When it comes to controversy there are few subjects in rheumatology care that equal how to actually use glucocorticoids in care pathways for patients with rheumatoid arthritis (RA). From the initial euphoria inspired by the Nobel award winning discovery of cortisone over a half century ago to the bitter reality that their effect was both transient and toxicity-limiting, left clinicians in unclear waters. Their use clearly has face validity based merely on what our patients tell us, at least for a while. Their toxicity also has face validity as all clinicians, and especially rheumatologists know, based on their own experience and observations. So what are we to do?

It has recently been editorialized and suggested that combination therapy including glucocorticoids may be the new gold standard in the early treatment of RA. Kirwan points out in his editorial that low doses (i.e., < 10 mg per day of prednisone) suppresses radiographic progression when used in combination with methotrexate and reminds us that even high dose induction of 60 mg per day, as part of combination therapy as in the COBRA trial, can be equivalent to a TNF inhibitor based regimen in slowing disease. Furthermore, numerous randomized trials have also demonstrated that at low doses, patients on prednisone generally have similar or fewer side effects than the comparator. A recent randomized trial comparing the effects of a tight control regimen with or without low dose prednisone demonstrated that improvement in the prednisone group was more rapid (as would be expected) and associated with non-progression in 78% of the patients. The conclusion from these and other studies is clear, that glucocorticoids when used at low dose in combination with standard DMARDs are potent agents leading to enhanced symptom control and reduced radiographic progression with generally low and acceptable toxicity. Furthermore, as discussed by Dr. Cutolo in his webcast, advances in chronobiology have led to the development of delayed released formulations of prednisone with enhanced effectiveness that may be able to be used at even lower doses. So then, why the confusion?

Several things contribute, including personal experience with high dose glucocorticoids and their attendant toxicity; steroid induced osteoporosis before adequate prevention and treatment guidelines; and our own “official” guidelines on how to treat RA. In the 2012 ACR guidelines, it states that “despite their frequent use in RA, [review of glucocorticoids] was not part of the ACR charge or the purview of
the endeavor and were not included in the recommendations. Thus, for practicing clinicians in the United States we have little to lean on in terms of solid recommendations. EULAR on the other hand has clear guidelines and while several years old are reasonable; and the new draft treatment guidelines for 2013 do include a recommendation to consider low dose glucocorticoids as part of the initial regimen for up to 6 months then taper.

In summary there are new reasons to think about low dose glucocorticoids supported by mounting evidence and new science. Rheumatologists vote with their feet and when push comes to shove most will admit that after all of this, low dose prednisone is a cornerstone of RA care.

References


