Case Report- Psychosis due to Iatrogenic causes, Cerebral Palsy or Spontaneous

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Mrs X presented to Royal North Shore Hospital on 14/01/17 with dystonia. She had a background of cerebral palsy with gait disorder requiring a stick and mild dysarthria. She had previous Botulinum toxin injections as a child. She had recurrent asthma. She was 42, happily married with no children and worked in clerical capacity. She was independent in self care and ADL.

She experienced an exacerbation of asthma early October. This did not respond to inhalers and prednisone was administered. Prednisone triggered an acute psychosis, lasting approximately 2 weeks including delusional thoughts and possible auditory hallucinations. She was commenced on Olanzapine by GP for psychosis, remained on it for one and a half months. There were nil psychotic features since. Unfortunately she developed drooling, muscle stiffness and tremors. She was seen by a psychiatrist who changed her to Aripiprazole from olanzapine the week prior to Christmas 2017. There was no change in symptoms. Her stiffness and mobility deteriorated further over the next 3 weeks and she attended GP who prescribed Madopar with no effect. This worsened with torticollis and she had difficulty speaking. She could no longer walk without assistance,

On admission, she had multiple investigations to exclude other causes. In particular CT and MRI scans of the brain were normal. Examination showed Limited eye movements in all directions, no facial asymmetry. Her muscle tone was increased generally - dystonic posturing particularly of the right wrist and fingers (flexed and supinating), cervical dystonia. Sher had positive Hoffman on the right, plantar negative. She had brisk reflexes generally, particularly in the knees and her strength was 4/5 generally. She was diagnosed with Extrapyramidal side effects (EPSE) from the antipsychotics.

She had her medications of Madopar and all antipsychotics stopped and she was put on Benztropine and Baclofen for her EPSE. She had Botulinum toxin injections to the neck with improvement. She was then referred to rehab at Ryde Hospital on 24/1/2017 to try and improve mobility where she came under my care. She had persistent plantarflexion of the right ankle and subsequently had injection of these muscles on 1/2/17 but had only mild improvement. She gradually improved and was discharged home in 21/2/17.By then she was walking.

At this stage, she and her family were considering suing the GP for her psychosis not being recognised and the psychiatrist for her likely tardive dyskinesia.

On 30/3/17, she was readmitted to RNSH with psychosis that had been present for about two weeks and was worsening. -Baseline bloods on admission were unremarkable. She was initially admitted under mental health inpatient unit and then commenced on quetiapine 12.5mg BD due to less propensity for

Extrapyramidal side effects. She was then transferred back to neurology for further investigation of organic causes of psychosis: She was aware of auditory hallucinations and could walk needed assistance of one.
-MRI- normal MRI brain. No evidence of a space-occupying lesion, acute or chronic infarct or extra-axial collection.
-EEG: Normal EEG. No evidence of an epileptiform abnormality.

She continued to have auditory hallucinations whilst in hospital and in consultation with psychiatry, patient dose of quetiapine was increased to 12.5/12.5/75 and she needed nurse constantly present. Investigations showed mildly raised CSF GAD antibodies, EMG of back and lower limbs was performed to rule out stiff-person syndrome. Nil abnormalities found. Management then consisted of ongoing review by psychiatry team with titration of quetiapine. The Baclofen was weaned and ceased due to potential for psychotic side effects. Given her ongoing auditory hallucinations, antipsychotic was changed to clozapine and quetiapine weaned
She was transferred to psychiatry hospital.

Comments: Mrs X had very unusual presentation. Psychosis after prednisone initially seemed the likely cause but cannot explain the ongoing problems. Extrapyramidal side effects (EPSE) from antipsychotics are a recognised side effect but hard to initially diagnose with her cerebral palsy. No reports in the literature of increased incidence of these effects with CP. However, were these effects worse due to her background spasticity. Her EPSE did not respond as well as what is normally expected particularly as she was not on treatment for long.

Her slow speech and mild dysarthria may have delayed the onset of diagnosis of psychosis. She seems to have developed treatment resistant psychosis with hallucinations as well as the EPSE. Was the psychosis due to or triggered by prednisone, related to her CP and immature brain or was it spontaneous and unrelated?

In this case, it would seem prudent to wait for further developments and responses over time before making definitive conclusions