Clinicopathologic Correlations in Older Adults with Schizophrenia

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Introduction
Cognitive impairment is a pervasive and severe symptom domain in schizophrenia, affecting attention, learning and memory, working memory, and executive function. It may present prior to the prodromal phase of schizophrenia and is established once the patient is diagnosed with this condition. The cognitive deficits have been shown to remain relatively stable over the course of illness but then accelerate with aging. This accelerated cognitive decline remains poorly understood. We therefore sought to evaluate whether it correlated with post-mortem neuropathology.

Methods
39 older adults with chronic schizophrenia.
• Data includes clinical assessment, neurocognitive testing, and detailed post-mortem neuropathological evaluation.
• Groups evaluated in terms of their demographic, clinical and neuropathological characteristics.

Results
• The mini mental status exam (MMSE), along with other neurocognitive tests such as digit span forward/backward (DSF/DSB), were not significantly different between neuropathological groups while significantly different between neuropsychiatric groups.
• Patients that passed away at an older age and experienced a longer duration of disease were more likely to have a higher burden of neurodegenerative pathology, and this was correlated with lower scores on dementia-related testing (CERAD) and global functioning assessments (GAF).
• The positive and negative symptoms scales (PANSS positive/negative) were significantly different among neuropsychiatric groups.

Discussion
These results suggest that the accelerated age-related cognitive impairments seen in chronic schizophrenia occur independent of neurodegenerative and other pathological changes that commonly explain cognitive deficits in the older adult population.

Future directions
Future histological studies in this cohort will aim to provide further insights into alternative factors responsible for this phenomenon.

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Age-related cognitive decline observed in schizophrenia occurs independent of neurodegenerative and other pathologies.

Table 1
Distribution of patients between neuropathological and neuropsychiatric groups.

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Score</th>
<th>Mild executive dysfunction</th>
<th>Significant neuropathology</th>
<th>Total (Row)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild ExD</td>
<td></td>
<td>7</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>Moderate ExD</td>
<td>8</td>
<td>8</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Severe ExD</td>
<td>8</td>
<td>6</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
<td>16</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 1
Mini Mental Status Examination (MMSE) was not significantly different among neuropathological groups, while significantly different (p=0.0001) among neuropsychiatric groups.

Figure 2
Digit Span Forward (DSF) was not significantly different among neuropathological groups, while significantly different (p=0.001) among neuropsychiatric groups.

Figure 3
Digit Span Backward (DSB) was not significantly different among neuropathological groups, while significantly different (p=0.0001) among neuropsychiatric groups.

Figure 4
Age at Death (AAD) was significantly different (p=0.001) among neuropathological groups, and significantly different (p=0.0165) among neuropsychiatric groups.

Figure 5
Disease Duration (DD) was significantly different (p=0.0058) among neuropathological groups, and significantly different (p=0.0073) among neuropsychiatric groups.

Figure 6
Consortium to Establish a Registry for Alzheimer’s Disease (CERAD) was significantly different (p=0.0298) among neuropathological groups, and significantly different (p=0.0012) among neuropsychiatric groups.

Figure 7
Global Assessment of Functioning (GAF) was significantly different (p=0.0002) among neuropathological groups, while not significantly different among neuropsychiatric groups.