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Clinical impact of drug induced tubulointerstitial nephritis

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Drug induced tubulointerstitial nephritis is an immunologic renal injury associated with medication, and presents various kinds of renal pathologic and clinical manifestations. Drug induced interstitial nephritis is usually idiosyncratic as immune related acute and delayed hypersensitivity reaction. It begins with antigen recognition by T cells, then dendritic and T cells are interacting, and various cytokines amplify immune reaction. It is not dose related, and can re-occur when same drug is used. More than 250 of drugs were suspected to make drug induced acute or chronic interstitial nephritis such as antibiotics, NSAIDs, proton pump inhibitors, diuretics, allopurinol, anti-epileptic drugs, and chemotherapeutic agents. Some herbal remedies such as Aristolochiacaea cause interstitial nephritis and kidney injury.

The incidence of drug induced tubulointerstitial nephritis is increasing because of increased chances of medical services and increased patients age. Unfortunately, adverse drug reactions are hardly predictable, moreover, identification of the offending medicine may be complicated because of multi-drug are using simultaneously. Diagnosis of drug induced tubulointerstitial nephritis is based on its clinical and laboratory manifestation with causative drug. Clinical signs and Sx are skin rash, fever, arthralgia, and eosinophilia. Kidney biopsy shows typical morphological change. Variable numbers of macrophages and plasma cells are observed, tubules shows acute and chronic epithelial injury. Increased numbers of T cells could be expected to steroid responsiveness.

The most important issue in the treatment of drug induced tubulointerstitial nephritis is anticipation and early diagnosis. Past history of allergic reaction should be detected by physician and the patient. If kidney injury is ongoing or biopsy shows increased risk of permanent injury, steroid treatment deserves consideration. Steroids decrease T cell and eosinophil associated immune reactions then prevent interstitial inflammation. In cases of vasculitis associated interstitial nephritis, plasmapheresis would be an treatment option. For prevention, history of drug allergy must be described on the patient's medical record. Skin tests and genetic information could expect drug hypersensitivity. Desensitization would be tried only for the patients with inevitable continuity of medicine.

In conclusion, drug induced tubulointerstitial nephritis is hard to be anticipated and shows personal variability. Although genetic researches are developing for early detection of this disease, clinician's role for anticipating, detecting,

treating and preventing is more important to prevent kidney injury by drug induced tubulointerstitial nephritis.