



ECT for refractory secondary tourettism with dermatillomania following CO poisoning

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'Secondary tourettism' is euphemistically applied to refer to tics with underlying causations or associations mostly affecting basal ganglia, contrasted with the commonest cause of tics, that is, de la Tourette syndrome [1]. Causes are legion [2] as in Table 1. Treatment modalities for tics are protean [3] with differing qualities of evidence-base. Table 2 summarizes available options. Electro-convulsive therapy (ECT) for treatment-resistant tics is limited to case reports in literature. These cases were quite complex, Tourette syndrome with comorbid major depression disorder, obsessive-compulsive disorder, or, self-injurious behaviours. Tics as catatonic signs were also reported in two cases including autism [4].

More than 30% of patients with acute carbon monoxide (CO) may experience delayed onset of neuropsychiatric sequelae including, inter alia, motor, cognitive, and, affective aftermath [5].

Data in literature speak to the idea that Hyperbaric Oxygen Therapy (HBOT) could mitigate or halt the progression of such complications [6].

Here, we are reporting a case of CO poisoning, presented to us with difficult-to-treat secondary tics and dermatillomania that responded ultimately to ECT. This could open new treatment venues in such complicated clinical scenarios.

Case report

A 36-year-old Indian fireman referred to our casualty from neurologic clinic for treatment-resistant tics and disfiguring skin-picking. He had a history of CO poisoning 3 weeks prior to the development of this movement disorder. He was treated then in ER receiving normobaric oxygen and dismissed. He had been tried sequentially on haloperidol (2.5 mg/d), but he developed acute dystonic reactions (torticollis and oculogyric crisis). He developed severe akathisia on low-dose aripiprazole (5 mg/d). He couldn't tolerate clonidine (100 µg/d) for symptomatic hypotension and bradycardia. He got oversedated on clonazepam (1 mg/d). Levetiracetam (1000 mg/d), for a

whole month, was tried, but, deemed futile and hence, was discontinued. Table 3 portrays medications chart.

There was no genetic load of neuropsychiatric illness. He was non-smoker with no history of use of illicit substances. No medical comorbidities were of relevance.

Neuro-radiology revealed bilateral hyperintense pallidal signals on MRI-T2WI. Differential [7] is shown in Table 4.

Yale-Global Tic Severity Scale (Y-GTSS) scored 85 (severe). Milwaukee Inventory for the Dimensions of Adult Skin Picking (MIDAS) read 27 for automatic subscale and 24 for focused subscale.

Across the interview, patient was markedly distressed by multiple motor (blinking, shoulder shrugging, and sniffing) and phonic tics (grunting). Trunk and arms were covered by multiple sores of excoriated skin, some were pustulated and oozing.

As the patient was demoralized with depressive overlay and endorsed passive death wishes, we opted for a course of ECT. Introducing an SSRI remained a viable option at that stage for both obsessive and depressive domains, but, the patient was too desperate and resistive to embark on a new medication trial. Modified bi-temporal ECT at 20% energy (up to 30% as sessions advanced) was administered using Thymetron device during this drug-free interlude. Six sessions were given (3 sessions/week) under general anaesthesia (using methohexital and suxamethonium) over a period of two weeks. Patient's mood soon brightened after 2 sessions only, tics significantly diminished by the 4th session, and dermatillomania totally abated by the end of sessions. Y-GTSS was readministered and read 15. MIDAS scored 2 (for each subscale).

The response was well-sustained at follow-up weeks 4, 8, 12 and 16.

On week-8, fluvoxamine and sulpride were introduced to maintain ECT response and guard against possible relapse.

Impressive ECT response in our case goes in tandem with a number of case reports in literature [8-14]. These reports, however, capitalize on de la Tourette syndrome. To our knowledge,

Table 1. Secondary tourettism.

Infections	Von Economo's encephalitis, CJD, HD
Drugs	Stimulants, L-dopa, phenytoin, antipsychotics (tardive tics)
Toxins	CO poisoning
Developmental	Static encephalopathy, MR
Others	Trauma, schizophrenia, phakomatosis, neuroacanthocytosis, degenerative, (subcortical) strokes

Table 2. Pharmacologic options in TS.

- Antipsychotics (haloperidol, pimozide, risperidone, ziprasidone, amisulpride...)
- DA depleters (tetrabenazine)
- α 2 agonists (clonidine, guanfacine)
- BDZ (clonazepam)
- Anticonvulsants (topiramate)
- Dopaminomimetics (ropinirole)

Table 3. Medications chart prior to ECT.

Drug	Dose	Duration	Reason to discontinue
Haloperidol	2.5 mg	1 week	Acute dystonias
Aripiprazole	5 mg	10 days	Severe akathisia
Clonidine	100 µg	2 days	Hypotension & Bradycardia
Clonazepam	1 mg	5 days	Oversedation
Levetiracetam	1000 mg	1 month	Lack of efficacy

Table 4. Basal Ganglia T2 hyperintensity.

Ischemic	Venous infarction
Toxic	CO poisoning
Neurodegenerative/Metabolic	Wilson disease, HD, CJD, Leigh's disease, extrapontine myelinolysis, hypoglycaemia
Neoplastic	Lymphoma

this case is one of early reports demonstrating ECT effectiveness in secondary tourettism and dermatillomania.

Although definitive mechanism remains elusive, we can theorize that modulation of monoaminergic transmission by ECT is contributory.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

Authors' contributions	AN	PS	AT
Research concept and design	--	✓	--
Collection and/or assembly of data	✓	--	--
Data analysis and interpretation	--	--	--
Writing the article	✓	✓	--
Critical revision of the article	--	--	✓
Final approval of article	--	--	✓
Statistical analysis	--	--	--

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