Covert dysphagia and recurrent pneumonia related to antipsychotic treatment

Jonathan T. Stewart, MD

A 65-year-old man with a lifelong history of paranoid schizophrenia had been treated with 50 mg of risperidone microspheres every 2 weeks for the past 10 years and had tolerated this therapy well. Approximately 1 year ago some drooling and a modest resting tremor developed, but there were no other signs of parkinsonism. He also experienced gradually worsening pleural effusion. Six months after these symptoms developed, he experienced 3 prolonged hospital admissions for aspiration pneumonia over a period of 2 months. A swallowing evaluation at that time revealed severe oropharyngeal dysphagia, and a percutaneous endoscopic gastrostomy tube was placed. Owing to concerns of possible antipsychotic-induced dysphagia, risperidone was discontinued and substituted with 400 mg/d of quetiapine. The patient’s condition improved over the subsequent 3 months, with no further pneumonia and advancement back to a regular diet. The pleural effusion resolved; it was probably a parapneumonic effusion related to chronic silent microaspiration.

There is growing evidence that antipsychotic treatment is an important risk factor for the development of pneumonia, both in geriatric5-5 and in younger populations. Although the mechanisms of this association have not been entirely elucidated, antipsychotic-induced dysphagia likely plays a major role. Antipsychotic-induced dysphagia is a common but often unappreciated problem. Regan and colleagues, for example, reported a prevalence of 23% in a group of 60 hospitalized patients with schizophrenia, and a dose-related association between antipsychotics and dysphagia has been well established.

Healthy individuals commonly aspirate small amounts of oral secretions, but sometimes aspirate food and drink. Aspiration pneumonia ensues only when the amount of aspirate (or the virulence of the bacteria aspirated) overwhelms the patient’s defenses. Laryngeal closure during the pharyngeal phase of swallowing is a critical defence that minimizes the quantity of aspirate. It is known that antipsychotics, including second-generation agents, can profoundly affect the oral and pharyngeal phases of swallowing. Changes in the pharyngeal phase of swallowing are most predictive of aspiration and include delayed, slowed and incomplete laryngeal elevation (and therefore glottic closure); poor pharyngeal peristalsis; and pooling in the piriform sinuses with subsequent spillover into the airway.

These changes may occur even in the absence of any other signs of parkinsonism and may occur with any first- or second-generation antipsychotic. Other mechanisms, including sedation,1-7 and possibly immune dysfunction,15 have been implicated in the association with pneumonia, but drug-induced pseudo-parkinsonism is likely foremost. Although drug-induced parkinsonism is the most important mechanism leading to aspiration, it is not the only one. Dystonic reactions and tardive dyskinesia can both affect swallowing and lead to aspiration.7 Drug-induced xerostomia may lead to difficulties with swallowing and ultimately to aspiration.7 Conversely, clozapine-induced salivation is a risk factor for aspiration pneumonia. Finally, abnormal eating and swallowing habits, predominantly fast eating and taking inappropriately large boluses, have been reported in individuals with schizophrenia for more than 60 years, well before antipsychotics were in common use.7,16,17 These behaviours often lead to choking, acute asphyxia and aspiration pneumonia.

Management of patients taking antipsychotics who show evidence of aspiration or experience recurrent pneumonia begins with careful evaluation. If the cause is not obvious, evaluation by a speech pathologist can be of great help. Management of aspiration or recurrent pneumonia related to drug-induced parkinsonism usually begins with an attempt to minimize the antipsychotic dosage or to switch to an agent with less extensive dopaminergic blockade (either from a first- to a second-generation agent, or to another agent within the same class). There are numerous reports supporting this strategy, although some larger studies have suggested equal risk of pneumonia (but not necessarily of dysphagia) with first- and second-generation agents.7,12,14 There have also been reports of successful treatment with either an anticholinergic agent19 or with amantadine.11 No studies have specifically addressed feeding or dietary modifications, but most authors believe that modifications similar to those made for patients with Parkinson disease are reasonable.7 These include thickening liquids, eating fully upright, tucking the chin before swallowing, minimizing distractions and encouraging smaller bites and sips (using volume control cups, straws and utensils).

Affiliation: From the James A. Haley VA Hospital, University of South Florida College of Medicine, Tampa, Florida, USA.

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References


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