Antipsychotics and OCD: Boon or Bane?

Sir,

Some of the major changes in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition are the relocation of obsessive-compulsive disorder (OCD) and acknowledging a delusional specifier. This should be borne in mind while working the differential for OCD/psychosis interface which might also entail – obsessive-compulsive symptoms (OCS) as a psychotic prodroma, schizotypal PD with superimposed OCD, Asperger's syndrome, "schizo-obsessive" disorder, and atypical antipsychotic (AAP)-induced OCS.

AAPs are a common augmentative strategy in treatment-resistant OCD. Moreover, some have contended that dopamine blockade is critical to facilitate 5-hydroxytryptamine (5HT) transmission in the cortico-striato-pallido-thalamo-cortical neurocircuitry in OCD. Indicators to use this tactic might include tics, schizotypal personality disorder, autism spectrum disorder, poor insight, and mood instability. Of the AAPs, best available evidence is for risperidone as shown in a meta-analysis by Dold *et al*.[1] and also aripiprazole as demonstrated by Ducasse *et al*.[2] in a meta-regression analysis. This would translate clinically into a lower threshold to employ AAPs in the pharmacotherapy of OCD earlier than later.

Paradoxically, APPs have been notoriously reported to induce *de novo* or exacerbate preexisting OCD in schizophrenia. This seems to be related chiefly to the primary diagnosis. It has also been reported in bipolar mood disorders, intellectual disability, delusional disorder, and psychotic depression. Lim *et al*.[3] have found an incidence of 12% of AAPs-induced OCS in a cohort of schizophrenia and schizo-affective disorder (n = 209).

Since 5HT dysregulation is central to OCD neurobiology, it seems that AAPs by virtue of 5HT2A antagonism might mechanistically induce these OCS as demonstrated by Zink.[4]

Agents with higher 5HT2A affinity seem to have a stronger potential to induce OCD notably clozapine. Lin *et al*.[5] have found that patients developing clozapine-induced OCS had higher concentrations of clozapine and norclozapine. Kwon *et al*.[6] have demonstrated the association between SLC1A1 gene and AAPs-induced OCS in schizophrenia.

Poyurovsky *et al*.[7] have proposed clinical criteria to diagnose AAPs-induced OCS. These include male preponderance, preexisting OCS in schizophrenia, compulsions, during the 1st week of treatment, and typically with higher doses.

Treatment typically entails dose reduction or entirely discontinuation of culprit AAP, introduction of selective serotonin reuptake inhibitors (SSRIs), or both. Aripiprazole and amisulpride by virtue of unique secondary pharmacodynamic properties remain viable options.

To pull it together, the use of APP, especially risperidone or aripiprazole, *ab initio*, is highly indicated in cases of bona fide OCD, earlier rather than later and more so if coupled with clinical correlates highlighted above (e.g., tic-related OCD). On the other hand, if the primary diagnosis is schizophrenia and patients phenomenologically endorse OCS, which

is commonplace in 5%–45%, then clinicians might opt to try aripiprazole or the substituted benzamide amisulpride, and closely clinically monitoring for OCS accentuation, in which case, add-on SSRI might be a reasonable course of action, given additive neuroprotective actions by virtue of SSRI-enhanced dentate gyrus neurogenesis. A caution, however, should be exercised here regarding significant pharmacokinetic interactions between SSRIs known to be potent cytochrome P450 inhibitors (e.g., fluoxetine on 2D6) and AAPs (e.g., risperidone as a substrate for 2D6).

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