Early-onset schizophrenia (EOS) is operationally defined by an onset prior to age of 18 years, with those antedating the age of 13 being designated as very early onset schizophrenia. These psychoses are generally notorious to have male preponderance, heavy genetic loads, soft neurologic signs, incipient onsets, negative and cognitive domain presentations, poor antipsychotic responses that ultimately culminate into poor prognostication (1). Current state-of-the-art psychopharmacotherapy of psychosis in pediatric population favors atypical antipsychotics (AAP) over conventional antipsychotics defamed for their neurologic side-effects, namely, extrapyramidal syndromes (EPS). Of these APPs, Risperidone, paliperidone, quetiapine, olanzapine and aripiprazole only are FDA-approved for a schizophrenia age group of 13 on (2). Nonetheless, virtually all antipsychotics are used off-label in “real-life” practice. Paliperidone, 9-hydroxyrisperidone, is AAP, serotonin-dopamine antagonist, active metabolite of high-potency risperidone, and advantageous given OROS technique facilitating once-daily dosing, rapid steady-state and hence better compliance. Moreover, lack of CYP enzyme interactions, more alpha-2 affinity, alleged less propensity for EPS and availability of long-acting injection formulation all render paliperidone a very appealing treatment option in psychoses (3). Gabapentin is anticonvulsant, GABA analogue, binds selectively to alpha-2 delta-1 subunit of Ca channels, renally-cleared that has been used in psychiatry for a multitude of indications including, inter alia, generalized anxiety disorder, restless leg syndrome, alcoholism and bipolarity (4). Here we report on a case of EOS that favorably responded to paliperidone but incurred EPS in the form of head tremor that failed to be suppressed by propranolol. The addition of gabapentin successfully alleviated head tremor, helped with concomitant agitation and insomnia and, surprisingly, augmented antipsychotic response. We postulate GABA potentiation by gabapentin boosts dopamine blockade in the mesolimbic pathway and attenuates serotonergic tone to the mesocortical pathway. This goes hand-in-hand with hypothesized GABA deficiency underpinnings of schizophrenic neurobiology (5). It was achieved with high tolerability. We conclude that gabapentin is a very attractive augmentative strategy to the psychopharmacologic armamentarium of schizophrenia.

A 15-year-old male Syrian youngster was accompanied by his parents for scholastic failure coupled with behavioral oddities. This dates back two years with gradual onset of social isolation, neglected self-hygiene, fragmented sleep, scholastic deterioration, pseudophilosophical speech and at times inordinate laughter. This ran a progressive course to end with the boy muttering under his breath, moving with an awkward gait and displaying a caricatured facial demeanor. He is the eldest of three siblings from a monogamous, consanguineous family. He had unremarkable developmental trajectories prior to illness. A family history is positive for a schizophrenic paternal uncle. No medical history of relevance or known drug allergies were noted. No history of illicit substance use or ongoing legal problems was reported. Mental state examination revealed a lanky habitus, tousled, shabby, brief eye contact, lack of rapport, a bit agitated, slavering, ungrounded laughter, disjointed, gibberish speech, restricted and at times incongruous affect, formal thought disorder and hallucinatory attitude; a whole profile reminiscent of classic hebephrenics. Baseline lab and neuro-imaging were within a normal range and the toxic screen was negative. The patient was given paliperidone 3mg morning dose, escalated to 6mg over a week. At Wk-3, some tangible improvement was noticed in the form of less disorganized symptom cluster. But sleep was still fitful and bouts of agitation were occasionally noted. The dose was pushed higher to 9mg/d. At Wk-5, much improvement in positive domain was seen, but strikingly, he started to exhibit “no-no” head tremors (tremblement negatif). Propranolol was tried at 30mg/d, to no avail. Neurologic consult was summoned where a diagnosis of paliperidone-induced dyskinesia was entertained and gabapentin 300mg night dose prescribed. At Wk-7, tremors totally abated, sleep improved, agitation markedly diminished and the patient kept improving. This was attributed to soporific effects of gabapentin. At Wk-10, we attempted withdrawing gabapentin, but the patient began to show psychotic decompensation with re-emergence of tremors. Gabapentin was resumed; tremors disappeared.
with attenuation of psychotic symptoms. Follow-ups at Wk-12, 16 and 20 demonstrated plateau improvement with no EPS. Gabapentin appeared to counteract EPS of paliperidone but also helped insomnia, agitation and above all augmented antipsychotic response. This was achieved with great tolerability. This response goes in tandem with an open-label pilot study in adults by Gabriel (6) showing promising outcomes. We opine that gabapentin might be a viable option to augment antipsychotic response and mitigate neurologic side effects in pediatric population.

Disclosures:
Author declares no conflicts of interest.

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