Iranian Journal of Neurology

Clinical Note

Iran J Neurol 2016; 15(3): 180-181

Follow-up of hypertension in patients with multiple sclerosis

Received: 17 Mar 2016
Accepted: 14 May 2016

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Keywords
Multiple Sclerosis; Hypertension; Follow-up

The prevalence of hypertension is estimated over 10% in the multiple sclerosis (MS) population and increases with age.[1] In some studies comparing the prevalence of hypertension in the MS population with a comparator, hypertension was reported more commonly among these patients.[2-6] Only one cohort study reported the incidence of hypertension over a maximum follow-up of 30 years as 3.73% in patients with MS.[7]

Sometimes, new-onset hypertension could be a presenting sign of an adverse event. Transient hypertension may be an adverse event of intravenous methylprednisolone. Hypotension, a known adverse effect of interferon (INF), is a known risk factor of ischemic colitis and ischemic colitis is one of the serious adverse events of treatment with IFNs type I. Ischemic colitis should be considered in INF and acetylcholine inhibitors (AChI) and calcium channel blockers (CCB) co-administration.[8,9]

Treatment with IFN type I could predispose the patient to develop an autoimmune disease.[10]

Some reports define INF-induced de novo Raynaud's phenomenon, sometimes with progression to systemic sclerosis. A new-onset accelerated arterial hypertension could be a part of systemic sclerosis triad.[11] Similarly, new-onset hypertension could be a sign of INF-induced systemic lupus erythematosus (SLE).[12]

Thrombotic microangiopathy is a known rare adverse event of INF-therapy and new-onset hypertension is one of its important presentations advised to be evaluated carefully and controlled regularly in patients with MS receiving IFN-β.[13]

Hypertension is reported in approximately 10% of patients with MS exposed to glatiramer acetate in premarketing studies. During post marketing period, there are reports of hypertensive crisis with glatiramer acetate complicated with acute pulmonary edema and myocardial ischemic injury.[14]

Fingolimod could cause vasodilation and associated hypotension via activation of the endothelial nitric oxide synthase/nitric oxide (eNOS/NO) pathway.[15-18] As a result, in some patients experiencing a slight transient hypotension after the initiation of fingolimod therapy, it is not strange. Sometimes, this is followed by a small hypertension (~3 mmHg systolic and ~1 mm Hg diastolic blood pressure); but after 6 months of treatment, hypertension is placed in a stable plateau level.[19]

After the infusion of natalizumab and typically following two days, there are some reports of hypertension but much less frequent; this side

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effect is defined as probable and very likely.[20]

In teriflunomide trials, hypertension is reported in 3.1 and 4.3% of the patients treated with 7 or 14 mg of teriflunomide compared with 1.8% for the placebo.[21] In a phase-II teriflunomide clinical trial, high blood pressure was a cause of withdraw.[22] European medical agency recommends careful hypertension history taking and appropriate management during the treatment with teriflunomide.[23] Hypertension could be a common side effect of alemtuzumab.[24]

Up to now, there is not any information on arterial hypertension induced by dimethyl fumarate.

Essential hypertension is common in patients with MS similar to general population and probably could affect mortality, morbidity and final disability. New-onset hypertension could be a presenting sign of a treatment adverse event. MS healthcare professionals should measure and observe patients' blood pressure in follow-up visits and manage it appropriately.

Conflict of Interests

The authors declare no conflict of interest in this study.

How to cite this article: Baghbanian SM. Follow-up of hypertension in patients with multiple sclerosis. Iran J Neurol 2016; 15(3): 180-1.

References

1. Marrie RA, Reider N, Cohen J, Stuve O, Trojano M, Cutter G, et al. A systematic review of the incidence and prevalence of cardiac, cerebrovascular, and peripheral vascular disease in multiple sclerosis. Mult Scler 2015; 21(3): 318-31.
2. Kang JH, Chen YH, Lin HC. Comorbidities amongst patients with multiple sclerosis: a population-based controlled study. Eur J Neurol 2010; 17(9): 1215-9.
3. Lavela SL, Prohaska TR, Furner S, Weaver FM. Chronic diseases in male veterans with multiple sclerosis. Prev Chronic Dis 2012; 9: E55.
4. Lu E, Zhao Y, Zhu F, van der Kop ML, Synnes A, Dahlgren L, et al. Birth hospitalization in mothers with multiple sclerosis and their newborns. Neurology 2013; 80(5): 447-52.
5. Sheu JJ, Lin HC. Association between multiple sclerosis and chronic periodontitis: a population-based pilot study. Eur J Neurol 2013; 20(7): 1053-9.
6. Fuvesi J, Bencsik K, Losonczi E, Fricska-Nagy Z, Matyas K, Meszaros E, et al. Factors influencing the health-related quality of life in Hungarian multiple sclerosis patients. J Neurol Sci 2010; 293(1-2): 59-64.
7. Christiansen CF, Christensen S, Farkas DK, Miret M, Sorensen HT, Pedersen L. Risk of arterial cardiovascular diseases in patients with multiple sclerosis: a population-based cohort study. Neuroepidemiology 2010; 35(4): 267-74.
8. Salk A, Stobaugh DJ, Deepak P, Ehrenpreis ED. Ischemic colitis with type I interferons used in the treatment of hepatitis C and multiple sclerosis: an evaluation from the food and drug administration adverse event reporting system and review of the literature. Ann Pharmacother 2013; 47(4): 537-42.
9. Chang L, Kahler KH, Sarawate C, Quimbo R, Kralstein J. Assessment of potential risk factors associated with ischaemic colitis. Neurogastroenterol Motil 2008; 20(1): 36-42.
10. Mondini M, Vidali M, De Andrea M, Azzimonti B, Airo P, D'Ambrosio R, et al. A novel autoantigen to differentiate limited cutaneous systemic sclerosis from diffuse cutaneous systemic sclerosis: the interferon-inducible gene IFI16. Arthritis Rheum 2006; 54(12): 3939-44.
11. Airo' P, Scarsi M, Rossi M, Mondini M. Onset and enhancement of systemic sclerosis after treatments for multiple sclerosis. Rheumatol Int 2008; 28(7): 703-7.
12. Bahri DM, Khiari H, Essouri A, Laadhar L, Zaraa I, Mrabet A, et al. Systemic lupus erythematosus induced by interferon beta1 therapy in a patient with multiple sclerosis. Fundam Clin Pharmacol 2012; 26(2): 210-1.
13. Vosoughi R, Marriott JJ. Thrombotic microangiopathy in Interferon Beta treated multiple sclerosis patients: Review of literature and report of two new cases. Mult Scler Relat Disord 2014; 3(3): 321-5.
14. Paulino R, Samavedam S, Shi Q, Kakde A, Ravi V, Crevecoeur L. Hypertensive Crisis Causing Acute Myocardial Ischemic Injury After Subcutaneous Injection of Glatiramer Acetate. J Hosp Med 2013; 8(suppl 2).
15. Tolle M, Levkau B, Keul P, Brinkmann V, Giebing G, Schonfelder G, et al. Immunomodulator FTY720 Induces eNOS-dependent arterial vasodilatation via the lysophospholipid receptor S1P3. Circ Res 2005; 96(8): 913-20.
16. Dantas AP, Igarashi J, Michel T. Sphingosine 1-phosphate and control of vascular tone. Am J Physiol Heart Circ Physiol 2003; 284(6): H2045-H2052.
17. Nofer JR, Van der Giet M, Tolle M, Wolinska I, von Wnuck LK, Baba HA, et al. HDL induces NO-dependent vasorelaxation via the lysophospholipid receptor S1P3. J Clin Invest 2004; 113(4): 569-81.
18. Budde K, Schmouder RL, Brunkhorst R, Nashan B, Lucker PW, Mayer T, et al. First human trial of FTY720, a novel immunomodulator, in stable renal transplant patients. J Am Soc Nephrol 2002; 13(4): 1073-83.
19. EMC. Gilenya 0.5mg hard capsules [Online]. [cited 2013 Dec 4]; Available from: URL: http://www.medicines.org.uk/emc/medicine/24443/SPC/Gilenya+0.5mg+hard+capsules
20. Fragoso YD, Alves-Leon SV, Arruda WO, Carvalho MJ, Comini-Frota ER, Correa EC, et al. Natalizumab adverse events are rare in patients with multiple sclerosis. Arq Neuropsiquiatr 2013; 71(3): 137-41.
21. U.S.Food and Drug Administration. Teriflunomide (Aubagio) detailed view: Safety labeling changes approved by FDA center for drug evaluation and research (CDER) [Online]. [cited 2014]; Available from: URL: http://www.fda.gov/Safety/MedWatch/SafetyInformation/ucm423101.htm
22. O'Connor PW, Li D, Freedman MS, Bar-Or A, Rice GP, Confavreux C, et al. A Phase II study of the safety and efficacy of teriflunomide in multiple sclerosis with relapses. Neurology 2006; 66(6): 894-900.
23. European Medicines Agency. Annex I summary of product characteristics [Online]. [cited 2014 Dec 26]; Available from: URL: http://www.ema.europa.eu/docs/en_GB/document_library/EPAR_-_Product_Information/human/002514/WC500148682.pdf
24. European Medicines Agency. Annex I summary of product characteristics [Online]. [cited 2013 Dec 10]; Available from: URL: http://www.ema.europa.eu/docs/en_GB/document_library/EPAR_-_Product_Information/human/003718/WC500150521.pdf