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CT Brain Abnormalities in Early- Versus Late-Onset Psychosis: A Comparative Evaluation Conducted at a Tertiary Care Hospital

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ABSTRACT

Background: Psychosis is a heterogeneous psychiatric condition with variable clinical presentation and underlying neurobiological mechanisms. Age at onset may influence the pattern of structural brain abnormalities, with late-onset psychosis potentially showing a greater contribution from age-related and cerebrovascular changes. Computed tomography (CT) brain imaging can help identify structural abnormalities that may contribute to the clinical presentation.

Aim: To compare CT brain abnormalities in patients with early-onset and late-onset psychosis and to assess factors associated with abnormal CT findings.

Materials and Methods: This hospital-based comparative observational study included 70 patients with psychosis at a tertiary care hospital. Patients were divided into early-onset psychosis (n=35) and late-onset psychosis (n=35) groups. Detailed demographic, psychiatric, medical, vascular, and neurological assessments were performed. Psychotic symptoms were evaluated using the Positive and Negative Syndrome Scale where applicable. CT brain scans were assessed for cortical and cerebellar atrophy, ventricular dilatation, white-matter hypodensities, infarcts, calcifications, encephalomalacia, and other structural abnormalities. Data were analysed using IBM SPSS Statistics, with $p<0.05$ considered statistically significant.

Results: CT brain abnormalities were significantly more frequent in late-onset than early-onset psychosis (68.57% vs 28.57%; p=0.001). Cerebral cortical atrophy (42.86% vs 11.43%; p=0.003), ventricular dilatation (37.14% vs 14.29%; p=0.029), white-matter hypodensities (31.43% vs 5.71%; p=0.006), lacunar infarcts (22.86% vs 2.86%; p=0.028), and old ischemic changes (25.71% vs 2.86%; p=0.006) were significantly more frequent in the late-onset group. Late-onset psychosis was associated with higher odds of CT abnormalities (OR=5.45; 95% CI: 1.96–15.18; p=0.001). Hypertension, diabetes mellitus, dyslipidaemia, and neurological abnormalities were also significantly associated with abnormal CT findings.

Conclusion: Late-onset psychosis was associated with a substantially greater burden of structural CT brain abnormalities, particularly atrophic and cerebrovascular changes. CT brain evaluation may provide useful diagnostic information in patients presenting with psychosis at an older age.

KEYWORDS: Psychosis; Late-Onset Psychosis; Early-Onset Psychosis; Computed Tomography; Brain Abnormalities.

INTRODUCTION

Psychosis represents a heterogeneous group of psychiatric conditions characterized by disturbances in perception, thought, behaviour, and interpretation of reality. Its principal manifestations include delusions, hallucinations, disorganized thinking or behaviour, negative symptoms, and varying degrees of cognitive and functional impairment. Psychotic symptoms may occur in schizophrenia spectrum disorders, mood disorders, delusional disorders, substance-related

conditions, neurological diseases, and several systemic medical disorders. The age at which psychotic symptoms first emerge has considerable clinical importance because the underlying etiological factors, symptom profile, associated medical conditions, treatment response, and structural brain characteristics may differ between individuals developing psychosis earlier and those presenting later in life.[1] Early-onset psychosis commonly develops during adolescence or young adulthood, a period characterized by continuing maturation of cortical networks, synaptic organization, and higher cognitive functions. Schizophrenia and related psychotic disorders have increasingly been conceptualized within neurodevelopmental and neurobiological frameworks in which structural and functional alterations involving multiple cerebral regions contribute to the development and persistence of symptoms. Structural neuroimaging has demonstrated that abnormalities may involve cortical grey matter, subcortical structures, ventricular systems, and white-matter pathways. These alterations are heterogeneous and may be influenced by age at onset, illness progression, treatment exposure, substance use, and associated physical disorders.[2] Late-onset psychosis, generally referring to the first appearance of persistent psychotic symptoms after 40 years of age, represents an important diagnostic group because new psychotic symptoms at an older age require careful exclusion of secondary causes. Neurological disorders, cerebrovascular disease, neurodegenerative processes, metabolic abnormalities, medication effects, infections, and other organic conditions can produce symptoms resembling primary psychiatric illness. Accordingly, neuroimaging becomes particularly relevant when psychosis appears later in life, especially when accompanied by cognitive deterioration, abnormal neurological findings, seizures, headache, altered consciousness, or vascular risk factors. A systematic assessment of the brain may therefore assist in differentiating primary psychiatric disorders from potentially identifiable intracranial abnormalities.[3] Age at onset may also correspond to differences in the nature and distribution of structural cerebral changes. Earlier onset of schizophrenia occurs while maturation of the brain, particularly the prefrontal and association cortices, is still progressing. Alterations occurring during this developmental period may affect cortical organization and subsequently influence clinical manifestations. Conversely, patients developing psychosis later in adulthood may have additional contributions from ageing-related cerebral changes, cerebrovascular pathology, metabolic disease, and accumulated vascular risk. These different biological contexts provide a rationale for comparing structural brain findings between early-onset and late-onset groups rather than considering psychosis as a single radiologically homogeneous disorder.[4] White-matter integrity is another important aspect of the neurobiology of schizophrenia and related psychotic disorders. Cerebral white matter contains the major pathways connecting frontal, temporal, parietal, limbic, and subcortical regions involved in perception, cognition, emotional regulation, and behaviour. Disruption of these connections has been proposed as one component of impaired neural integration in psychosis. Structural abnormalities affecting white matter may arise through neurodevelopmental mechanisms in younger patients, whereas ischemic and small-vessel changes may contribute increasingly with advancing age. Neuroimaging assessment of white-matter abnormalities, together with cortical and ventricular changes, can therefore provide a broader representation of structural brain involvement across different age-at-onset groups.[5] Structural alterations have also been demonstrated specifically in early-onset schizophrenia, involving cortical morphology, surface characteristics, regional volumes, and other measures of cerebral architecture. Such observations support the concept that psychosis beginning earlier in life may be associated with alterations in brain maturation and cortical development.[6] Nevertheless, structural abnormalities are not restricted to early-onset disorders. In older patients, interpretation of brain imaging becomes more complex because age-related atrophy, ventricular enlargement, vascular lesions, white-matter changes, old infarcts, and intracranial calcifications may coexist with psychiatric manifestations. Distinguishing these findings according to the timing of onset of psychosis may therefore improve understanding of the relationship between age, psychiatric symptoms, and cerebral structural changes.

MATERIALS & METHODS

This hospital-based comparative observational study was conducted at Department of Radiodiagnosis and Department of Psychiatry, National Institute of Medical Sciences & Research, NIMS University, Jaipur, Rajasthan (India) to assess and compare computed tomography (CT) brain abnormalities among patients with early-onset and late-onset psychosis. Patients presenting to the Department of Psychiatry, either through outpatient or inpatient services, who fulfilled the predefined eligibility criteria were enrolled. CT brain evaluation was performed through the Department of Radiodiagnosis using a standardized imaging protocol. A total of 70 patients diagnosed with psychotic disorders were included in the study. Participants were divided into two groups according to the age at onset of psychotic symptoms. Group I consisted of 35 patients with early-onset psychosis, defined as onset of the first sustained psychotic episode at or before 40 years of age, while Group II consisted of 35 patients with late-onset psychosis, defined as onset after 40 years of age.

Consecutive eligible patients were recruited until the required sample size was achieved.

Inclusion Criteria: Patients aged 18 years or older with a clinical diagnosis of a psychotic disorder according to standard diagnostic criteria were eligible. Patients with schizophrenia spectrum disorders, persistent delusional disorder, acute and transient psychotic disorder, or other primary psychotic disorders were considered when psychotic symptoms constituted the principal clinical presentation. Patients or their legally acceptable representatives were required to provide informed consent for participation and CT examination.

Exclusion Criteria: Patients with psychosis exclusively attributable to acute intoxication or withdrawal, delirium, previously established major neurocognitive disorder, known intracranial malignancy, previously diagnosed major structural brain lesion, central nervous system infection, congenital neurological disorder, or recent significant head injury were excluded. Patients who were clinically unstable or unable to undergo CT examination were also excluded.

Methodology

Clinical and Psychiatric Assessment: A detailed history was obtained regarding age, sex, educational status, occupation, residence, age at onset of psychotic symptoms, duration of illness, mode of onset, number of previous psychotic episodes, previous hospitalization, treatment history, medication use, treatment adherence, family history of psychiatric illness, and substance use. Current psychopathology was documented, including delusions, hallucinations, disorganized speech or behaviour, negative symptoms, affective symptoms, aggression, and suicidal behaviour. Severity of psychotic symptoms was assessed using the Positive and Negative Syndrome Scale (PANSS), wherever applicable.

Medical and Neurological Assessment: All participants underwent general physical and detailed neurological examination. Blood pressure, body mass index, diabetes mellitus, hypertension, dyslipidaemia, smoking status, alcohol use, and other vascular risk factors were documented because of their possible association with structural brain abnormalities. Neurological parameters included level of consciousness, cranial nerve examination, motor and sensory function, reflexes, gait abnormalities, tremor, rigidity, focal neurological deficits, seizures, and abnormal involuntary movements. Cognitive status was also clinically assessed, with standardized cognitive screening used when indicated.

CT Brain Protocol: CT brain examination was performed using a multidetector CT scanner. Non-contrast axial images of the brain were obtained from the skull base to the vertex using standard institutional protocols, with multiplanar reconstruction whenever required. Contrast-enhanced CT was performed only when clinically indicated. All scans were evaluated systematically by an experienced radiologist who was provided with the relevant clinical indication but was not involved in psychiatric categorization of the patients.

Assessment of CT Brain Abnormalities: CT scans were assessed for cerebral cortical atrophy, cerebellar atrophy, ventricular dilatation, asymmetry of the lateral ventricles, widening of cortical sulci, white-matter hypodensities, lacunar infarcts, territorial infarcts, basal ganglia calcification, intracranial calcification, focal encephalomalacia, old ischemic changes, hydrocephalus, cerebral edema, space-occupying lesions, and other significant structural abnormalities. Cerebral atrophy and ventricular enlargement were recorded as absent or present and further categorized according to radiological severity wherever feasible. The presence of single or multiple CT abnormalities was also documented.

Outcome Measures: The primary outcome was the difference in the overall frequency of CT brain abnormalities between patients with early-onset and late-onset psychosis. Secondary outcomes included comparison of individual radiological findings such as cortical atrophy, ventricular enlargement, white-matter changes, cerebrovascular lesions, intracranial calcification, and other structural abnormalities. Associations of CT abnormalities with age, sex, duration of illness, PANSS score, neurological findings, substance use, hypertension, diabetes mellitus, and other vascular risk factors were also evaluated.

Data Collection and Quality Control: Data were recorded in a structured case record form containing demographic, psychiatric, clinical, neurological, and radiological variables. Clinical records and CT findings were reviewed for completeness before data entry. Standardized definitions were used for major study variables to minimize classification errors.

Statistical Analysis

Data were entered and analysed using IBM SPSS Statistics. Continuous variables were expressed as mean ± standard deviation or median with interquartile range according to data distribution, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro-Wilk test. Independent-samples t-test or Mann-Whitney U test was used to compare continuous variables between the two groups. Chi-square test or Fisher's exact test was applied for categorical variables. Binary logistic regression analysis was performed, where appropriate, to identify independent factors associated with the presence of CT brain abnormalities after adjustment for relevant confounding variables. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic and Baseline Clinical Characteristics of the Study Groups

The mean age at assessment was 34.90 ± 8.10 years in the early-onset psychosis group and 56.80 ± 8.60 years

in the late-onset psychosis group, and this difference was statistically significant (p<0.001). Similarly, the mean age at onset was significantly lower in the early-onset group compared with the late-onset group (28.20 ± 6.70 years versus 52.90 ± 7.10 years; p<0.001). The mean duration of illness was 6.70 ± 4.40 years among patients with early-onset psychosis compared with 3.90 ± 2.80 years among those with late-onset psychosis, with a statistically significant difference (p=0.002). Males constituted 62.86% of the early-onset group and 45.71% of the late-onset group, while females constituted 37.14% and 54.29%, respectively; the sex distribution did not differ significantly between the groups (p=0.150). Rural residence was reported in 51.43% of early-onset and 60.00% of late-onset patients, whereas urban residence was noted in 48.57% and 40.00%, respectively, with no significant difference (p=0.470). A family history of psychiatric illness was significantly more frequent in the early-onset group, being present in 15 patients (42.86%) compared with 5 patients (14.29%) in the late-onset group (p=0.008). Substance use history was documented in 31.43% of early-onset patients and 20.00% of late-onset patients, and the difference was not statistically significant (p=0.274).

Table 2: Psychiatric Characteristics and PANSS Scores in Early-Onset and Late-Onset Psychosis

Delusions were observed in 30 patients (85.71%) with early-onset psychosis and 31 patients (88.57%) with late-onset psychosis, with no significant difference between the groups (p=0.721). Hallucinations were present in 71.43% of early-onset and 57.14% of late-onset patients (p=0.212). Disorganized behaviour or speech was recorded in 48.57% of patients in the early-onset group compared with 28.57% in the late-onset group, although the difference did not reach statistical significance (p=0.086). Negative symptoms were significantly more common in the early-onset group, occurring in 21 patients (60.00%) compared with 12 patients (34.29%) in the late-onset group (p=0.031). Affective symptoms were observed in 25.71% and 40.00% of the two groups, respectively (p=0.203), while aggressive behaviour was present in 28.57% of early-onset and 20.00% of late-onset patients (p=0.403). Previous psychiatric hospitalization was reported in 51.43% of early-onset patients and 28.57% of late-onset patients (p=0.051), whereas treatment non-adherence was recorded in 45.71% and 25.71%, respectively (p=0.081). The mean PANSS positive score was 23.60 ± 5.40 in the early-onset group and 21.80 ± 5.20 in the late-onset group (p=0.160). The mean PANSS negative score was significantly higher in the early-onset group (20.70 ± 5.60) than in the late-onset group (17.90 ± 5.10; p=0.032). The mean general psychopathology score was 40.30 ± 7.20 versus 39.50 ± 7.00 (p=0.639), while the mean total PANSS score was 84.60 ± 13.20 and 79.20 ± 12.50, respectively (p=0.083).

Table 3: Medical, Vascular and Neurological Characteristics of the Study Groups

The mean body mass index was 24.80 ± 3.50 kg/m² in patients with early-onset psychosis and 25.70 ± 3.80 kg/m² in patients with late-onset psychosis, with no significant difference between the groups (p=0.306). Hypertension was documented in 3 patients (8.57%) in the early-onset group and 13 patients (37.14%) in the late-onset group, and the difference was statistically significant (p=0.004). Diabetes mellitus was present in 5.71% of early-onset patients compared with 25.71% of late-onset patients (p=0.022). Dyslipidaemia was also significantly more frequent among patients with late-onset psychosis, affecting 12 patients (34.29%) compared with 4 patients (11.43%) in the early-onset group (p=0.023). Current or past smoking was reported in 28.57% and 25.71% of patients, respectively (p=0.788), while alcohol use was present in 20.00% of early-onset and 17.14% of late-onset patients (p=0.759). Any neurological abnormality was observed in 4 patients (11.43%) in the early-onset group and 11 patients (31.43%) in the late-onset group, showing a statistically significant difference (p=0.041). Focal neurological deficit was present in 5.71% versus 17.14% (p=0.259), gait abnormality in 5.71% versus 20.00% (p=0.151), and history of seizures in 5.71% versus 8.57% (p=1.000), with none of these individual differences reaching statistical significance.

Table 4: Comparison of CT Brain Abnormalities Between Early-Onset and Late-Onset Psychosis

Overall, CT brain abnormalities were identified in 10 patients (28.57%) with early-onset psychosis and 24 patients (68.57%) with late-onset psychosis, and the difference was statistically significant (p=0.001). Cerebral cortical atrophy was present in 4 patients (11.43%) in the early-onset group compared with 15 patients (42.86%) in the late-onset group (p=0.003). Ventricular dilatation was observed in 14.29% and 37.14% of patients, respectively, with a significant difference (p=0.029). White-matter hypodensities were detected in 2 patients (5.71%) in the early-onset group and 11 patients (31.43%) in the late-onset group (p=0.006). Lacunar infarcts were present in 2.86% of early-onset patients compared with 22.86% of late-onset patients (p=0.028), while old ischemic changes were identified in 2.86% and 25.71%, respectively (p=0.006). Basal ganglia or intracranial calcification was observed in 5.71% versus 14.29% (p=0.428), cerebellar atrophy in 2.86% versus 11.43% (p=0.356), ventricular asymmetry in 8.57% versus 14.29% (p=0.710), and focal encephalomalacia in 2.86% versus 11.43% (p=0.356); these differences were not statistically significant. A normal CT brain was recorded in 25 patients (71.43%) with early-onset psychosis and 11 patients (31.43%) with late-onset psychosis (p=0.001). A single CT abnormality was present in 7 patients (20.00%) in the

early-onset group and 10 patients (28.57%) in the late-onset group, whereas multiple CT abnormalities were detected in 3 patients (8.57%) and 14 patients (40.00%), respectively. The overall distribution of normal CT, single abnormality, and multiple abnormalities differed significantly between the two groups (p=0.001).

Table 5: Factors Associated with Presence of CT Brain Abnormalities in the Overall Study Population

Among the 34 patients with CT brain abnormalities, 24 (70.59%) had late-onset psychosis, compared with 11 of 36 patients (30.56%) with normal CT findings. Late-onset psychosis was associated with higher odds of having a CT brain abnormality, with an odds ratio of 5.45 (95% CI: 1.96–15.18; p=0.001). Hypertension was present in 12 patients (35.29%) with CT abnormalities compared with 4 patients (11.11%) with normal CT scans, corresponding to an odds ratio of 4.36 (95% CI: 1.24–15.31; p=0.016). Diabetes mellitus was present in 26.47% of patients with abnormal CT findings and 5.56% of those with normal CT findings, with an odds ratio of 6.12 (95% CI: 1.21–30.83; p=0.016). Dyslipidaemia was recorded in 35.29% of patients with CT abnormalities and 11.11% of patients with normal CT findings, yielding an odds ratio of 4.36 (95% CI: 1.24–15.31; p=0.016). Neurological abnormalities were observed in 12 patients (35.29%) with abnormal CT findings compared with 3 patients (8.33%) with normal CT findings, with an odds ratio of 6.00 (95% CI: 1.52–23.74; p=0.006).

Smoking was present in 32.35% of patients with CT abnormalities and 22.22% of those with normal CT findings, but the association was not statistically significant (OR=1.67, 95% CI: 0.58–4.85; p=0.341). A family history of psychiatric illness was documented in 23.53% of patients with CT abnormalities and 33.33% of patients with normal CT findings, with no statistically significant association (OR=0.62, 95% CI: 0.21–1.76; p=0.364).

Table 1. Demographic and Baseline Clinical Characteristics of the Study Groups

Parameter	Early-Onset Psychosis (n=35)	Late-Onset Psychosis (n=35)	p value
Age at assessment (years), mean ± SD	34.90 ± 8.10	56.80 ± 8.60	<0.001*
Age at onset (years), mean ± SD	28.20 ± 6.70	52.90 ± 7.10	<0.001*
Duration of illness (years), mean ± SD	6.70 ± 4.40	3.90 ± 2.80	0.002*
Male sex	22 (62.86%)	16 (45.71%)	0.150
Female sex	13 (37.14%)	19 (54.29%)	—
Rural residence	18 (51.43%)	21 (60.00%)	0.470
Urban residence	17 (48.57%)	14 (40.00%)	—
Family history of psychiatric illness	15 (42.86%)	5 (14.29%)	0.008*
Substance use history	11 (31.43%)	7 (20.00%)	0.274

*Statistically significant at p<0.05.

Table 2. Psychiatric Characteristics and PANSS Scores in Early-Onset and Late-Onset Psychosis

Psychiatric Parameter	Early-Onset Psychosis (n=35)	Late-Onset Psychosis (n=35)	p value
Delusions	30 (85.71%)	31 (88.57%)	0.721
Hallucinations	25 (71.43%)	20 (57.14%)	0.212
Disorganized behaviour/speech	17 (48.57%)	10 (28.57%)	0.086
Negative symptoms	21 (60.00%)	12 (34.29%)	0.031*
Affective symptoms	9 (25.71%)	14 (40.00%)	0.203
Aggressive behaviour	10 (28.57%)	7 (20.00%)	0.403
Previous psychiatric hospitalization	18 (51.43%)	10 (28.57%)	0.051
Treatment non-adherence	16 (45.71%)	9 (25.71%)	0.081
PANSS positive score, mean ± SD	23.60 ± 5.40	21.80 ± 5.20	0.160
PANSS negative score, mean ± SD	20.70 ± 5.60	17.90 ± 5.10	0.032*
PANSS general psychopathology score, mean ± SD	40.30 ± 7.20	39.50 ± 7.00	0.639
PANSS total score, mean ± SD	84.60 ± 13.20	79.20 ± 12.50	0.083

*Statistically significant at p<0.05.

Table 3. Medical, Vascular and Neurological Characteristics of the Study Groups

Parameter	Early-Onset Psychosis (n=35)	Late-Onset Psychosis (n=35)	p value
BMI (kg/m²), mean ± SD	24.80 ± 3.50	25.70 ± 3.80	0.306
Hypertension	3 (8.57%)	13 (37.14%)	0.004*
Diabetes mellitus	2 (5.71%)	9 (25.71%)	0.022*
Dyslipidaemia	4 (11.43%)	12 (34.29%)	0.023*
Current/past smoking	10 (28.57%)	9 (25.71%)	0.788
Alcohol use	7 (20.00%)	6 (17.14%)	0.759
Any neurological abnormality	4 (11.43%)	11 (31.43%)	0.041*
Focal neurological deficit	2 (5.71%)	6 (17.14%)	0.259
Gait abnormality	2 (5.71%)	7 (20.00%)	0.151
History of seizures	2 (5.71%)	3 (8.57%)	1.000

*Statistically significant at p<0.05.

Table 4. Comparison of CT Brain Abnormalities Between Early-Onset and Late-Onset Psychosis

CT Brain Finding	Early-Onset Psychosis (n=35)	Late-Onset Psychosis (n=35)	p value
Any CT brain abnormality	10 (28.57%)	24 (68.57%)	0.001*
Cerebral cortical atrophy	4 (11.43%)	15 (42.86%)	0.003*
Ventricular dilatation	5 (14.29%)	13 (37.14%)	0.029*
White-matter hypodensities	2 (5.71%)	11 (31.43%)	0.006*
Lacunar infarcts	1 (2.86%)	8 (22.86%)	0.028*
Old ischemic changes	1 (2.86%)	9 (25.71%)	0.006*
Basal ganglia/intracranial calcification	2 (5.71%)	5 (14.29%)	0.428
Cerebellar atrophy	1 (2.86%)	4 (11.43%)	0.356
Ventricular asymmetry	3 (8.57%)	5 (14.29%)	0.710
Focal encephalomalacia	1 (2.86%)	4 (11.43%)	0.356
Normal CT brain	25 (71.43%)	11 (31.43%)	0.001*
Single CT abnormality	7 (20.00%)	10 (28.57%)	—
Multiple CT abnormalities	3 (8.57%)	14 (40.00%)	—

Table 5. Factors Associated With Presence of CT Brain Abnormalities in the Overall Study Population

Factor	CT Abnormality Present (n=34)	CT Normal (n=36)	Odds Ratio (95% CI)	p value
Late-onset psychosis	24 (70.59%)	11 (30.56%)	5.45 (1.96–15.18)	0.001*
Hypertension	12 (35.29%)	4 (11.11%)	4.36 (1.24–15.31)	0.016*
Diabetes mellitus	9 (26.47%)	2 (5.56%)	6.12 (1.21–30.83)	0.016*
Dyslipidaemia	12 (35.29%)	4 (11.11%)	4.36 (1.24–15.31)	0.016*
Neurological abnormality	12 (35.29%)	3 (8.33%)	6.00 (1.52–23.74)	0.006*
Smoking	11 (32.35%)	8 (22.22%)	1.67 (0.58–4.85)	0.341
Family history of psychiatric illness	8 (23.53%)	12 (33.33%)	0.62 (0.21–1.76)	0.364

*Statistically significant at p<0.05.

DISCUSSION

In the present study, the mean age at assessment was 34.90 ± 8.10 years in the early-onset psychosis group and 56.80 ± 8.60 years in the late-onset group (p<0.001), while the corresponding mean ages at onset were 28.20 ± 6.70 and 52.90 ± 7.10 years, respectively (p<0.001). Males constituted 62.86% of the early-onset group compared with 45.71% of the late-onset group, whereas

females constituted 37.14% and 54.29%, respectively. Although the difference in sex distribution was not statistically significant (p=0.150), relatively more females were represented in the late-onset group. This finding was comparable with Lindamer et al. (1999), who assessed 124 older patients with schizophrenia, including 90 with early-onset and 34 with late-onset schizophrenia. They reported that a significantly greater proportion of women than men had late-onset schizophrenia (41% versus 20%). Their observations were therefore consistent with the greater female representation noted among patients with late-onset psychosis in the present study.[7]

Family history of psychiatric illness showed a significant difference between the two groups in the present study, being reported in 15 patients (42.86%) with early-onset psychosis compared with only 5 patients (14.29%) with late-onset psychosis (p=0.008). Substance use was numerically more frequent in the early-onset group than in the late-onset group (31.43% versus 20.00%), although the difference was not statistically significant (p=0.274). The mean duration of illness was also significantly longer among early-onset patients (6.70 ± 4.40 years) than among late-onset patients (3.90 ± 2.80 years; p=0.002). Howard et al. (1997) studied 269 first-degree relatives of patients with late-onset non-affective psychosis and 272 first-degree relatives of healthy elderly controls. With an age-at-risk interval of 15–50 years, the estimated lifetime risk of schizophrenia was 1.3% in relatives of both cases and controls; when the age-at-risk interval was expanded to 15–90 years, the corresponding risks were 2.3% and 2.2%. Their findings demonstrated relatively low familial loading for schizophrenia in late-life psychosis, which was comparable with the significantly lower frequency of family psychiatric history observed in the late-onset group of the present study.[8]

Regarding psychiatric manifestations, delusions were common in both groups and were recorded in 85.71% of patients with early-onset psychosis and 88.57% of those with late-onset psychosis (p=0.721). Hallucinations were present in 71.43% and 57.14%, respectively (p=0.212). In contrast, negative symptoms were significantly more frequent among early-onset patients (60.00%) than among late-onset patients (34.29%; p=0.031). The mean PANSS negative score was also significantly higher in the early-onset group (20.70 ± 5.60) compared with the late-onset group (17.90 ± 5.10; p=0.032). Castle et al. (1997) compared 72 patients with very-late-onset schizophrenia-like illness with 192 patients with very-early-onset illness. Patients with very-late onset were more frequently female and were more likely to exhibit persecutory delusions and hallucinations, whereas they were less likely to have negative schizophrenic symptoms. The lower prevalence of negative symptoms among patients with later onset in that study was consistent with the significantly lower frequency of negative symptoms and lower PANSS negative scores observed in the late-onset group of the present study.[9]

The present study demonstrated a greater burden of vascular and neurological abnormalities among patients with late-onset psychosis. Hypertension was present in 37.14% of patients with late-onset psychosis compared with 8.57% of those with early-onset psychosis (p=0.004), diabetes mellitus in 25.71% versus 5.71% (p=0.022), and dyslipidaemia in 34.29% versus 11.43% (p=0.023). Any neurological abnormality was documented in 31.43% of patients in the late-onset group compared with 11.43% in the early-onset group (p=0.041). Breitner et al. (1990) examined eight patients with late-onset psychosis and eight elderly controls using magnetic resonance imaging. All eight patients with late-onset psychosis demonstrated significant leukoencephalopathy or vascular pathology, with temporoparietal and occipital abnormalities being particularly prominent, whereas little comparable pathology was evident among controls. Although MRI rather than CT was used, their observation of substantial vascular and white-matter pathology in late-onset psychosis was comparable with the greater vascular and neurological burden found in the late-onset group of the present study.[10]

A major observation of the present study was the significantly higher frequency of structural CT brain abnormalities among patients with late-onset psychosis. Abnormal CT findings were present in 24 of 35 late-onset patients (68.57%) compared with 10 of 35 early-onset patients (28.57%; p=0.001). Conversely, CT findings were normal in 71.43% of early-onset patients compared with only 31.43% of late-onset patients. Gewirtz et al. (1994) performed CT brain examinations in 168 patients during their first admission for psychosis and reported cerebral cortical atrophy in 40% of patients. The frequency of cortical atrophy increased with increasing age. Other noteworthy CT abnormalities were detected in 6.6% of patients and included cerebral infarctions, arachnoid cysts, venous angioma, colloid cyst, cavum vergae, and post-traumatic changes. Their observation that CT-detected atrophy increased with age was comparable with the substantially greater overall frequency of structural abnormalities observed among the older, late-onset patients in the present study.[11]

Among individual structural abnormalities in the present study, cerebral cortical atrophy was significantly more common among patients with late-onset psychosis than among those with early-onset psychosis (42.86% versus 11.43%; p=0.003). Ventricular dilatation was similarly more frequent in the late-onset group (37.14%) than in the early-onset group (14.29%; p=0.029). Malla et al. (2002) performed CT brain examinations in 114 patients with first-episode schizophrenia or schizophreniform psychosis and demonstrated modest enlargement of the

cerebral sulci and ventricles together with altered sylvian fissure asymmetry. Age was found to be the only independent predictor of these regional CT changes, while clinical symptoms, sex, and duration of untreated psychosis were not related to CT ratings. In the present study, cortical atrophy was approximately 3.75 times and ventricular dilatation approximately 2.60 times as frequent in the late-onset group as in the early-onset group, corresponding with the age-related structural changes described by Malla et al.[12]

Cerebrovascular-type abnormalities on CT also showed marked differences between the two groups. White-matter hypodensities were identified in 31.43% of patients with late-onset psychosis compared with 5.71% of those with early-onset psychosis (p=0.006). Lacunar infarcts occurred in 22.86% versus 2.86% (p=0.028), while old ischemic changes were identified in 25.71% versus 2.86% (p=0.006), respectively.

Tonkonogy et al. (1999) compared magnetic resonance imaging findings in 13 patients with late-onset paranoid psychosis, with a mean age of 66.33 years, and 35 elderly patients with early-onset paranoid schizophrenia, with a mean age of 63.89 years. Their study identified differences in structural brain abnormalities between late-onset and early-onset paranoid psychosis and specifically examined the contribution of white-matter lesions after minimizing the effect of age. The greater occurrence of white-matter and ischemic-type abnormalities in the late-onset group of the present study similarly demonstrated a distinct pattern of cerebral structural involvement according to age at onset.[13]

In the overall analysis of the present study, late-onset psychosis was associated with 5.45-fold higher odds of an abnormal CT brain finding (95% CI: 1.96–15.18; p=0.001). Neurological abnormality was associated with an OR of 6.00 (95% CI: 1.52–23.74; p=0.006). Hypertension (OR=4.36; 95% CI: 1.24–15.31; p=0.016), diabetes mellitus (OR=6.12; 95% CI: 1.21–30.83; p=0.016), and dyslipidaemia (OR=4.36; 95% CI: 1.24–15.31; p=0.016) were also significantly associated with abnormal CT findings. In contrast, smoking and family history of psychiatric illness were not significantly associated with CT abnormalities. Battaglia et al. (1988) performed CT brain examinations in 45 patients admitted with a first episode of psychosis and reported that 42 patients (93.33%) showed no evidence of intracranial pathology, whereas 3 patients (6.67%) demonstrated positive CT findings that correlated with neuropsychiatric symptomatology. Although their overall CT abnormality rate was considerably lower than the 48.57% (34/70) recorded in the present study, their finding that positive CT abnormalities were associated with relevant neuropsychiatric features was compatible with the significant relationship between neurological abnormalities and abnormal CT findings demonstrated in the present study.[14]

CONCLUSION

The present study demonstrated that CT brain abnormalities were significantly more frequent in patients with late-onset psychosis than in those with early-onset psychosis. Cerebral cortical atrophy, ventricular dilatation, white-matter hypodensities, lacunar infarcts, and old ischemic changes were particularly common in the late-onset group. Vascular risk factors and neurological abnormalities were also significantly associated with abnormal CT findings. CT brain evaluation may therefore provide useful structural information, particularly in patients presenting with psychosis at a later age.

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