CASE REPORT

Limiting progressive hippocampal metabolic abnormalities after smoke inhalation injury

Edward Tobe, Basant K Pradhan

Department of Psychiatry,
Cooper Medical School of
Rowan University, Marlton,
New Jersey, USA

Correspondence to

Dr Edward Tobe,
edward.tobe@comcast.net

Accepted 6 February 2014

SUMMARY

A 46-year-old man had a smoke inhalation injury. Within 1 month, he developed neuropsychiatric problems including toxic encephalopathy, cognitive disorder, depression symptoms and personality change. From 3 to 14 years after the toxic inhalation injury, the patient received treatment with sertraline and methylphenidate. The ^{18}F -fluorodeoxyglucose positron emission tomography scan at 3 years after injury showed deterioration of glucose metabolism in the hippocampus and orbital frontal region; at 14 years after injury, the hippocampus had no significant change but the orbital frontal region had deterioration of glucose metabolism. It was hypothesised that sertraline may have provided selective hippocampal neuroprotection. Further study is justified to evaluate sertraline as a possible neuroprotective agent after smoke inhalation injury.

BACKGROUND

Toxic inhalation is common. There are >23 000 injuries and 5000–10 000 deaths in the USA annually caused by smoke inhalation.¹ People may be exposed to toxins from inhaled synthetic chemicals that may compromise intracellular neuronal metabolism.¹

In a previous report, abnormalities in glucose metabolism were noted in several brain regions in a patient who had a smoke inhalation injury.² Further discussion of this case was performed at a grand rounds presentation, and it was hypothesised that sertraline may have provided selective hippocampal neuroprotection. The purpose of the present report was to describe the additional evaluation of this case and encourage other physicians to evaluate brain pathology and attempt to prevent regional brain deterioration after smoke inhalation injury.

CASE PRESENTATION

As previously reported, a 46-year-old man had a smoke inhalation injury that caused chronic pulmonary illness, several hospitalisations for pneumonia, and obstructive sleep apnoea.² The pulmonary condition necessitated daily treatment with oral corticosteroids. The patient also developed neuropsychiatric problems including toxic encephalopathy, cognitive disorder, depression symptoms and personality change within 1 month after toxic smoke inhalation injury.

The patient previously held leadership positions at work and at two non-profit national organisations. Within 3 years after the smoke inhalation injury, he resigned from the non-profit organisations because of impaired memory and

concentration. During the next 11 years, he had no further deterioration of memory or concentration.

INVESTIGATIONS

Quantitative comparison of ^{18}F -fluorodeoxyglucose positron emission tomography scans made at 3 and 14 years after injury showed significant differences in improvement or deterioration of glucose metabolism in different regions of the brain.² Quantitative analysis of regional glucose metabolism between 3 and 14 years after the smoke inhalation injury did not support an association between regional brain connections. Within 3 years after injury, the hippocampus and orbital frontal region had significant deterioration of glucose metabolism; at 14 years after injury, the hippocampus had no significant change but the orbital frontal region had deterioration of glucose metabolism on the ^{18}F -fluorodeoxyglucose positron emission tomography scan compared with the scan at 3 years.

TREATMENT

From 3 to 14 years after the toxic inhalation injury, the patient received treatment with sertraline and methylphenidate. He also received valproic acid for 22 months, beginning at 3 years after the injury, but this was changed to topiramate because of weight gain.

OUTCOME AND FOLLOW-UP

The ^{18}F -fluorodeoxyglucose positron emission tomography scan made at 14 years after smoke inhalation injury showed that some areas of the brain improved and other areas deteriorated. The hippocampal abnormalities were unchanged on scans from 3 to 14 years after injury.

DISCUSSION

Smoke inhalation injury can produce neuropsychiatric changes, including depression, personality change and cognitive disorder. Alterations in brain glucose metabolism after smoke inhalation injury may be shown on ^{18}F -fluorodeoxyglucose positron emission tomography scans.³ Literature review showed no other published cases of quantitative evaluation of long-term improvement or deterioration of brain metabolism on ^{18}F -fluorodeoxyglucose positron emission tomography scans.

The differences in the scans at 3 and 14 years after injury may be explained by regional differences in vascularity, myelination and sensitivity to neurotoxins and exogenous steroids.²

This case was discussed at Psychiatry Grand Rounds at Cooper Medical School of Rowan

CrossMark

To cite: Tobe E, Pradhan BK. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202157

Learning points

- ▶ Toxic smoke inhalation may impair brain tissue morphology and function acutely and chronically.
- ▶ The hippocampus may facilitate memory, concentration and affect regulation.
- ▶ Further study is justified to evaluate sertraline as a neuroprotective agent for the hippocampus after toxic smoke inhalation injury.

University, including a videotaped interview of the patient. Although deterioration of the hippocampus was expected from 3 to 14 years after injury, no hippocampal deterioration was noted on the ^{18}F -fluorodeoxyglucose positron emission

tomography scans. It was hypothesised that sertraline may have provided selective hippocampal neuroprotection. Further study is justified to evaluate sertraline as a possible neuroprotective agent after smoke inhalation injury.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Rehberg S, Maybauer MO, Enkhbaatar P, *et al.* Pathophysiology, management and treatment of smoke inhalation injury. *Expert Rev Respir Med* 2009;3:283–97.
- 2 Tobe E. Progressive neuropsychiatric and brain abnormalities after smoke inhalation. *BMJ Case Rep*. Published online: 9 Aug 2012. doi:10.1136/bcr-02-2012-5945
- 3 Nibuya M, Nestler EJ, Duman RS. Chronic antidepressant administration increases the expression of cAMP response element binding protein (CREB) in rat hippocampus. *J Neurosci* 1996;16:2365–72.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow