PE1408/DD

Petitioner Letter of 22 February 2016

Scottish Parliament - Petition: PE 1408

Petitioner - Mrs Andrea MacArthur

22nd February 2016

I reply to the Scottish Haematology Society's comments, in response to my previous submission in November 2015, and thank them for being prepared to circulate my questions amongst their members.

I am though disappointed in that, again, the specific point I made about gastric parietal cell antibodies (PCAbs) has not been addressed. SHS say that testing for the presence of PCAbs is done routinely but then confirm that it is not used for diagnostic purposes, so why is it done at all if no notice is taken of a positive test result? This is especially relevant when the patient has tested negative for intrinsic factor antibodies (IFAbs) and the dismissal of a positive PCAbs test would make it far less likely that they receive diagnosis and treatment.

However, this was not the actual point of my question. I was seeking their explanation as to how patients with PCAbs are expected to be able to access B12 since it is the parietal cells that produce intrinsic factor, a substance vital for the absorption of B12. It is accepted that when PCAbs are present, this causes the parietal cells to atrophy, resulting in the loss of both intrinsic factor and hydrochloric acid. In this case, it would be irrelevant whether or not they had IFAbs since there would be no intrinsic factor at all. Therefore patients in this situation would actually be worse off than those who instead tested positive for IFAbs as they would have the same degree of malabsorption, due to the lack of intrinsic factor, yet they would find it more difficult to obtain a diagnosis of PA, since a positive PCAbs test would be disregarded as being '*not specific to PA*.' Whether or not it is specific to PA is not what matters – all that matters is if the presence of PCAbs prevents patients from producing intrinsic factor, thereby causing permanent PA, and existing advice from NICE [1] confirms this to be the case:

The parietal cells also produce intrinsic factor, a protein needed for absorption of vitamin B12 in the gut. Destruction of parietal cells leads to a lack of intrinsic factor.

As if that wasn't bad enough, no one with defective or absent intrinsic factor would be able to access biliary B12 either since you also need intrinsic factor for that process. I made this observation in my last submission but it was not addressed.

Since it is reckoned that around 80% of patients with PA have PCAbs, then ignoring the above finding will prevent many of them from obtaining a definite diagnosis of PA, especially if they don't have an officially deficient serum B12 level, even although the unreliability of the serum B12 test has also been admitted. The same applies to patients who, like myself, have developed permanent gastric atrophy due to long-term prescribed acid-suppressant medicine. Despite testing negative for IFAbs and PCAbs, gastric atrophy has left me with little ability to absorb nutrients. However, because patients like

me typically have low normal B12 levels, and no gastric antibodies, most find it impossible to access trial B12 injections.

I have already praised the new draft guidelines for being clear and concise and urging doctors to commence immediate treatment for those with clear relevant symptoms. However, no guidance is given to on-going management of these patients, even if they then test positive for PCAbs so, assuming they respond well to the injections, will these patients have the right to expect to continue to be treated thereafter, even if subsequent confirmatory testing proves inconclusive? Without this being specifically stated, patients have no way of challenging a withdrawal of treatment, as this is frequently what happens in reality.

There has also been no mention of the management of all those patients whose main problem is an inadequate level of maintenance injections. This was my main reason for submitting the petition in 2011 and the only acknowledgement of this problem was a brief mention in the 2014 BCSH Guidelines: [2]

Although there is little evidence that more frequent dosing is harmful, specific objective studies demonstrating clinical benefit are absent, and the GWG cannot make specific recommendations.

I'm afraid this does nothing to help the significant number of patients who find themselves deteriorating between injections. Their injection frequency may keep them alive but they cannot function for much of that period and many state that they are gaining less from each injection until it stops providing any relief, and their deterioration continues to progress. As someone who required 3 injections a week to remain stable, I feel distressed at their unnecessary suffering, which drives some to obtain B12 from other sources and learn to self-inject. This was my reason for stating the fact that several of the EU countries make injectable B12 available off prescription in pharmacies. Since the NHS feels they cannot make a recommendation for this significant group of patients, then surely the least they can do is give them a safe and convenient way of obtaining it themselves. It is perhaps not coincidental that B12 support groups encounter significantly fewer members from the countries where injectable B12 is freely available.

I urge you please to seriously consider what I have written before agreeing to introduce the new guidelines in their present form. This is too widespread a problem to risk issuing updated advice that does not meet the real needs of a large number of patients, and we are prepared to wait a while longer in order to have effective guidelines introduced rather than find the proposed one falls short of patients' needs, closing the door to our opportunity to have this situation properly resolved.

[1] https://www.nice.org.uk/advice/mib40/resources/active-b12-assay-for-diagnosing-vitamin-b12-deficiency-63499159342789

[2] http://www.bcshguidelines.com/documents/BCSH_Cobalamin_and_Folate_Guidelines_(2).docx.pdf