- ## Abstract:-

Through this topic will be learning a bout, Relationship between Gout and kidney disease, How Gout increase the risk of kidney Disease, and increase the level of uric acid.

Hyperuricemia and gout, the clinical manifestation of monosodium urate crystal deposition are common in patients with chronic kidney disease CKD.

While the presence of CKD poses additional challenges in gout management. effective urate lowering is possible for most patients with CKD, initial doses of urate lowering therapy are lower than in the non CKD population, while incremental dose escalation is guided by regulars monitoring of serum urate to reach the target.

management of gout flares with presently available agents can be more challenging due to potential nephrotoxicity and or contraindication in the setting of other common comorbidities.

- ## Introduction:-

Uric acid is the final oxidation product of purine metabolism and is really excreted serum uric acid levels are seen in patients with reduced glomerular filtration rate CGFRL , However in recent years , it has been proposed that uric acid itself plays a role in the pathophysiology of chronic kidney disease and possibly in acute kidney injure –

Demostrates uric acid – related cellular changes that contributes to renal disease. Thus far, it remains unclear whether these changes are reversible upon treatment of hyperuricemia, it also remains unclear whether uric acid level can be a maker of impending renal decline.

Aim of study:-

The aim of study was to determine the prevalence of gout and it’s treatment among patients with chronic kidney disease (CKD).

- ## Material and methods:-

Using the 5% random sample of Medicare claims, assessed whether gout is associated with higher risk of incident chewl CKD in adult 65 years or older, using multi variable adjusted Cox regression analyses, adjusted for demographic (age, gender, race), medical comorbidity and common medications, calculated hazard rations (CHR) and 95% confidence interval CCL). Sensitivity analyses varied comorbidity variable or limited CKD to the most specific codes.

- **Result:-**

Of the 1,699,613 eligible people, 1_68,065 developed incident CKD, 150,162 people without gout and 17,903 people with gout.

Respective crude incidence rates were 15.6 vs .78 . I per 1000 person-years.

Found that gout was associated with a higher risk of incedant CKD in multivariable _ adjusted analyses, HR was 3.05 (95% cl, 2.99, 3.10) with minimal attenuation in sensitive analyses with HR 2.96 ((95% cI, 2.91, 1.011)) and 2.59 (95% cI, 2.54, 2.63) individual charlson _ Roman ox Comorbidities plus hypertension, heart disease, obesity, coronary artery disease)).

The CKD diagnostic codes to more specific codes, confirmed findings from the main models with respective HRs of 3.03 (95% CI, 3.05, 3.15)

Table 1 :- Demograpic and clinical charactrestic of people with vs with out Gout

	No Gout CN=(1,623,304)	**Gout CN=(76,309)**
Age, Maen (SD)	75.3(7.6)	74.9(7.0)
Gender, (NC%)		
Male	762.755(41.4%)	44.740(58.6%)
Female	950.549(58.6%)	31.569(41.4%)
Heart Failure	172.489(10.6%)	14.630(19.2%)

3.03 (95% CI, 2.97, 3.08)

Table 2 :- Association of gout and other risk factor with incident CKD:-

	Multivariable- adjusted (Model 1)		**Multivariable- adjusted (Model 2)**		**Multivariable- adjusted (Model 3)**	
	HR (95%.cl)	P-value	HR (95%.cl)	P-value	HR (95%.cl)	P-value
Age (inyears)						
65-<75	Ref		Ref		Ref	
75-<85	1.62(1.60,1.63	<0.0001	1.60(1.58,1.62)	<0.0001	1.55(1.53,1.57)	0.0001
Gender						
Male	Ref		Ref		Ref	
Female	0.78(0.77,0.79)	<0.0001	0.78(0.77,0.78)	<0.0001	0.76(0.75,0.76)	<0.0001

and 2.60 (95% CI, 2.56, 2.65)

Table 3 :- Association of gout with incident CKD in pre-defined subgroup analyses by race, gender and Age:-

	Multivariable- adjusted (Model 1)	**Multivariable- adjusted (Model 1)**	**Multivariable- adjusted (Model 1)**
Gout	Black	White	Other
	3.06(2.93,3.21)	3.04(2.98m3.10)	3.13(2.91,3.37)
Gout	Female	Male	
	3.18(3.09,3.26)	2.96(2.90,3.03)	
Gout	65-75 year	75-85 year	>85 years
	3.34(3.26,3.43)	2.87(2.80,2.95)	2.66(2.54,2.80)

- **Discussion:-**

The gout was associated with higher hazard of CKD, independent of other risk factor including demographics, medical comorbidity including diabetes, hypertension, and cardio vascular disease and the use of medications for cardio vascular disease and gout an important finding was that the strength of association of gout with incident CKD changed minimally in multiple sensitivity analyses and only decreased and commonly used medications that additionally adjusted for hypertension, hyperlipidemia obesity and coronary artery disease and examined individual Carlson _Romano comorbidities.

Gout was independently associated with incident CKD in adult 65 years or older after controlling for common risk factor CKD, including hypertension, (age, race, gender,) and other medical comorbidities.

Gout is characterized by the formation of monosodium rate (MSV) crystals, which are phagocytized by macrophages or monocytes, which level to disruption of lysosome and the activation of ((NALP3)) inflammasome.

This process results in the formation of 1L-1B and other pro _inflammatory cytokines.

Innate immune system is also involved in the pathogenesis of CKD as demonstrated in the following observations, NAIP3 mRna levels were increased and correlated with renal function ona (variety) of non diabetic kidney disease and chronic kidney disease ((Activation of NAIP3)) inFlammasome and secretion of IL-1B and IL-18 causes the development of 1-u blunter initial disease in diabetic nephropathy.

- **Conclusion:-**

This study has demonstrated gout to be a risk factor for incident CKD after adjustment for (age) gender, comorbidities, deprivation, NsAID use, Frequency of hospital admission and GP at tendonce, in clinical practice, rend function monitoring is often, suboptimal in gout suggesting an area for improvement.

Further research examining the mechanisms by which gout may increase risk of CKD is suggested including the role of hyperuricemia and possible linked inflammatory processes.

- **References:-**

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