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**Kidney injury associated with immunotherapy**

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The development of immune checkpoint inhibitors (ICIs) has driven a revolutionary change in cancer treatment. Enhancing or suppressing T cell activation via costimulatory or coinhibitory molecules modifies effector T cell response and provides “checkpoints” for immune regulation. Therapies that inhibit these checkpoint pathways therefore have the potential to enhance endogenous anti-tumor immune responses. Indeed, monoclonal antibodies directed against CTLA4 and PD-1 have demonstrated clinical efficacy against a number of cancer types, contributing to a paradigm shift in the therapeutic approach to cancer therapy. However, these ICIs can also induce off- target effects through unintentional activation of immune responses in non- target organs.

The incidence of immune-related adverse events in patients receiving ICIs ranges from 60% to 85%, with the skin, gastrointestinal tract, and liver being the most common organs affected. Acute kidney injury is one of complication of ICIs. The most common reported underlying pathology is acute tubulointerstitial nephritis, but immune complex glomerulonephritis and thrombotic microangiopathy have also been observed also been described. Early treatment with corticosteroids typically reverses kidney injury.

American Society of Clinical Oncology Clinical Practice Guideline recommend to forego kidney biopsy and to proceed directly with immunosuppressive therapy (ex, corticosteroid) if alternative causes of AKI ruled out. The dose of corticosteroid and resumption of ICIs depend on the degree of kidney function decline and recovery. In recent mutli-center retrospective data, most patients rechallenged with an ICI did not experience recurrence of ICI-AKI. Failure to recover from ICI-AKI was associated with reduced survival.