PE1408/AA

Petitioner Letter of 26 November 2015

Scottish Parliament - Petition: PE 1408

Petitioner - Mrs Andrea MacArthur

26th November 2015 (Addendum)

My previous comments were made prior to being given sight of the aforementioned Draft Guidelines. These have now been forwarded to me and I can now give my opinion of their content.

Overall, I am fairly impressed with what has been prepared but I do have a few issues with specific parts of the advice.

These are:

1. Testing:

a) *'Patients with clear and relevant symptoms, or blood count abnormalities, should be treated immediately (see below) and then reassessed.'*

It is not clear in what way that reassessment is to be conducted. Retesting the serum B12 level will then be of no value as oral B12 can significantly raise the serum level without necessarily providing any relief, and so this must not be used as a reason to stop treatment again. How long will they be given B12 injections, and at what frequency, before reassessment? Will any coexisting low normal, or deficient, folate level be immediately treated as a matter of course so that they have the potential to benefit from the injections they receive?

It is also normally expected that patients will respond within the 2-week loading period but experience of communicating with 1,000s of patients reveal that many of them do not notice an improvement for anything up to 4 months.

It is for the same reason that the advice issued in the BCSH Guidelines, which '*suggests review at 3 weeks*' is not helpful to those who take longer to respond. It may mean they lose their window of opportunity to be successfully treated. You will see from my previous correspondence (15/12/2014) that I have major concerns with that advice as it, in effect, cancels out the opportunity for patients to be treated for a realistic length of time before deciding whether or not the injections are addressing their symptoms.

b) *'Gastric Parietal Cell antibodies are sensitive but not specific and not recommended for diagnosing pernicious anaemia.'*

Yet the following article has this to say:

'In most cases of PA, antibodies are produced against the parietal cells causing them to atrophy, lose their ability to produce intrinsic factor, and secrete hydrochloric acid.'

http://www.fao.org/docrep/004/y2809e/y2809e0b.htm

Now surely, if 80% of PA patients have parietal cell antibodies, progressing to gastric atrophy and the loss of intrinsic factor, then it would be irrelevant whether or not they had IF antibodies, and it would also mean these patients would not therefore respond to oral B12. Neither would they be able to access their liver store since you need intrinsic factor for this process too. The same article confirms this point:

'In addition to causing malabsorption of dietary vitamin B12, PA also results in an inability to reabsorb the vitamin B12 which is secreted in the bile.'

Assuming the above information is correct, then it calls into question the whole practice of expecting patients to draw from their liver store over the course of 2-3 months since it is stated that they cannot access it at all without intrinsic factor. Despite this, patients are frequently told they have enough B12 in their liver to last them several years.

It is certainly appropriate to trial oral B12 on those who do not have either Intrinsic Factor or Parietal Cell antibodies to determine whether or not their deficiency is due to lack of hydrochloric acid. Patients obviously know themselves whether or not their deficiency could be diet-related. However, these are the only two causes that would seem to be appropriate for oral B12.

Another cause of deficiency now being encountered (with or without accompanying low serum B12 levels) is parasitic infection and it is almost impossible to obtain testing or a diagnosis. This is despite these patients showing occasional slight eosinophilia, which is simply ignored. Perhaps clinicians should be advised to pay more attention to this blood test result and arrange stool tests for those with an elevated level. However, the stool test has its limitations too and it would perhaps be appropriate to make a cestodal anthelmintic medicine available to GPs without the need for a definite diagnosis so they had the option to issue the medicine if there was good reason to suspect the presence of an intestinal tapeworm.

3. Indications for requesting serum vitamin B12 assay

Macrocytosis (MCV > 100fl)

Many people with advanced symptoms of B12 deficiency do not have an MCV above 100fl. One reason for this can be that they also have a coexisting iron deficiency, and the opposing lowering effect of this cancels out the elevation from B12/folate deficiency, thereby resulting in a false normal result. The same may happen with other levels included in a Full Blood Count. Indeed, I was in that position myself but, fortunately, my GP was prepared to give trial injections because of my advanced symptoms, and to which I had an immediate and spectacular response.

In summary, there appear to be so many different reasons for someone to develop a B12 deficiency, and not all of them able to be diagnosed, that it surely makes most sense to just give a reasonable course of trial injections to gauge response. B12 is such a safe treatment yet it is withheld in favour of much more serious medicines which cannot address the underlying cause of the symptoms.

It is also significant that some of the other EU countries (France, Germany and Spain) make injectable B12 available in pharmacies without the need for a prescription. There is no indication that this is being abused, or causing injury to anyone, so perhaps this is also something the Scottish government can look at. The problem at the moment is not that B12 is considered dangerous but that it is restricted solely because it is an injected substance.