Short Communication

iMedPub Journals
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Journal of Headache & Pain Management
ISSN 2472-1913

2018
Vol.3 No.1:2

DOI: 10.4172/2472-1913.100046

Multiple Chemical Sensitivity: A Neurotoxicity Issue

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Received: March 15, 2018; **Accepted:** March 30, 2018; **Published:** April 10, 2018

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Citation: Lange JH (2018) Multiple Chemical Sensitivity: A Neurotoxicity Issue. J Headache Pain Manag Vol.3 No.1:2.

Introduction

Various pollutants or toxicants at low concentrations have been reported to cause numerous neurological and immunological problems in sensitive individuals. These interactions have been called multiple chemical sensitivity (MCS), which exhibits very diffusive symptoms and varies among individuals in sensitivity and response [1]. Historically, MCS was associated with indoor air pollution and polluted areas. Today, this problem has been associated with the occupational environment and has been recognized as a potential occupational "injury" [2,3]. However, suggestions of a separate disease and distinct state that could be categorized as MCS has been reported and in some ways recognized since the 1950's [4]. It is likely that much of these diverse characteristics observed are a result of genetic polymorphisms and an increasing variety of chemicals in today's environment. Some of these events are also likely due to complex interactions among chemicals and gene systems; although, any relationship has not been clearly established in the literature. Findings from one study indicated there was a lack of "statistical" significance for gene activation for those experiencing MCS [5]. However, this may be due to the small number of people evaluated and the P-450 system (cytochrome system) having a major controlling influence in MCS events [6]. There may also be an interaction not recognized with cytokines and the P-450 system regarding MCS. The study observing a lack of significance [5], for increased gene activity associated with MCS did report higher cytokine levels raising the hypothesis of other unrecognized interactions being responsible for observed effects. This could mean there is no statistical significance, while a true biological significance may exist [7]. Thus, gene activation may be significantly irrelevant biologically.

Reports have suggested that some of the "insult" occurring as part of MCS is a result of interaction with the neurological system [8]. This original suggestion focused on the Olfactory-Limbic system. These concepts have expanded to other functional areas of the brain to include the bilateral putamen and hippocampus and changes in metabolism [9]. Changes in various systems of the brain have also included variation in blood flow, neurotoxicity and other modifications in brain biochemistry/structure, as observed through different forms of neurological imaging [10]. Such events when combined with impacts to the P-450 system and cytokines may increase hemicals or as a combination resulting in a multiplicative effect.

MCS may be a result of changes in susceptibility related to smell. It has been suggested that those with olfactory disorders have a higher sensitivity to environmental stress [11]. This raises the hypothesis that these changes are associated with variations in metabolic patterns in the brain, likely involving the hippocampal, amygdala and olfactory cortex areas. Such events may be multi-focal and act in a synergic fashion enhancing responses to various environmental stimuli, such as low level chemical mixtures. Thus, there may be a relationship between first being exposed to olfactory-impairing agents which result in neurological change leading to MCS when additional challenges to the same or similar agents occur [12]. This, in part, explains sensitivity to the multitude of differing chemicals and varying symptoms along with increased response from those experiencing previous exposure. Such events have been noted in the literature for decades with little explanation [13,14]. MCS may actually be a result of changes in neuro-response which eventually results in interaction among different organ systems causing a symptomology. This could be the reason why researchers have a difficulty in coordinating effects in study populations, with initial insults acting as a priming mechanism.

Traditionally, many of the cases associated with MCS were related to solvent exposure events. These types of agents have been well recognized in causing memory issues [15]. This can explain some of the neurological symptoms observed for those reporting MCS. These observations also support the mechanism of suggested degenerative neurological damage observed from positron-emission tomography studies [9].

This issue in understanding MCS has results in attempts to define and establish criteria for diagnosis [4]. One report by Rossi and Petridis suggested a multiple stage approach based on the progression of symptoms [4]. The last stage in this categorization involves evaluation of the central nervous system. Much of this determination appears to be related to the psychological and social issues associated with MCS. This has some relation to exposure events in the occupational environment; although, exposures are generally occurring at higher concentrations. These various events have resulted in claims for compensation resulting from MCS [2,3]. The issue with many of these cases is reliability in establishing a relationship between exposure (events) and MCS [16].

Establishing MCS as a successful workman's compensation claim is difficult since diagnostic criteria appear to vary among individuals and types of exposure experienced [2,17]. It is even more difficult to identify specific agents responsible for these symptoms. Most would categorize this form of disease event as an idiopathic event. Investigations have reported that about 15% of the United States population may experience issues associated with MCS, with occurrences being higher in woman [18]. The prevalence of those reporting sensitivity related to MCS appears to be increasing with a possible association with asthma [19]. Some of this increase may be attributed to changes in the global climate. Pollution is becoming a more common event worldwide [19]. Such events, even though low level, are possibly being enhanced because of changes in weather patterns, including increased temperatures. Similar events have been observed during various types of regional/local pollution occurrences [20]. Reports have suggested those with MCS experience asthma-type issues, gastrointestinal problems, headache, neurological disease characteristics and odor sensitivity to name a few, which appears to be seen during general pollution events (e.g. Gulf of Mexico Oil Spill) [15,20].

One of the major difficulties in evaluating MCS is a lack of clear diagnostic procedures [4]. When this is combined with poor exposure information and past histories of varying health events, a clear ability to identify MCS may be functionally impossible, especially when examining legal standards in defining a disease state (e.g. Daubert Rule). However, further longitudinal studies may aid in establishing a pattern of categorization for defining this disease [4]. Certainly, this type of disease will become more common and literature reports will increase pushing research to establish reliable criteria for identifying those afflicted.

Summary

In summary, it appears MCS has a relationship with the neurological system, biochemical pathways and multiplicative interaction (synergism) of chemicals. These interactions are likely the cause of confusion in attempting to identify MCS as a single "disease" entity. Most likely MCS can be best described as a multi-symptomatic state requiring interpretation of varying characteristics as methodology for diagnosis. This approach may be providing a more workable basis in establishing diagnostic criteria.

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