

Case Report

Respiratory dyskinesia — an under-recognized side-effect of neuroleptic medications

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Abstract

Respiratory dyskinesia is an under-recognized side effect of neuroleptic administration. There are only few studies that have addressed the prevalence of respiratory dyskinesia in patients with tardive dyskinesia. Our case report highlights the need to regularly examine patients on antipsychotics for any evidence of dyskinetic movements including respiratory musculature. Since RD is under-recognized and misdiagnosed, early detection can improve long term prognosis as treatment options are few and usually of only limited effect.

A 62-year-old Asian male, retired civil engineer, had more than 20 years history of depressive illness, developed antidepressant induced hypomania, and was given risperidone upto 1 mg per day. He developed extrapyramidal side effects as tremors, rigidity and later dyskinetic movements of lips with shortness of breathing, dyspnoea, grunting or gasping. He was referred to the pulmonologist who got the necessary medical work up done, which was normal. A diagnosis of respiratory dyskinesia was made.

Respiratory dyskinesia is an under-recognised and distressing condition that clinicians need to be aware of when treating patients with anti-psychotic medications. And also there is a need to regularly examine patients on antipsychotics for any evidence of dyskinetic movements including respiratory musculature for early diagnosis and better outcome. This case report also is worth reading for professionals of other specialties also because of the presentation of this patient, it can be easily misdiagnosed and result in poor outcome.

Keywords: Respiratory dyskinesias, Antipsychotic agents, Adverse effects.

Introduction

Respiratory dyskinesia (RD) is an under-recognized

side effect of neuroleptic administration. It manifests as irregular respiration, dyspnoea, grunting or gasping, and abnormal chest or oesophageal movements. RD occurs almost exclusively in association with other tardive effects of neuroleptics, such as tardive dyskinesia (TD) and tardive akathisia.¹ When reviewing tardive dyskinesia, textbooks and articles either fail to discuss this respiratory variant or dismiss it as rare. It may mimic other respiratory or cardiac disorders and is often overlooked or misdiagnosed.²⁻⁴

Case Report

Mr. G.W, a 62 years old male, retired civil engineer, had a +20 years history of depressive illness. He was being treated with Mianserin for last 19 years, primarily to treat his insomnia. He remained symptom free till January 2008, when, as a result of non-availability of Mianserin he experienced a relapse of his depressive symptoms as well as insomnia. His treating psychiatrist started him on Risperidone 1mg/day for insomnia, which he took for 2-3 weeks without any improvement. Consequently he experienced involuntary movements of the neck along with tremors in hands. Risperidone was therefore replaced with Quetiapine 100mg/day. In addition he was started on Sertraline 50mg/day for symptoms of depression such as sadness, decreased interest in daily activities, sleep disturbance and irritability. However as no improvement was seen, Propranolol 30mg per day was added. He took these medications till April 2008, when he discontinued the Quetiapine by himself but continued Sertraline. The tremors disappeared within 2 weeks.

In June 2008 he presented at the psychiatry outpatient clinic at Aga Khan University Hospital (AKUH) with recurrence of his depressive symptoms. Following evaluation, his Sertraline was increased up to 100mg/day. This led to improvement in his depressive symptoms but he also started showing symptoms of hypomania with elated mood, over-

spending, grandiosity and psychomotor over-activity. Hence Risperidone 1mg/day was re-introduced and Sertraline was decreased. He responded well to this regime within a week and, subsequently Risperidone was gradually reduced.

On reaching the dose of 0.5mg of Risperidone he started experiencing shortness of breath. There was no complaint of orthopnoea, paroxysmal nocturnal dyspnoea, cyanosis, or peripheral edema. He demonstrated stereotypic movements of lips and loud grunts and gasps with inspiration and expiration were observed. Significantly, his symptoms disappeared completely during sleep. The patient did not smoke, and there was no history of tuberculosis or chronic obstructive lung disease.

He was evaluated for his respiratory complaints by both a pulmonologist and an ENT specialist. On examination his respirations were very irregular with a rate of 28 per minute. Investigations including sodium, potassium, glucose, blood urea nitrogen, Creatinine, liver functions test, complete blood count, electrocardiograph, serum IgE, thyroid function tests, CT scan neck and chest, were all normal. Pulmonary function tests were attempted but could not be carried out as he could not apply his lips effectively to the apparatus due to dyskinetic movements of lips and irregular breathing. However, a six minutes walking test was carried out which was normal and there was no drop in O₂ saturation.

A provisional diagnosis of "reactive airway disease" was made and was given a trial of steroids, Montelukast, and bronchodilators. However, as no improvement was observed on follow-up visits, it was concluded that his dyspnoea and breathing difficulties was most likely due to "psychogenic causes" and he was referred for a psychiatric evaluation.

On assessment of his mental state our initial impression was that the dyspnoea and difficulty in breathing was due to anxiety, so he was started on Clonazepam 1mg/day, along with Sertraline 100mg/day. On follow-up visits there was no improvement in his condition and a provisional diagnosis of Respiratory Dyskinesia (RD) was made. In view of the fact that some patients with RD respond to small dose of antipsychotics,⁴ Risperidone 1mg/day was re-introduced. This resulted in almost immediate relief in his symptoms. However, as the respiratory symptoms re-emerged within next few weeks Risperidone was reduced and discontinued, as it is advisable in cases of TD to withdraw the antipsychotics if symptoms re-emerge or worsen.⁴

He was re-referred for a pulmonology opinion which again came out to be normal. A neurology opinion was also sought, which confirmed the diagnosis of respiratory dyskinesia and was advised Tetrabenazine 75mg/day along with Clonazepam 2 mg (titrated upto 4 mg) per day in

divided doses. This resulted in significant improvement in symptoms which also reduced the distress associated with breathing problem to some extent.

At the time of his last out-patient visit, August 2009, the patient had maintained his improvement with an almost 80% reduction in his symptoms. At that time he was on the treatment regime of Clonazepam 2mg per day, Sertraline 100mg per day and Trazadone 100mg day.

Discussion

The most common form of tardive dyskinesia (TD) involves dyskinetic movements of the orofacial and buccolingual musculature, manifesting as grimacing, facial tics, lips smacking, chewing and wormlike movements of the tongue. Involvement of the neck, axial, and extremity musculature may also occur in the form of choreo-athetoid movements, which on rare occasions may involve laryngopharyngeal and respiratory muscles.⁵ RD generally starts months or years after treatment with an antipsychotic.⁶

While the incidence of RD is not known, TD has an estimated yearly incidence of 5% among adults and as high as 25% in the elderly who receive continuous conventional antipsychotic therapy.⁴ The risk of developing TD is reported to increase with age and tends to be higher in certain ethnic groups; female gender, substance use, presence of mood disorders and early onset of EPS have also been associated with increased risk of TD.⁵ There are no clearly defined risk factors for RD, although older age and organic brain syndrome are associated with the condition.⁹

The cause of TD is uncertain, however it is thought to be secondary to up-regulation of postsynaptic dopamine receptors in the basal ganglia secondary to long term dopamine blockade. This explanation is consistent with the observation that TD may be aggravated by stopping of antipsychotic drugs or by the administration of anticholinergic anti-parkinsonian drugs.⁷

Our case report highlights the need to regularly examine patients on antipsychotics for any evidence of dyskinetic movements including respiratory musculature. Since RD is under-recognized and misdiagnosed, early detection can improve long term prognosis as treatment options are few and usually of only limited effect.

There are only few studies that have addressed the prevalence of RD in patients with tardive dyskinesia. When data from these different studies are put together, the prevalence of RD among patients with TD is on average about 16 percent though prevalence figures in individual studies range from 3-45 percent.²

The clinical presentation of our patient was very similar to the cases of RD reported in the literature,⁸⁻¹⁰

particularly the irregular pattern of breathing, tachypnoea, grunting, with worsening of the symptoms with anxiety and complete remission during sleep. Also, our patient responded to tetrabenazine and benzodiazepines as reported in other case reports.

In summary, respiratory dyskinesia is an under-recognised and distressing condition that clinicians need to be aware of when treating patients with anti-psychotic medications. There is also need for further studies to establish reliable diagnostic criterion and to establish more effective treatment options for this distressing condition.

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