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Underlying Mediators of Immune Dysregulation in TED

Announcer:
Welcome to CME on ReachMD. This episode is part of our MinuteCE curriculum.

Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. McGee:
Thyroid eye disease is an autoimmune condition with complex pathophysiology that is beginning to be better understood. What are the underlying regulators of TED?

This is CME on Reach MD, and I'm Dr. Selina McGee.

Dr. Smith:
I'm Dr. Terry Smith.

As someone who has been studying TED for 35 years, this is an exciting time. Pathogenic orbital fibroblasts recruited to the orbit from the bone marrow; it in turn recruit immune cells that infiltrate the orbit, leading to enhanced T cell and B cell activation and secretion of cytokines such as IL-1 beta, TNF-alpha and IL-6. The thyroid-stimulating hormone receptor, or TSHR, and the insulin-like growth factor 1 receptor, or IGF-1R, become activated by specific autoantibodies unique to Graves' disease and lead to tissue remodeling. TSHR and IGF-1R colocalize on the cell membrane and act as molecular gatekeepers to orbital fibroblast activation. Activated orbital fibroblasts differentiate into adipocytes and can cause fibrosis of orbital tissues. Agents that target IL-6 and autoantibodies may reduce inflammation and orbital changes.

Dr. McGee:
Are there other nonspecific targets for the treatment of TED? You just walked us through some very specific targets, but are there other nonspecific targets?

Dr. Smith:
The answer, Selina, is yes. The neonatal fragment crystallizable receptor, or FcRn, is an emerging target, which performs a recycling function that prevents degradation of autoantibodies targeting and activating molecules such as TSHR and IGF-1R. When FcRn is absent, there is an accelerated rate of autoantibody disposal. Thus, blocking FcRn accelerates antibody disposal and shortens the half-life of autoantibodies, along with other antibodies. FcRn function is not TED-specific.

Dr. McGee:
Thank you, Dr. Smith. That was very exciting and as a practitioner who's taking care of patients for 22 years, these are really exciting times because historically we've just managed these patients' symptoms, and we've not had targeted therapies like IGF-1R and IL-6. And then looking at nonspecific targets like FcRn, those are all things that are going to help us help our patients manage their disease process, and the more that we understand, the more we can utilize these types of therapies to improve the actual underlying disease pathology of TED instead of just managing symptoms that present with patients.

Well, that's all the time we have for today. Thank you to our audience for tuning in, and, Dr. Smith, as always, thank you for sharing our knowledge. Thank you for joining me today.

Dr. Smith:
Thank you, Selina.

Announcer:
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