Trans-Radial Coronary Interventions: A “Win-Win” for Both Patient and Operator

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**KEY WORDS:** trans-radial coronary catheterization, percutaneous coronary intervention (PCI), trans-femoral intervention, acute coronary syndrome

The main obstacle for this access is radial spasm, which in the majority of cases can be overcome by proper technique and the use of arterial vasodilators. Second, cannulation of coronary ostia can also be challenging due to anatomical variation of the great arteries of the aorta and lack of dedicated catheters. However, once mastery of the radial approach is obtained, it is difficult to conceive of a return to indiscriminate use of the femoral approach. Vascular complications are reported much less frequently with the use of the radial approach. Nonetheless, this report by Goldberg et al. [2] reminds us that no technique is infallible and that complications, while infrequent, do occur.

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**References**


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**Capsule**

**Phosphorylation of FOXP3 controls regulatory T cell function and is inhibited by TNFα in rheumatoid arthritis**

Regulatory T (Treg) cells suppress autoimmune disease, and impaired Treg cell function is associated with rheumatoid arthritis. Nie and team demonstrate that forkhead box P3 (FOXP3) transcriptional activity and, consequently, Treg cell suppressive function are regulated by phosphorylation at Ser418 in the C-terminal DNA-binding domain. In rheumatoid arthritis-derived Treg cells, the Ser418 site was specifically dephosphorylated by protein phosphatase 1 (PP1), whose expression and enzymatic activity were induced in the inflamed synovium by tumor necrosis factor-alpha (TNFα), leading to impaired Treg cell function. Moreover, TNFα-induced Treg cell dysfunction correlated with increased numbers of interleukin-17 (IL17)+ and interferon-γ (IFNγ)+CD4+ T cells within the inflamed synovium in rheumatoid arthritis. Treatment with a TNFα-specific antibody restored Treg cell function in subjects with rheumatoid arthritis, which was associated with decreased PP1 expression and increased FOXP3 phosphorylation in Treg cells. Thus, TNFα controls the balance between Treg cells and pathogenic TH17 and TH1 cells in the synovium of individuals with rheumatoid arthritis through FOXP3 dephosphorylation.


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**Capsule**

**Aggregates as a mechanistic insight into the pathogenesis of FTLD/ALS**

Several recent papers have revealed the unexpected genetic and pathological overlap between frontotemporal lobar degeneration (FTLD) and amyotrophic lateral sclerosis (ALS). The most common genetic cause is the GGGGCC hexanucleotide repeat expansion upstream of the C9orf72 coding region affecting about 10% of all patients. It is currently unknown how repeat expansion might lead to neurodegeneration. C9orf72 patients show two distinct types of ubiquitinated inclusions in the central nervous system, one of which was identified as phosphorylated TDP-43 protein. However, all inclusions in the cerebellum and most inclusions in the hippocampus and neocortex lack TDP-43, and the actual disease protein is unknown. Mori et al. discovered that most of these characteristic inclusions contain poly-(Gly-Ala) and, to a lesser extent, poly-(Gly-Pro) and poly-(Gly-Arg) dipeptide-repeat proteins that are generated by non-ATG–initiated translation from the expanded GGGGCC repeats in three reading frames. The findings yield mechanistic insight into the pathogenesis of FTLD/ALS with C9orf72 repeat expansions and directly link this common mutation to the characteristic pathology.

*Science* 2013; 339: 1335

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**Capsule**

**Lowered serum amyloid-β1-42 autoantibodies in individuals with lifetime depression**

Reduced levels of naturally occurring autoantibodies against amyloid-β (Aβ) have been described in Alzheimer's disease (AD). Lifetime depression doubles the risk of AD, thus these autoantibodies may also be reduced in this group. Maetzler and colleagues measured serum immunoglobulin G autoantibody titers against Aβ1-42, S100b and α-synuclein in 214 individuals with depression and 419 controls. Titers against Aβ1-42 were lower in individuals with lifetime depression (5544.6 ± 389.3) compared to controls (7208.7 ± 482.4, P = 0.048). Titers against S100b and α-synuclein were comparable between the cohorts. These data suggest an AD-like impairment of the humoral immune response in a relevant proportion of individuals with depression.

*J Alzheimers Dis* 2012; 32 (1): 95

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