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Understanding CIAS: What Does Current Literature Say About the Etiology and Pathophysiology of CIAS?

Announcer:
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Dr. Strassnig:
This is CME on ReachMD and I'm Dr. Martin Strassnig. Here with me today is Dr. Phil Harvey.

Now, Phil, let's dive right in. What do we know about the pathophysiology of cognitive impairment associated with schizophrenia, or CIAS?

Dr. Harvey:
Yeah, let's start by talking about the characteristics of cognitive impairment and the prevalence. Cognitive impairments in schizophrenia is very wide ranging. It occurs before the onset of psychotic symptoms. It affects a number of different cognitive abilities, including processing speed, working memory, and executive functioning. In contrast to cortical dementia like Alzheimer's disease, cognitive impairment in schizophrenia is not marked by rapid forgetting. So what we see is a different syndrome that's commonly been referred to as frontal striatal cognitive impairments. Interestingly enough, we've discovered after the syndrome was characterized that the origin of cognitive impairment in schizophrenia may be a lack of good connection between the striatum and the cortex. NMDA receptor hypoactivity involving the glutamatergic system is implicated in reducing the level of activation of the dopamine projections to the cortex. So in contrast to the idea that blockading dopamine in the striatum reduces psychotic symptoms, we think that cognitive impairment in schizophrenia is produced by not enough dopaminergic activity in the cortex.

So the idea is the circuitry, which involves glutamates, glycine, it also involves the muscarinic cholinergic receptor system and dopamine, are all implicated in the circuit failure that leads to cognitive impairment in schizophrenia. Since it starts early, it's obviously something that's going on before we start to see psychotic symptoms, and it may very well temporally coincide with the presence of negative symptoms of the illness as well, which are also commonly seen before the onset of psychotic symptoms.

So I think we need to keep in mind that cognitive impairment in schizophrenia is wide ranging, it is prevalent across the majority of people up to 85%, and the NMDA receptor system is actually implicated in driving the origins of cognitive impairment, like it may very well be in terms of other features of the illness, including psychotic symptoms and negative symptoms.

Dr. Strassnig:
What's important here, from my perspective, that concept of NMDA hypofrontality that is in the frontal lobe, there is basically a deficit, often GABAergic neuronal input, that kind of slows the brain processing down. It basically causes impaired cognitive function.

Dr. Harvey:
Well, I think it's also important to keep in mind that dopamine may be the transmitter that's most proximally associated with both

cognitive impairment and psychosis, because the NMDA hypoactivity leads to increased striatal dopamine activity, which is dampened down by antipsychotics, and decreased cortical dopaminergic activity. So dopamine tends to be the transmitter in effect at the point of the impairment, but what's driving those dopaminergic alterations is probably glutamatergic and GABAergic, just like you said.

Dr. Strassnig:
Yes. And I agree with that. So it is relatively complex and I think we'll cover some of the mechanisms in a later podcast.

But for now, I think this has been a great bite-sized discussion. That's our time. And thanks for listening.

Announcer:
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