

Hepatic Steatosis and Fibrosis in Patients with Gout Using a FibroScan

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Introduction

Gout, the most common inflammatory arthritis, is manifested by persistently elevated serum urate (SU) levels associated with multiple comorbidities. Gout and elevated SU levels are associated with an increased risk for non-alcoholic fatty liver disease (NAFLD). A liver biopsy is the gold standard for evaluating fatty liver and liver fibrosis. However, due to its invasive nature, noninvasive approaches, including the FibroScan and fibrosis (FIB-4) Index formula, have been used. FibroScan scores include the Fibrosis (E) score (kPa) which measures liver stiffness and the controlled attenuation parameter dB/m (CAP) score (200 – 400) which measures degree of steatosis. FIB-4 scores <1.3 are classified as not having advanced fibrosis, while scores >2.67 are classified as having advanced fibrosis.

Aim

The aim of this study was to determine how common hepatic steatosis and fibrosis were in patients with gout using the FibroScan technology.

Methods

Our study was a retrospective chart review of patients diagnosed with gout seen in the Rheumatology clinic from 11/1/2016 - 11/12/2021 who had a FibroScan performed. We documented demographics, clinical characteristics, medications, FIB-4 scores, and FibroScan results. Descriptive and summary statistics were performed on all variables. Our Institutional Review Board approved this study.

References

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Results

47 gout patients (7 females, 14.9%; 40 males, 85.1%) were evaluated. The mean age was 59.8 years and the mean BMI was 30.95 kg/m². Tophi were present in 11 (26.2% of those with recorded information). Disease duration ranged from 0-49 years. Comorbidities included dyslipidemia (86.7%), diabetes (31.1%), hypertension (63.6%), CHF (12.8%), CAD (12.8%), chronic kidney disease (19.15%), known liver disease (33.3%) and current alcohol consumption (46.8%). 53.7% (n=29) had hyperuricemia (SU>6.8 mg/dL) and 54.4% had elevations of either ALT or AST.

Fibro Scan scores

E score (kPa) = measure of liver stiffness

| E score (kPa) | # of patients |
|----------------|---------------|
| F0 – F2 | 36 |
| F3 | 1 |
| F4 (cirrhosis) | 8 |

E Score (kPa) [$< 7 = F0-F1$, $7.1 < n < 7.8 = F2$, $7.9 < n < 10.2 = F3$, $> 10.2 = F4$]

Controlled Attenuation Parameter (CAP) = measure of steatosis

| CAP (dB/m) | # of patients |
|------------|---------------|
| <238 | 7 |
| 238 - 260 | 6 |
| 260 - 290 | 9 |
| >290 | 25 |

CAP Score [$238 < n < 260$ dB/m = S1 (11-33%), $260 < n < 290$ dB/m (34-66%), $n > 290$ dB/m = 67% or higher)

Hepatic steatosis (CAP >238 dB/m) was found in 40 (85.1%), but was not significantly different in males or females ($p=0.37$). CAP correlated with BMI ($r=0.53$, $p=0.0001$). By Fibro scan, 9 (19.1%) had evidence of fibrosis (E score >7), including 1 with moderate and 8 with severe fibrosis (cirrhosis). Moderate or severe fibrosis was significantly associated with age ($p=0.03$), and known liver disease ($p=0.003$). The Fib-4 score was significantly greater in those with severe or moderate fibrosis (3.77) versus those with no or mild fibrosis (1.59, $p=0.0045$).

Discussion

Individuals with gout are more likely to have metabolic syndrome. Metabolic syndrome is often accompanied by elevated serum urate (SU) levels and an enhanced influx of FFAs to the liver resulting in hepatic steatosis.

One study showed an independent association between gout and the risk of NAFLD, following adjustment for confounders (eg, age, sex, metabolic syndrome, CKD). The prevalence of NAFLD was higher in subjects with gout compared to those without. Mean fatty liver score was significantly higher in subjects with gout.

A significant dose-response relationship of serum uric acid (SU) with NAFLD was seen in both subjects with and without gout. The ORs for NAFLD was higher in subjects with gout compared to those without at all SU levels.

Studies have shown that insulin resistance reduces urinary uric acid excretion and increases uric acid absorption. This may partly explain why elevated SU levels are observed in patients with metabolic syndrome and NAFLD.

Further studies are warranted to elucidate the pro-inflammatory environment in patients with gout, metabolic syndrome, and NAFLD.

Conclusion

Patients with gout are commonly associated with metabolic syndrome. Although it has not received as much attention as other comorbidities, we found that most patients with gout in our cohort have hepatic steatosis, as detected via FibroScan. There was a significant positive correlation between the FibroScan and the FIB-4 results. Severe gout manifested by tophi, hyperuricemia, and long disease duration was associated with cirrhosis. To the best of our knowledge, this is the first study looking at hepatic steatosis and fibrosis in gout patients using a FibroScan. We propose that patients with gout be routinely screened for hepatic steatosis.



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