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Delayed cognitive and psychiatric symptoms following methyl iodide and manganese poisoning: Potential for misdiagnosis

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ABSTRACT

This paper describes two patients who were exposed to toxic substances in the workplace, but for whom diagnosis proved difficult, particularly in case 2. Case 1 was exposed to methyl iodide and case 2 to manganese. Poisoning was characterised by delayed onset of symptoms following exposure and symptom progression after cessation of exposure. The clinical consequences of exposure to these substances include cerebellar and Parkinsonian symptoms followed by the development of cognitive impairment and the late appearance of psychiatric disturbances. Both cases were evaluated by physicians with little training in toxicology. Apart from abnormal liver function in case 1 and decreased power, coordination and proprioception in case 2, results of most routine medical investigations were normal. Both cases were referred for MRI brain scan and neuropsychological assessment. Abnormalities were noted on MRI but reported as being absent initially in case 1and of unknown significance in case 2. There was evidence of cognitive impairment in both and personality change in case 1 of sufficient severity to prevent both cases from returning to work and to impact on family life. There is no antidote to methyl iodide or manganese poisoning. Successful treatment requires early diagnosis and cessation of exposure, but neurotoxic syndromes are difficult to diagnose when a time lag exists between exposure and symptom onset and there is no biomarker of exposure. These syndromes may initially be confused with other neurodegenerative conditions, infectious processes, and psychiatric disorders. Clinician's lack of familiarity with the potential toxicity of environmental and industrial chemicals can lead to misdiagnosis and mismanagement, and this lack of recognition can lead to continued exposure. These cases highlight the importance of taking a detailed occupational history in patients who present with atypical neurological symptoms.

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1. Introduction

Over the last seventy years the production of industrial chemicals has increased 15 fold and concerns have been raised about the impact of these chemicals on the environment, human and animal health (American College of Obstetricians and Gynaecologists, 2013). It has been estimated that over 100,000 toxic substances are in commercial use and approximately 2,300 new chemicals are developed and submitted for registration every year [Environmental Protection Agency Office of Pollution Prevention and Toxics (OPPT), 2011; European Commission Joint Research Centre Institute for Health and Consumer Protection, 2011].The capacity of industry to produce chemical substances outstrips research which means our knowledge regarding the health effects of many substances is limited.

All of us are exposed to chemicals in everyday life, but some individuals are at greater risk of being harmed than others because of developmental or individual differences in the capacity to metabolise and detoxify certain chemicals; and/or because they are employed in occupations which require the application or manufacture of chemicals (e.g., farm workers, chemical plant workers, laboratory workers, painters etc.). Many chemicals interfere with central and peripheral nervous system function which can result in highly selective damage to particular regions or generalised disruption of many bodily systems. Neurotoxic substances can impair cognition, emotional regulation and behaviour and may do so immediately after exposure, but as we will illustrate in this paper, may produce delayed effects which appear days or weeks later. The signs and symptoms of neurotoxic damage are often non-specific and can be confused with other conditions such as degenerative neurological illnesses, infectious processes, metabolic or psychological disorders, resulting in misdiagnosis. To complicate matters further, few health care professionals in the UK receive training in toxicology and are therefore unlikely to consider a toxic cause for a patient's symptoms. Although medical training includes teaching on the potential toxicity of prescribed medicines, little time is devoted to the toxicity of industrial and environmental chemicals (Joint Royal Colleges of Physicians Training Board; Federation of the Royal Colleges of Physicians). The patients themselves are unlikely to attribute their symptoms to chemical exposure unless they have been advised of the risks associated with the products they have been working with. By the time they seek medical help, the toxicant may have been excreted from the body and objective evidence of exposure may no longer be available.

Misdiagnosis or failure to diagnose neurotoxic syndromes is common. Austin Bradford Hill, an occupational physician and epidemiologist, listed criteria to help determine causation between specific factors such as chemical exposure and injury/disease (Hill, 1965). These criteria were originally developed for research purposes but some of Hills' assumptions are made without question by healthcare professional engaged in differential diagnosis. According to Hill (1) a temporal relationship should exist between exposure and the onset of symptoms (2) a dose–response relationship should be apparent (3) removal from exposure should modify the effect (4) sound epidemiological studies should exist which have demonstrated a strong association between the risk factor and disease outcome (5) the findings must be consistent and observed in different populations in different study designs and at different times (6) the proposed cause–effect relationship must be biologically plausible and consistent with laboratory findings (7) the cause–effect relationship must be relatively specific (8) the relationship should agree with current knowledge regarding the natural history/biology of the disease (9) evidence should exist of analogous problems caused by similar agents.

The current paper describes two patients who were exposed to toxic substances in the workplace, but for whom a definitive diagnosis was not easily reached. Both cases challenge a number of Hill's criteria, particularly the notion that a temporal relationship should exist between exposure and the onset of symptoms and that removal from exposure should modify the effect (criteria 1 & 3). In both cases, there was a time lag between exposure and symptom onset and symptom progression continued long after cessation of exposure. In addition, few studies exist in the literature regarding the health effects and biological action of the chemical substances these two cases were exposed to. Case 1 was exposed to methyl iodide in a chemical manufacturing plant and was admitted to hospital suffering from slurred speech and confusion, but went on to develop cerebellar signs, which subsequently resolved, but were followed by symptoms of psychosis over ensuing weeks. Case 2 was employed by a water authority and was exposed to manganese whilst sampling water from rivers and reservoirs. He initially presented with symptoms of chronic fatigue, then developed loss of speech followed by psychiatric and then Parkinsonian symptoms. In both cases diagnosis was delayed and in case 2 a definitive diagnosis has never been reached. This paper will highlight the importance of taking a good occupational history in patients presenting with atypical neurological symptoms and illustrate the important role neuropsychologists have to play in evaluating patients and establishing an explanation for their symptoms.

2. Case reports

2.1. *Case 1: methyl iodide poisoning*

A 45 year old Caucasian man employed in a chemical manufacturing plant presented at an Accident and Emergency Department in 2012 with sudden onset of slurred speech, disorientation and confusion. Glasgow Coma Score (GCS) on admission was 13/15 and bedside cognitive testing revealed impaired cognition. He had fallen ill at work that day whilst engaged in the drying and filtering of methyl iodide. He had commenced work at the plant two weeks earlier and although personal protective clothing was provided in the form of overalls, glasses and a hard hat, he was told he would require a full face mask with filter which would need to be ordered and he was told to wear a dust mask in the interim. He reported that no person on site advised him of the danger associated with working with methyl iodide if the correct facemask and filter were not worn. He frequently suffered

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