Sir,

Very few centers provide specialty treatment services for adolescent drug users in India. When an adolescent presents with chronic drug use, often the resultant psychosocial complications are assessed in detail, however the physical complications (unless gross or with overt manifestation) are often overlooked in general clinical settings.

Inhalant abuse is known to be especially more prevalent among children and adolescents. Toluene, present in many inhaled products, has lipophilic propensity and neurotoxic effects, leading to multifocal brain damage and peripheral neuropathies. However, very few cases of optic neuropathy have been reported. Adolescent inhalant/drug users often have co-occurring nutritional deficiencies, which may cause or enhance their vulnerability to optic neuropathies.

We discuss the case of an adolescent inhalant user, who presented to the specialty clinic at the NDDTC, AIIMS, New Delhi, and though did not have any subjective visual complaints, he was detected to have optic neuropathy, which could later be partially reversed over 3-month follow-up.

A 14-year-old male, school drop-out, from lower socioeconomic status, presented with regular use of inhalants for 2 years. Inhalant use continued largely uninterrupted (1–2 tubes/day adhesive glue) with no abstinence and was associated with significant psychosocial dysfunction. He also smoked tobacco (10–15 beedis/day) regularly with an occasional use of cannabis for 5 years. There is no significant past or family history. On examination, the patient was thinly built (BMI= 17 kg/sq.m), and pallor was noticed.

In routine physical examination, constriction of visual fields was detected on confrontation visual field examination (no subjective complaints). The patient was admitted for further assessment and management with an International Classification of Diseases-10 diagnosis of tobacco dependence syndrome (F17.2) and volatile solvent dependence syndrome (F18.2). Hemogram and biochemistry tests were within normal limits (except hemoglobin: 9.2 g/dl). Urinalysis did not reveal any opioids, benzodiazepines or cannabis. Intelligence quotient (IQ) assessment yielded a score of 84.

Referrals were sought from an ophthalmologist, otorhinolaryngologist, and neurologist. Ophthalmological examination revealed mildly sluggish pupillary reactions (visual acuity: right: 6/6, left: 6/9). Fundus showed bilateral (B/L) temporal optic disc pallor. Visual function assessment revealed normal color vision and contrast sensitivity. Humphrey’s visual field testing revealed B/L global depression of sensitivity and constricted visual fields [Figure 1]. Findings were indicative of optic neuropathy. No abnormality was detected in otorhinolaryngological examination (including pure tone audiometry) and neurological examination. Patient was prescribed an iron supplement, Vitamin B complex, Vitamin C, and coenzyme Q10. Intensive psychosocial interventions were provided and the patient was discharged after a month.

Postdischarge, patient remained abstinent from all substances (confirmed by parent’s report and repeated urinalysis) and continued taking Vitamin B complex. At 3-month follow-up, an ophthalmology consultation was resought. The disc pallor was present but the visual fields returned to near-normal [Figure 2]. The visual acuity and IQ assessment showed same findings as the previous one.

The presence of other possible conditions, for example, local trauma, genetic factors, inflammatory conditions, chemical exposure, etc., was ruled out by a thorough history and examination. The toxic cause due to regular inhalant use, along with underlying nutritional deficiencies, is likely to have caused, contributed, or enhanced the vulnerability for optic nerve damage. The patient was provided a detailed feedback of health-related harm and he continued to be in regular follow-up.

Interestingly, optic neuropathy could be reversed partially with treatment. In the few available reports...
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on inhalant users, some cases of optic neuropathy had a reversible course\(^2,3\) while others had a progressive course leading to optic atrophy.\(^4,5\) Several factors such as the dose and duration of toxic exposure and degree and type of optic nerve damage are responsible for the variable course. Treatment with vitamins, steroids, gabapentin, or cessation of toluene exposure has been described to have an improvement in visual acuity and other symptoms in toluene neurotoxicity.\(^3,5\) With continued long-term exposure, the nerve damage may be permanent with a poorer visual prognosis. Therefore, an early and timely identification of such complications is crucial in all cases of adolescent substance users.

To conclude, the case emphasizes the need to routinely examine for early optic neuropathy in all adolescent patients presenting with chronic inhalant/drug abuse, even if the subjective visual complaints are not reported.

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There are no conflicts of interest.

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Sir,

Clozapine remains the drug of choice for treatment-resistant schizophrenia and useful anti-suicidal and anti-aggressive medication in patients with schizophrenia and other psychotic disorders. It has also shown benefits for treatment of tardive dyskinesia and psychosis in patients with Parkinson's disease. [1]

Clozapine-induced agranulocytosis is a rare adverse drug event that occurs in 1% of treated individuals and can be fatal if not detected early. [2] As a result, clozapine is underused despite its superior efficacy. [2]

Previous U.S Food and Drug Administration (FDA) guideline for monitoring agranulocytosis in patient's on clozapine recommended that both white blood cell (WBC) count and absolute neutrophil count (ANC) should be done.

On September 2015, the U.S FDA announced changes to the requirements for monitoring, prescribing, dispensing, and receiving clozapine, to address continuing safety concerns and current knowledge about neutropenia. These changes are being made to address continuing concerns and current knowledge about neutropenia.

These changes include:

• FDA has clarified and enhanced the prescribing information for clozapine that explains how to monitor patients for neutropenia and manage clozapine treatment. In addition, the agency announced a new, shared risk evaluation and mitigation strategy (REMS) called the clozapine REMS program to improve the monitoring and management of patients with severe neutropenia. This program, which will require prescribers, pharmacies, and patients to enroll in a single centralized program, replaces the six existing clozapine registries maintained by individual clozapine manufacturers.
• Neutropenia will now be monitored by the ANC only, rather than in conjunction with the WBC count.
• Treatment should be interrupted if neutropenia is suspected to be clozapine-induced for ANC < 1000 cells per microliter. The requirements for ANC are modified so that patients will be able to continue clozapine treatment with a lower ANC a change that will allow continued treatment for a greater number of patients.
• Patients with benign ethnic neutropenia (ethnic groups who have low counts even before they start the treatment), who previously were not eligible for clozapine treatment, will now also be able to receive the medicine. For patients with BEN, treatment should be interrupted if neutropenia is suspected to be clozapine-induced for ANC < 500 cells per microliter.

New Food and Drug Administration Recommendations for Clozapine Prescribing and Monitoring Requirements