

study as bias will be much less in community based study, though in our study, generalization to other community of India may not be possible. This may leads to the presumption that even in India, diabetes is the commonest cause of CRF /CKD as against chronic glomerulonephritis, which was being considered a commonest cause few years back. Even while comparing our own data from outpatients between two time period, (1987-98 and 1999-2004) it was found that there was increase in diabetes as cause of CRF from 28.4% to 33.6% (Unpublished data) Fig.2.

Another source of information about etiology of CKD is the pilot

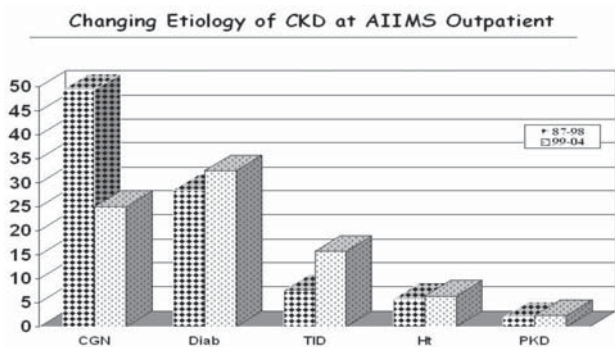


Fig.2 Changing Pattern of Etiology in Outpatient at AIIMS.

project started by Indian Society of Nephrology since June 2005, of which AIIMS was one of the member centers. This project was aimed at finding out many issues related to CKD in India, including spectrum of diseases causing CKD. It is to recall that it is also a hospital based data, though being done all over country at many centers. Till the data of 840 subjects being enrolled in this project from our center, males constituted 71% of these subjects and mean age was 47.3 years. Majority of patients were in stage CKD 3-5 groups. Diabetes mellitus as cause of CKD was seen most commonly in 29.7% patients followed by chronic glomerulonephritis and tubulointerstitial disease in 17.5%

and 11.8% respectively. Thus, in this prospective hospital-based data from our center, diabetes was commonest cause of CKD. Similar are the results of pooled data of this registry from all over the centers (Unpublished), though at present it cannot be published.

In addition to these studies, there are many more centers who are doing screening program regarding CKD in India and a multicentric study is also being conducted for CKD in collaboration with Boston medical school on the lines of 'KEEP' but the details of these ongoing studies are not yet known.

To conclude, CKD and its late stage that is end stage renal disease is a major problem for India and with increasing diabetes burden, it is going to increase further. Managing whole population of these patients will be impossible for India, where many other issues demand more priority than CKD. However, the money invested at this time in establishing prevention program for CKD in India is definitely going to give results in years to come and ultimately on long-run will still be cost effective; the saved money can be utilized for other health care programs. But, in my opinion, it is going to be difficult to convey this idea and to impress upon the current policy makers/political system of the country.

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Drug Profile

DEFLAZACORT

Pharmacodynamic Properties—Deflazacort is short acting oxazoline derivative of prednisolone; the drug offers potent anti-inflammatory & immunosuppressant actions with less side effects like diabetes, osteoporosis & lipid abnormalities.

Pharmacokinetic properties—Orally administered Deflazacort appears to be well absorbed and is immediately converted by plasma esterases to the pharmacologically active metabolite (D 21OH), which achieves peak plasma concentrations in 1.5 to 2 hours. Deflazacort is 40% protein-bound; its elimination plasma half-life is 1.1 to 1.9 hours. Elimination takes place primarily through the kidneys; 70% of the administered dose is excreted in the urine; remaining 30% is eliminated in the faeces.

Therapeutic indications - Deflazacort is indicated in wide range of indications, similar to corticosteroids such as, asthma & allergic disorders, rheumatoid arthritis, juvenile chronic arthritis, polymyalgia rheumatica, nephrotic syndrome, renal transplantation, neurological disorders.

Dosage & Administration: Deflazacort is a glucocorticoid derived from prednisolone, 6 mg of Deflazacort has approximately the same anti-inflammatory potency as 5 mg prednisolone or prednisone. For acute disorders, up to 120 mg/day Deflazacort may be needed; maintenance dose is - 3-18 mg/day.

In hepatic impairment, the dose should be adjusted to the minimum effective dose. *Renally impaired patients*, no special precautions are necessary. In *children*, the indications are the same as for adults; alternate day administration may be appropriate; dose of Deflazacort usually lies in the range 0.25-1.5 mg/kg/day. **Deflazacort withdrawal:** Deflazacort shows less HPA axis suppression; hence the only can be easily withdrawn with less risk of withdrawal symptoms. Once, a daily dose equivalent to 9 mg Deflazacort is reached, dose reduction should be slower to allow the HPA-axis to recover, particularly if more than 73 week therapy is given.

Contraindications : *Systemic infection* unless specific anti-infective therapy is employed; *hypersensitivity* to deflazacort or any of the ingredients; patients receiving *live virus immunization*.

Special warnings : (a) **Adrenal suppression** : Withdrawal must always be gradual to avoid acute

adrenal insufficiency, being tapered off over weeks or months.

(b) **Anti-inflammatory/immunosuppressive effects and infection** : Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. The clinical presentation may often be atypical and serious infections such as septicaemia and tuberculosis may be masked and may reach advanced stage before being recognized.

Special precautions : The following clinical conditions require special caution and frequent patient monitoring is necessary: (a) *Cardiac disease* or congestive heart failure (except in the presence of active rheumatic carditis), hypertension, thromboembolic disorders. Glucocorticoids can cause salt, water retention and increased excretion of potassium; dietary salt restriction and potassium supplementation may be necessary; (b) *Gastritis or oesophagitis, diverticulitis, ulcerative colitis* if there is probability of impending perforation, abscess or pyogenic infections, fresh intestinal anastomosis, active or latent peptic ulcer; (c) *Emotional instability* or psychotic tendency and epilepsy; (d) Previous *corticosteroid-induced* myopathy; (e) *Liver failure*; (f) *Hypothyroidism and cirrhosis*, which may increase glucocorticoid effect; (g) *Ocular herpes simplex* because of possible corneal perforation.

Pregnancy : Deflazacort does cross the placenta; caution should be exercised **Lactation**: Corticosteroids are excreted in breast milk, no data are available for Deflazacort.

Fluid and electrolyte disturbance: Sodium and water retention with hypertension, oedema and heart failure, potassium loss, hypokalaemic alkalosis, are less with the drug.

Drug interactions: The same precautions should be exercised as for other glucocorticoids; it is recommended to increase the maintenance dose of Deflazacort when liver enzyme inducers, are co-administered, e.g. rifampicin, rifabutin, carbamazepine, phenobarbitone, phenytoin, primidone and aminoglutethimide. For ketoconazole which inhibit liver enzymes, reduction in dose is required; in patients taking estrogens, corticosteroid requirements may be reduced. The efficacy of coumarin anticoagulants may be enhanced by concurrent corticosteroid therapy. The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication. Antacids may reduce bioavailability; leave at least 2 hrs between administration of deflazacort and antacids.