

Dr. Mazzantini replies

To the Editor:

When a patient is diagnosed with polymyalgia rheumatica (PMR), glucocorticoid (GC) therapy is mandatory, whatever the individual's comorbidity. Strategies to reduce GC toxicity in high-risk patients include a lower starting GC dose than the usual 15–20 mg prednisolone, the association with methotrexate (MTX), the withdrawal of GC as soon as remission is achieved, a strict followup, and a pharmacological approach when appropriate (e.g., bisphosphonates to reduce fracture risk), along with general measures of prevention. With regard to MTX, to date there is no sound evidence of its ability in reducing GC-related adverse events in the long term, although its use is popular as a steroid-sparing agent in PMR, especially in difficult cases. Early treatment of PMR may be associated with a short duration of GC therapy, as suggested by Karaahmet, *et al* in their case report¹: GC were started a few weeks after disease onset and were withdrawn after a few months.

As for the cardiovascular effects of low-dose GC in chronic inflammatory diseases, our perspective is different from that of Dr. Bartoloni's group; they emphasize the potential protective effect of GC in PMR patients with regard to aortic stiffness² and vascular endothelium³ after a 1-month course of GC, but this beneficial effect is likely to be transient, lasting as long as inflammation is active. In fact, patients with PMR take most of their longterm low-dose GC when inflammation is very low or even inactive, and when the disease is not able to induce vascular damage; during this long phase, GC can be harmful for the cardiovascular system. Indeed, longterm GC therapy was associated with the development of arterial hypertension in our study⁴, and undoubtedly this can predispose to cardiovascular diseases. Similar results were found in rheumatoid arthritis⁵. There are probably only 2 ways to reduce the risk of GC-related cardiovascular adverse events: first, to shorten the GC duration/cumulative dose whenever possible, not relying on the safety of low-dose GC; and second,

to strictly monitor arterial pressure and protect the cardiovascular system with the available prevention strategies. This approach should be applied to all the conditions that require longterm low-dose GC, independently from the disease being treated.

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REFERENCES

1. Karaahmet OZ, Bal A, Unlu E, Cakci A. A difficult case of polymyalgia rheumatica [letter]. *J Rheumatol* 2012;39:1757.
2. Schillaci G, Bartoloni E, Pucci G, Pirro M, Settimi L, Alunno A, et al. Aortic stiffness is increased in polymyalgia rheumatica and improves after steroid treatment. *Ann Rheum Dis* 2012 Jan 20 [E-pub ahead of print].
3. Pirro M, Bocci EB, Di Filippo F, Schillaci G, Mannarino MR, Bagaglia F, et al. Imbalance between endothelial injury and repair in patients with polymyalgia rheumatica: Improvement with corticosteroid treatment. *J Intern Med* 2011 Dec 30 [E-pub ahead of print].
4. Mazzantini M, Torre C, Miccoli M, Baggiani A, Talarico R, Bombardieri S, et al. Adverse events during longterm low-dose glucocorticoid treatment of polymyalgia rheumatica: A retrospective study. *J Rheumatol* 2012;39:552-7.
5. Mazzantini M, Talarico R, Doveri M, Consensi A, Cazzato M, Bazzichi L, et al. Incident comorbidity among patients with rheumatoid arthritis treated or not with low-dose glucocorticoids: A retrospective study. *J Rheumatol* 2010;37:2232-6.

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