

OPTIC NEURITIS INDUCED BY OCCUPATIONAL EXPOSURE TO SOLVENTS – A CASE REPORT⁽¹⁾

Juliana Midori Hayashide⁽²⁾, Renata Suguimoto Vido⁽²⁾, Octávio Augusto Camilo de Oliveira⁽²⁾,
Claudia Esteban⁽³⁾, José Tarcísio Penteado Buschinelli⁽⁴⁾, Luiz Carlos Morrone⁽⁵⁾

(1) Case attended at the Specialized Outpatient Service of Occupational Medicine from Irmandade da Santa Casa de Misericórdia de São Paulo (ISCMSP)

(2) Resident Physician in Occupational Medicine from ISCMSP, (3) Toxicologist Physician, (4) Toxicologist Physician and Assistant Professor of Occupational Medicine from Faculdade de Ciências Médicas da Santa Casa de São Paulo, (5) Coordinator of Medical Residence of Occupational Medicine from ISCMSP

ABSTRACT: INTRODUCTION: Optic neuritis is manifested by loss of visual acuity and cecocentral scotoma, with variable prognosis, where even partial recovery can be observed. In workers exposed to neurotoxic chemicals, optic neuritis, excluding other non-occupational causes, should be considered a work-related disease (Group I of the Classification of Schilling).

CASE: 42-year-old male, assistant in a domestic auto body shop since January 2009, presented sudden loss of visual acuity, bilateral. He has reported no comorbidities or drug addiction, and the examination showed mydriatic pupils, fixed with reduction of photomotor response and bilateral papilledema. Forward to the laboratory exams, the main causes were excluded and the possibility of occupational cause was suggested. The team of Occupational Medicine visited the workplace and found that it has an area of 20 m², with no exhaust system, no ventilation and no windows. He worked the night before clinical onset with the door closed. He used about 6 cans of paint, and for each can of paint, used about 225 ml of paint thinner.

DISCUSSION AND CONCLUSION: Having the main causes of optic neuritis excluded and by the history of exposure to solvents on a high concentration and in a closed environment, the clinical condition may be related to the work. There are case reports in the literature like this, where the paint thinner was contaminated with methanol or methyl acetate, agents that cause optic neuritis. An analysis of sample of paint thinner that patient used was done, but those substances were not found. However, the hypothesis of occupational association is not discarded, since the absence in the country of mandatory registration of formulation of products such as glues, paint thinners and varnish can cause the composition varies even though the same brand or origin, but from different lots.

Key-words: optic neuritis, occupational exposure, solvents, automotive body repair workshop, occupational medicine

1. INTRODUCTION

Toxic optic neuropathies present the following characteristics: a history of exposure to the substance, bilateral involvement, presence of color blindness and central scotoma or important cecocentrais on the visual field^(1,2). Most cases show progressive recovery of visual acuity, but deficiencies in contrast sensitivity may be permanent^(2,3).

In workers exposed to neurotoxic chemicals, optic neuritis, when other non-occupational causes are excluded, should be considered a work-related disease, the Group I of the Classification of Schilling, in which the work is considered a necessary cause (4).

2. CASE

Patient of 42 years old, male, separated, born in João Pessoa/PB and residing in Sao Paulo/SP for 20 years, performs works of general services, such as bricklayer, painter and plumber for nearly 20 years, from 8 a.m. to 5.30 p.m..

During the night, works as assistant in an car body shop, improvised in the garage of his home since January 2009.

The night before the clinical onset, 17/11/2009, the patient had spent about five hours working on a car. He made the preparation of the car with a primer. After that, he mixed one can (800 ml) of polyurethane paint, with 225 mL of catalyst for polyurethane enamel and 225 ml of paint thinner. He used about 5-6 cans of paint to paint the car, with a compressor. As it was raining too much, he needed to close the doors and windows and with it, he mentions that the odor of paint and paint thinner was stronger than usual.

As he wasn't able to see the next morning, he was taken to the emergency department of Ophthalmology of the Hospital Santa Casa de São Paulo, complaining of loss of vision in both eyes. Ophtamological examination demonstred bilateral papilledema, with normal ocular pressure and ocular movements. Patient saw figures in the right eye and count fingers at ½ meter in the left eye. The Neurology team found ocular pain with palpation, mydriatic pupils fixed and with reduction of photomotor and consensual response, and cautious gait. The rest of the neurological examination and cranial nerves were normal.

Patient denied any underlying diseases, trauma, allergies, use of medications or drugs, alcohol and smoking. As family history, presented mother with hypertension and Diabetes Mellitus and father deceased due to heart disease at age 73. On admission, the rest of physical examination and vital signs were normal.

Laboratory tests are listed in Table 1. Lumber puncture were clear and colorless, with the following result: opening pressure = 29 mmH₂O; 3 cells/mm³ (80% lymphocytes and 20% monocytes), 10 blood cells/mm³, protein = 41 mg / dL, glucose = 82 mg / dL and Pandy cloudy.

Computed tomography scan was normal, magnetic resonance imaging showed bilateral hyperintensity of the optic nerve, and the resonance of orbit found inflammatory changes in the retrobulbar optic nerve segment, closing the diagnosis of optic neuritis.

As the tests of Table 1 were normal, and imaging tests showed no changes other than those mentioned, the main causes such as inflammatory, infectious, ischemic, nutritional deficiencies, compressive, and traumatic were ruled out. For the history of exposure to solvents before the onset of, the condition was suggested, therefore, the hypothesis of occupational origin.

During hospitalization, patient was treated with solumedrol 1 gr/day, for 3 days and prednisone 60 mg/day orally in the following days until he was discharged home. He was discharged home on dec/12th/2009, with partial improvement of visual acuity. In July/2010, counted fingers at two meters, could not see colors and could not even read, but was independent on anyone for daily life activities, living alone and coming to the clinic alone.

To a better clarity of events and substances involved, the team of Occupational Medicine made two visits to his workplace. The car body shop has an area of approximately 20 m², which holds two cars, with no exhaust system and without ventilation. The site has a single window that does not open, being closed and locked with wood siding. The only place to ventilation is a little broken piece on the wall between the door and ceiling (Figures 1 and 2). Masks and gloves are not used.

3. DISCUSSION

In the literature we found five other reports of patients who suffered episodes of acute blindness after exposure to high concentrations of organic solvents^(5,6,7,8,9). As in our case, these patients also had partial improvement in visual acuity.

The mechanism of production of toxic neuropathies is unknown, supposing that the injury occurs in axons and retinal ganglion cells⁽⁴⁾. There is no specific treatment, which increases the importance of prevention. The prognosis is variable and an improvement of visual acuity may be observed, even in the presence of atrophy of nerve fibers⁽¹⁰⁾.

Among the toxic chemicals that can potentially cause optic neuritis it is possible to include: methanol, methyl acetate, methylene chloride (dichloromethane) and other neurotoxic chlorinated solvents, methyl bromide, carbon sulfide and carbon tetrachloride^(4,15,16).

Activities of surface preparation and painting during the repair of cars present several potential risks to the health and environment, especially in domestic shops. These workers are exposed, with few security measures, to solvents, metallic pigments, resins and dyes, metal fumes, dust, noise and vibration^(11,12,13,14).

During the visit to patient's workplace, all the products that he had used had been registred. Also, a search on the Medical Sheets Safety Data (MSDS) was made. The possibility of this optic neuritis was caused by methanol, methyl acetate or methylene chloride was suggested, because they are substances that could be part of the composition, as a contaminant of paint thinners and paints.

Several brands of paint thinner were observed, but only one brand was collected to have its composition analysed. The analysis was performed at the Institute of Chemistry from University of São Paulo, by gas chromatography, and the substances found were ethanol, toluene, and butoxyethanol ethyl acetate, which does not confirm our hypothesis. However, this analysis does not discard the presence of methanol, methyl acetate, and other solvents toxic to the vision, because only one sample from one brand was collected, which was the one available when we visited the workshop, which occurred about one month after the beginning of clinical onset, when lots of products used the day before the onset of clinical status were not the same. The absence in the country of mandatory registration of formulation of products such as glues, paint

thinners and varnish can cause the variations in composition even though it is the same brand or origin, but from different lots⁽¹⁷⁾.

4. CONCLUSION

In view of the clinical characteristics, history of occupational exposure to solvents during several hours at high concentrations and in an environment without ventilation, and being the main causes of optic neuritis excluded, it is possible to conclude that the condition presented by the patient is consequence of poisoning by solvents.

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Table 1. Patient's laboratory exams results

Exams	Results	Exams	Results
Hemoglobin (g/dL)	13,5	Serum K (mEq/L)	4,4
MCV (fL)	81,9	Serum Na (mEq/L)	138
Leucocytes (mil/uL)	3,9	HBSAg	Not reagent
Platelets (mil/uL)	248	Anti-HBS	Not reagent
Urea (mg/100mL)	27	Anti-HBC total	Not reagent
Creatinin (mg/dL)	0,9	Anti-HCV	Not reagent
Glucose (mg/dL)	146	VDRL	Not reagent
INR	1,14	HIV	Not reagent
HSV (mm)	5	ANA	Not reagent
CRP	<0,4	Rheumatoid factor	Not reagent
Serum Ca (mg/100mL)	8,3		

MCV: mean corpuscular volume, INR: international normalized ratio, HSV: hemosedimentation velocity, CRP: C-reactive protein, ANA: antinuclear antibodies



Fig 1. Car being prepared to receive the paint, too close to the door, so, when it rains, patient have to close the door to continue working.



Fig 2. Front of the automotive body repair workshop. In detail (red arrow), the broken piece on the wall between the door and ceiling, being the only way of ventilation.