Elevated Blood Mercury Levels in Idiopathic Axonal Neuropathy

Mercury is an environmental neurotoxin that, in the United States, is most commonly acquired by ingestion of methylmercury in seafood.1 We report that a significant number of patients with idiopathic axonal neuropathy (IAN) have increased blood mercury levels.

Methods | The electronic records of all patients with neuropathy newly seen by one of the authors (N.L.) at the Weill Cornell Neuropathy Center from July 1, 2013, to June 30, 2014, were reviewed, with Weill Cornell Neuropathy Center institutional review board approval. Patient consent was waived as most patients were referred for a single visit and not available for follow-up. Neuropathies were classified as previously described.2,3 Blood mercury levels, determined at a commercial laboratory by mass spectroscopy, were considered elevated if greater than 10 μg/L in comparison with normal control individuals.4 Findings in patients with IAN or idiopathic small-fiber axonal neuropathy were compared with those with chronic inflammatory demyelinating neuropathy or diabetes mellitus using the χ2 test.

Results | Of 147 patients who were evaluated, 37 had IAN, 19 had diabetic neuropathy, 32 had chronic inflammatory demyelinating neuropathy, and 37 had small-fiber axonal neuropathy. Blood mercury was tested in 89.6% of the patients. The findings are presented in the Table. Mercury was elevated in 18% of patients with IAN and 9% with small-fiber axonal neuropathy (Figure) compared with none with chronic inflammatory demyelinating neuropathy or diabetic neuropathy, reaching a statistically significant difference between IAN and chronic inflammatory demyelinating neuropathy (P = .02).

Twenty-two of the 147 patients had other causes of neuropathy (2 had celiac disease, 2 had anti-myelin-associated glycoprotein antibodies, 2 had multifocal motor neuropathy, 1 had alcoholic neuropathy, 2 had hereditary neuropathy, 3 had chemotherapy-induced neuropathy, 5 had a low vitamin B12 level, 2 had a low vitamin B6 level, and 3 had a low vitamin B6 level). Of these, 1 with a low vitamin B6 level also had high mercury levels.

Discussion | Methylmercury from fish is eliminated in the gut and bile rather than the kidneys, so that blood mercury levels are a more accurate indicator of exposure.1,4 In our study, increased levels were found in a significantly higher number of patients with IAN and in some patients with small-fiber axonal neuropathy. Methylmercury toxicity is known to cause axonal neuropathy5,6; however, the concentration and duration of exposure required for neurotoxicity, or whether it caused or contributed to the neuropathy in our patients, is not known. Larger prospective studies are needed to determine whether elevated mercury levels are associated with neuropathy in other patient populations and whether dietary intervention would ameliorate the neuropathy.

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Table. Occurrence of Elevated Blood Mercury Levels in Patients With Neuropathy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Abnormal/Tested, No. (%)</th>
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<tbody>
<tr>
<td>IAN</td>
<td>6/34 (18)</td>
</tr>
<tr>
<td>Diabetic Neuropathy</td>
<td>0/16 (0)</td>
</tr>
<tr>
<td>Chronic Inflammatory Demyelinating Polyneuropathy</td>
<td>0/28 (0)</td>
</tr>
<tr>
<td>Small-Fiber Neuropathy</td>
<td>3/33 (9)</td>
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COMMENT & RESPONSE

Stroke Care Within the Golden Hour

To the Editor We read with interest the article by Ebinger and colleagues\(^1\) in which they demonstrated that the number of stroke patients treated within the golden hour can be increased up to 6 times compared with a hospital-based approach through a mobile unit staffed with a stroke neurologist and technical personnel as well as a computed tomographic scanner and point-of-care laboratory. In an editorial, Warach\(^2\) questioned the generality of this pack-and-load approach,\(^1\) which was tested in Berlin, Germany, based on financial, logistical, and clinical issues.\(^2\) In the United States, less than one-third of patients receive door-to-needle (DTN) treatment within 60 minutes,\(^3\) and only 30% of patients arrive within the golden hour. Therefore, it is not surprising that very few people receive thrombolytic therapy in less than the recommended 90-minute onset-to-treatment time,\(^3\) and even fewer in less than 60 minutes. The global situation is very different from Berlin's reality, where 31% of hospital-based patients are treated in fewer than 90 minutes onset to treatment, with a median onset-to-treatment time of 105 minutes.\(^4\)

Before investing in expensive technology, the entire process should be reviewed from time of alarm to treatment. The pack-and-load\(^1\) approach is a powerful way to reduce onset-to-treatment time in places with an effective DTN treatment time, such as in Berlin, with an average DTN time of 42.0 minutes (95% CI, 39.1-44.9).\(^4\) However, Berlin's DTN time is not the best reported. If they had invested in improving DTN time to 20 minutes, as reported in Helsinki, Finland,\(^6\) the onset-to-treatment time would be similar for the pack-and-load vs the stay-and-play approaches. Of course, every location has its own issues to understand and address. Scattered experiences demonstrating good practices for each element of the stroke survival chain point toward the possibility of achieving onset to treatment in less than 90 minutes and even 60 minutes.\(^1,5\) For locations with a reality different from Berlin, much can be done to improve the processes underlying each element of the stroke survival chain.\(^2\) Using a Lean Six Sigma approach, the entire process can be scrutinized to remove nonvalue-added steps, silos, duplicate processes, and bottlenecks to meet a new standard. Once the wastes are removed and the redesigned process is introduced, it can be optimized iteratively through continuous improvement to reduce variation and to ensure efficiency, resulting in faster execution.

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To the Editor In a study published in JAMA Neurology, Ebinger et al\(^6\) assessed the efficacy of prehospital thrombolytic therapy during the golden hour for acute ischemic stroke. The authors concluded that prehospital “thrombolysis entails no risk to the patients’ safety and is associated with better short-term outcomes.” However, limitations in the study’s methods create uncertainty in this assertion. The issue at hand is noted by the authors in the following statement, “Patients with stroke mimics who received [tissue plasminogen activator] were not included in this evaluation of treatment effects.” Thus,