RCPE Neurology Symposium

Tuesday 5 November 2019

What's new in the management of myasthenia gravis? – Dr Maria Farrugia

Q. Pred and Azathioprine combination is based on trial evidence, despite late 1990s. What evidence to support pred only?

A. None whatsoever. Except clinical experience has shown us that in some patients improvement occurs far earlier than what one would expect with azathioprine (takes 8-12 months to take effect). We also felt that in some patients treatment with both is clearly overkill. Aza is not without side effects. One sees horrible skin lesions (actinic keratosis and skin cancer) so we should not underestimate this drug's side effect profile. We don't know what role it has in causing malignancy including lymphoma. If one had to choose between maintenance pred 5mg vs azathioprine 75mg (reduced dose) I think one would choose prednisolone every time.

Q. What percentage of patients are able to come off steroids without requiring steroid sparing treatment?

A. Around 10-15%.

Q. How to predict MG crisis?

A. Patient's symptoms evolving quickly. not responding to treatment such as pyrido and prednisolone . Some are patients who are newly presenting and therefore undertreated or presenting explosively.

Q. Is it true that MG is more common in people with MS? What would prompt you to investigate?

A. I am not aware of this. What is true is that MG is associated commonly with other autoimmune disorders. Relevant CNS signs and symptoms would prompt investigation.

Q. Has consideration ever been given to using other high potency cholinesterase inhibitors such as donepezil or rivastigmine for myasthenia gravis?

A. No. They would not work on the neuromuscular junction.

Q. In seropositive patients, would you start prednisolone in combination with azathioprine directly or you will try to start with prednisolone first?

A. I would start pyrido and prednisolone first. I would only consider azathioprine early if there are contraindications to use steroids eg psychosis, severe osteoporosis, diabetes with complications etc.

Q. What is the maximum daily pyridostigmine dose you would use? How do you manage GI side-effects?

A. my max dose is 8 tablets x 60mg each daily. I use propantheline 15mg 15min before pyrido. Occasionally we use Loperamide too.

Q. How was the age range to classify whether MG is late or early in onset established?

A. 40-50 y is the arbitrary cut off

Q. What residual symptoms/signs would you accept when deciding to wean pyridostigmine and steroids in young patients?

A. I would accept some facial weakness including O oculi weakness. I try to wean off pyrido first (not simultaneously with steroids) to ensure that they are still not dependent on this and that symptom control is still precarious.

Q. Is there a role for treating with rituximab early in clinical course? ie DMARDS in Rheumatoid arthritis..

A. yes in musk-mg if severe from the start. If the onset is explosive (in any MG type) and they have not responded to ivIG or plasma exchange and appear refractory.

Q. Do patients with checkpoint inhibitor induced MG respond differently to Rx

A. They seem variable but there is some suggestion that they are more aggressive in presentation and do not necessarily respond as beautifully as the conventional MG patients

Q. How would a 'steroid dip' effect your treatment?

A. The steroid regime that most use is slow and gradual for that reason to avoid the steroid dip. So I start 5mg daily and increase by 5 mg every 3 rd dose ...if you start steroids at a high dose then you will definitely see a dip and in some situations this may aggravate the MG status significantly and provoke a crisis. High doses will probably work on reducing the AChR numbers (acutely) and reduce the fast twitch muscle fibre action and result in the steroid dip.

What's new in headache management? – Dr Alok Tyagi

Q. Given the placebo response rates, how important is it to consider non-pharmacological strategies?

A. The placebo responses for non-pharmacological strategies is even higher (if they do have a double blind trial to support their use as most do not).

Q. Is there a role for low dose amitriptyline or beta blockers in medication overuse headache?

A. Not in MOH due to simple analgesics or triptans.

Q. Do you routinely discuss the unlikely but possible occurrence of serotonin syndrome with patients on SSRI or SNRI who are prescribed a triptan?

A. No. In clinical practise, this is not an issue.

Physiotherapy in functional neurological disorders – Ms Paula Gardiner

Q: If patients are going to make improvements is there a typical time scale for this? Did this influence the number of sessions offered in the trial?

A: That is a hard question to answer. The sessions in the trial were based on past research showing intense treatment to have good outcomes. Therefore the trial is 9 sessions in three weeks if possible. Twice daily sessions where possible. Already I would say each patient is unique and we need to tailor to meet needs. This huge randomised control trial led by Glenn Nielson may bring light to what is needed in the future as there is a severe lack of robust research to support as yet.

Epilepsy treatments – Professor Matthew Walker

Q. What about drugs such as ethosuximide?

A. Ethosuximide is effective only in absence seizures, and is the treatment of choice for childhood absence epilepsy with only absence seizures. If a person has a generalised epilepsy with absences but also with other seizure types (eg juvenile myoclonic epilepsy) then valproate, levetiracetam and lamotrigine are better options

Q. What is the recommendation for absence seizure?

A. See above.

Q. Based on the current research on Cannabidiol would you recommend it based on the comparative success in cutting the amount of seizures in severe epilepsy, while exhibiting limited side effects?

A. Cannabidiol is only licenced as add-on for Lennox-Gastaut and Dravet syndrome and the only good randomised control data are in those conditions. It is not side-effect free and has quite significant side-effects at therapeutic doses including somnolence, decreased appetite, diarrhoea, and abnormal liver function test (especially when used in combination with valproate). It also has significant drug-drug interactions (in particular increasing the levels of clobazam).