Tetracycline has been my favourite antibiotic since 1983 ever since I started attending clinics in Government Medical College Patiala. As I was briefed by my teachers that the level of this antibiotic reaches many fold higher in gingival crevicular fluid as compared to blood. As I grew up in profession and my understanding about the subject became slightly deeper the overuse (rather misuse) of antibiotics in my practice started decreasing. Ability of the dental surgeon to physically remove the focus of infection by procedures like pulp extirpation, intra and extra coronal drainage of pus, SRP (Scaling root planning) and extraction of the tooth make the routine use of antibiotic unjustified. Now I keep my antibiotic prescription reserved only for immunocompromised patients, systemic features of infection, very severe cellulitis or space infection.

As the days rolled by I started teaching Periodontology, I was introduced to anti – collagenolytic effect of tetracycline and its members. This renewed my interest in tetracyclines especially when immunity is the hot topic for researchers, host response which is essentially protective by intent but paradoxically can result in tissue damage. The elevations in the proinflammatory or destructive mediators and enzymes (e.g IL-1, IL-6, PGE2, TNF, MMPs) in response to bacterial challenge are counterbalanced by elevations in anti-inflammatory or protective mediators such as the cytokines IL-4 and IL-10, as well as mediators such as IL-1ra (receptor antagonist) and tissue inhibitors of metalloproteinases (TIMPs). Under conditions of health the anti-inflammatory or protective mediators serve to control tissue destruction. If an imbalance occurs with excessive levels of proinflammatory or destructive mediators present in the host tissues, tissue destruction will ensue in the susceptible host. Focus of researcher is in finding agents which can down regulate the destructive aspects of the host response and up regulate the protective or regenerative responses, without actually switching off the normal defense mechanism.

Doxycyclines as well as other members of tetracycline inhibit the production of proinflammatory compounds such as cytokines, chemokines and MMPs.
They down regulates the activity of MMPs by variety of synergistic mechanisms including reduction in the cytokine levels, stimulate osteoblastic activity and new bone formation by upregulating collagen production. \[^1, 2\]

Subantimicrobial dose doxycycline (SDDI) 20mg is approved by US food and drug administration (FDA) in chronic periodontis as an adjunct to SRP. It has also been approved by FDA (Oracea) for the treatment of common skin disorder Rosacea in Sub antimicrobial dose. \[^3\]

SDDI has been proposed as a potential treatment option in various ocular conditions like ocular rosacea, meibomian gland dysfunction, recurrent corneal erosion (RCE) and chronic corneal wounds. MMPs are known to be upregulated in the cornea in patients with RCE, and doxycycline inhibits MMPs. For healing of chronic corneal wound a balance between tissue proteases and their inhibitors is required. Doxycycline significantly reduces inflammation and elevated levels of pro-inflammatory cytokine so it may be of value in the healing of chronic wounds. \[^4, 5, 6\]

In view of the above one should not be surprised if tetracycline can merge as immunomodulator with therapeutic benefits in chronic autoimmune degenerative diseases like rheumatoid arthritis that too at a remarkably affordable price.

References

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