comorbid Disorders	LOD (n=116)*		Control (n=90)*	
	No.	%	No.	%
HTN	77	66.38	63	70.0
DM	40	34.48	71	78.89
Cataract	33	27.59	60	66.67
B.P.H.	26	22.41	56	62.22
Arthritis	34	29.31	58	64.44
COPD	13	11.21	30	33.33
CAD	18	15.52	43	47.78
Anaemia	15	12.93	60	66.67
Parkinson's disease	2	1.72	1	1.11
CVA	14	12.07	35	38.89
Deafness	13	11.21	37	41.11
Hypothyroidism	2	1.72	3	3.33
PTB	2	1.72	6	6.67
Cancer	1	0.86	0	0.00
Renal Failure	1	0.86	0	0.00
Cirrhosis	1	0.86	0	0.00
Others	0	0.00	0	0.00

* Patients had multiple co-morbid disorders. χ^2=43.146, p=0.0001, significant.

Only 4.31% belonged to the higher group. The majority of depressive patients belonged to joint family (69.82%). The bulk of patients belonged to lower education group viz illiterate 56.9% and primary 20.69%. Most of the patients showed addiction dependency (72.41%) dominated by tobacco (52.59%), smoking (32.76%) and alcohol (11.21%) as shown in Table 1.

Most of the patients suffered from stressful events in their life time (88.9%), of which loss of spouse (39.66%) seems to be leading stressor followed by intrafamilial conflict (33.62%) (Table 2).

No genetic predisposition was found in the study. The associated co morbidities, in decreasing order were hypertension (66.38%), diabetes mellitus (34.48%), arthritis (29.31%), cataract (27.59%), BPH (22.41%), CAD (15.52%), anaemia (12.93%), cerebrovascular accident (12.07%), COPD (11.21%), hearing impairment (11.21%) as per Table 3.

Table 4: Depressive symptoms according to age of onset.

Depressive items	LOD (n=116)*		Control (n=90)*	
	No.	%	No.	%
Sleep disturbances	92	79.31	13	14.44
Retardation	52	44.83	0	0.0
Somatic complaints	61	52.59	30	33.33
Lack of interest	46	39.7	-	0.00
Loss of weight	40	34.48	02	2.22
Low mood	23	19.83	01	1.11
Anxiety features	37	31.90	27	30.00
Lack of Insight	32	27.59	-	0.00
Guilt felling	11	9.48	-	0.00
Suicidal Ideation	6	5.17	-	0.00
Agitation	28	24.14	5	5.56

* Patients had multiple complaints. χ^2=90.20, p≤0.0001, significant.

Table 5: Cognitive impairment.

Cognitive Function	LOD (n=116)		Control (n=90)	
	No.	%	No.	%
Orientation	25	21.55	15	16.67
Memory	78	67.2	14	15.56
Calculation	93	80.2	24	26.67
Reading and Comprehension	87	75.0	37	41.11

χ^2=10.941, p=0.012, significant.

Table 6: Severity of depression and cardiovascular risk factors.

Risk Factors*	MMSE Scoring					
	Minimal (>24)		Mild (20-24)		Mod.-Sev. (<20)	
	No.	%	No.	%	No.	%
HTN (77)	12	15.6	14	18.2	51	66.23
D.M. (40)	22	55.0	08	20.0	10	25.0
Dys-Lipidemia (37)	10	27.02	05	13.51	22	59.60

* Patients had multiple co-morbid disorders. χ^2=23.43, p=0.0001, Significant.

Most commonly observed symptoms in the present group were sleep disturbances (79.31%), somatic complaints (52.59%), retardation and apathy (44.83%), lack of interest (39.07%), loss of weight (44.48%), anxiety features (31.90%), agitation (24.41%), mood disturbance (19.83%), lack of insight (18.97%) (Table 4). Suicidal ideation was found in (5.17%) patients.

Table 7: LOD and CT scan findings.

CT Findings	LOD*	
	No. (31)	%
Dilated ventricles	8	25.80
Cerebral atrophy	12	38.71
Cerebral infarction	5	16.12
WNL	9	29.30

* Patients had multiple findings.

Table 8: LOD and MRI findings.

MRI Findings	LOD*	
	No. (5)	%
Periventricular lesions	2	40.0
Deep white matter lesions	3	60.0
Enlarged ventricles	3	60.0
Cerebral atrophy	2	40.0
Basal ganglia lesions	-	
Hippocampal lesions	1	20.0
WNL	-	

* Patients had multiple findings.

The mini-GDS scale emerged as a highly efficient tool for screening of elderly depressed patients as it was simple, answers were either affirmative or negative causing no observer bias, easily understood even by illiterates, quick to administer and can be correlated appropriately with MADRS scores.

Overall, the late onset depressed showed more cognitive dysfunction in all the aspects as evident by mean MMSE scores (16.36) as compared to control group (p<0.001), depicted in Table 5.

Majority of the patients belonged to moderate to severe category 67.24% (moderate 40.52% and severe 26.72%). In the present study, there were more impairment in calculation (80.20%) followed by reading and comprehension (75.0%) and memory impairment (67.2%). Mini mental scoring does not seem reliable tool for measuring in illiterate and lower educated patients. Among hypertensive and dyslipidemic patients most had moderate to severe cognitive impairment (66.23%, 59.60%). Diabetic patients had minimal cognitive impairment (55%), mentioned in Table 6.

Out of 31 CT scans, around 29.3% had no radiological findings, dilated ventricles were found in 25.80% and

cerebral atrophy in 38.71% (Table 7). Cerebral infarction was present in 16.12% patients. The most common observations were preventricular lesions, enlarged ventricles and cortical atrophy in 2 patients (40.0%), deep white matter lesions among 3 (60.0%). Hippocampal lesions were observed only in 1 subject.

Similarly, the most common lesion observed in MRI were deep white matter hyperintensities, enlarged ventricles, cortical atrophy, and periventricular hyperintensities. Hippocampal lesions were also found. The MRI scan revealed higher incidence of significant lesions in the geriatric depressed group (100%) as shown in Table 8.

DISCUSSION

In our study there were higher late onset depressives which could be explained as it was a cumulative representation of both functional as well as organic major depressions. This finding emphasized the role of additional underlying organic factors in the genesis of the same. Similar conditions have been described by Krishnan et al.[7] The majority of patients were from the younger old-age, with a mean age of 64.5±4.5 years, which reflected the preponderance of this age group in the geriatric mass in India.[17] Other reasons of having lesser number of older-age subjects could be poor identification of depression in the very old age group, a false perception of physical illness and ageing process masking the diagnosis of depression, denial for depression in old age and overall apathy towards this age group leading to an eventual decrease in consultation. The males outnumbered females similar to Rao et al and Nandi et al also reported a skewed representation towards male preponderance.[18,19] This may be explained due to several sociocultural and socioeconomic factors including male dominated society and them gaining higher priorities as compared to their female counterparts. In contrast, the bulk of the earlier studies have shown a greater prevalence of females to males in depressive illness including geriatric depression.[20-22]

The majority of patients were married, reflecting primarily the Indian sociocultural scenario wherein universality of marriage and rare breakups due to greater familial support are prevalent. The loss of spouse emerged out as an important clinical variable with late onset depression suggesting a crucial role of disruption of family support and interpersonal relation in precipitating the depression, similar to previous studies.[23,24]

The higher number of rural patients simply reflects the rural dominance of this geographical area. Most of the subjects belonged to lower socioeconomic status, the common section of economic strata in India. The unprecedented rise in social pressure, poor adaptation to various stressors and constant hue and cry and enhanced allostatic load in this group sets up various biological changes in the CNS leading to endogenous depression.

The bulk of subjects were from joint families, which may be due to higher pathological communication, communication gap, subordination, loss of psychological space, identity crisis, intrafamilial conflicts and overall generation gap. Prompt identification of depression in joint family set up may be another reason for increased medical consultation, thereby resulting in increased prevalence. However, various studies have failed to link family jointness with depression.[25,26] On the contrary, Rao et al showed major depression to be higher in nuclear family in India.[18]

The majority of patients in our study were illiterate corresponding to level of literacy in our country though no relationship was noted in the age of onset of depression to educational status. Although some studies have reported a negative association between depression in old age and education level, further studies are needed to probe the link between the two.[22] The farmers and housewives predominated the study in males and females respectively because of the rural background of most of the patients. It was worth to note that most of these patients had stopped working much before the onset of depression. It appeared that lack of engagement in work makes them more vulnerable to depressive decompensation. However, long term studies in a larger sample size were needed to scientifically establish the relation of working pattern, nature of work and lack of engagement with major depression.

It was worth to note that the magnitude of substance abuse was fairly high even in geriatric age and as high as 68% of them had some sort of substance abuse. Soft substances like tobacco and bidi were more common than hard substances like alcohol and cannabis, which may be due to social sanction and as a token of friendly gesture of social communication. Both tobacco as well as bidi affected the central cholinergic transmission and may play some etiological role in the genesis of depression in geriatric depressive phenotypes. However, high prevalence of such dependence with a relatively lower incidence of depression, dilutes such hypothesis.

The majority had some or other stressors, of which predominant was the loss of spouse followed by family maladjustment and intrafamilial conflicts. The late onset depressives were associated with significantly higher stress and adverse life events consistent with the earlier studies, highlighting the role of stressful events in the etiopathogenesis of late onset depression.[27] There was no genetic loading of affective disorder in late onset depressives.[28]

All the patients of depression in the present study had some or the other comorbid illness, with hypertension as the leading comorbidity, followed by diabetes, arthritis, cataract, BPH, COPD, parkinsonism and IHD in decreasing order. Many patients were having multiple comorbidities of which co-existence of hypertension and diabetes mellitus was most prevalent as also reported by

Acharya in 1999.[29] The comorbid physical disorders can mask and sometimes may increase the magnitude the underlying major depression leading to poor outcome and similarly depression may aggravate the common physical disorders and may modify its course and outcome. It will therefore be worthwhile to set up a well-coordinated geriatric-medical and psychiatric clinic on the same platform for prompt identification and management of such co-existence to decrease the spiral interaction between depression and physcial comorbidity, enabling us to achieve a better compliance and adherence to physical as well as psychiatric treatment, physiotherapy and rehabilitation.

It was observed that although there was a higher percentage of subjects in moderate to severe range of depression in all the three risk factors, only hypertension showed significance in terms of raised MADRS scores as compared to non-hypertensives. Hypertension thus emerged out as a significant risk factor positively associated with increased severity of depression similar to other studies.[1,7] A cause-effect relationship is being proposed between depression and hypertension. Depression may lead to poor adherence to medical advice leading to ineffective control of hypertension. Depression can induce hypertension, per se, in geriatric population by hypercortisolaemia, sympathetic overactivity, hyper-arousal state, and various biochemical and neurotransmitter alterations. On the other hand, hypertension may lead to microangiopathic changes causing lipohyalinosis and lacunar infarcts in the watershed areas supplied by penetrating arterioles thus, causing vascular depression.[12] The presence of vascular pathology linked to depression in old age, supported by clinical and neuroimaging evidence, had clinically relevant, preventive and treatment implications. Recent works in the field of vascular depression may open new horizons in this regard.[1]

The most frequent symptoms observed in our study was sleep disturbances, followed by somatic complaints and hvpochondriasis, retardation, apathy, and lack of interest in work and activities, loss of weight, anxiety, agitation, low mood, lack of insight, guilt feeling, and suicidal ideation supported; also reported by different studies.[30-32]

As majority of our subjects were illiterate and from rural population, calculations and vocabulary testing were not useful parameters. However, there were more than half of our subjects showing memory impairment, which clearly suggests a decline in their cognitive function, similar to the report by Abas et al, which stated that memory deficit and cognitive slowing was present in 70%, the severity of which was comparable to a group of patients with Alzheimer's disease.[33] The studies by Schweitzer et al further supports the present observation that late onset depressives are more likely to have cognitive impairment and deep white matter lesions.[34]

Only a few subjects were subjected to neuroimaging, due to financial constraints, or negligent attitude of family member towards old. The CT scans revealed changes ranging from normal to cerebral atrophy to dilated ventricles to infarction mainly in the left frontal region. The only significant finding was the association of dilated ventricles with late onset depressed patients. Similar were observations by Jacoby et al.[35] Krishnan et al reported infarction in left frontal cortex, similar to our study.[7] The cerebral atrophy and dilated ventricles may be non-specific findings akin to older age or may be more preponderant in late onset depressive patients. Larger sample size comprising age and sex matched healthy and late onset depressed subjects should be evaluated using neuroimaging to resolve the issue.

Similarly, a small sample size with few MRI scans limits any conclusion. Interestingly, lesions same as our study were observed in the study by Coffey et al.[36]

CONCLUSION

Subclinical cognitive impairment and MCI may precede years before the onset of vascular depression. A prompt detection and management of these entities and associated risk factors could prevent or postpone impending vascular depression. As the depression is the 2nd most common cause of DALYS, its early diagnosis and treatment may improve the quality of life. Setting up a well-coordinated geriatric-medical and psychiatric clinic on the same platform for prompt identification and management of such co-existence would enable us to achieve a better compliance and adherence to physical as well as psychiatric treatment, physiotherapy and rehabilitation.

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