"Together, We Can Go Further"
KSES 2026
The 33rd Annual International Congress of the Korean Shoulder and Elbow Society
March 27(Fri) ~ 28(Sat), 2026
BEXCO, Busan, Korea

- Abstract No. : F-0147
- Category : Shoulder
- Detail Category : Rotator cuff

Fenofibrate attenuates rotator cuff muscle fatty infiltration via modulation of the PPARα-FABP4 pathway

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Introduction and Background

The objective of the study was to evaluate whether fenofibrate, a peroxisome proliferator-activated receptor-α(PPARα) agonist approved for the treatment of hyperlipidemia, could prevent fatty infiltration by modulating FABP4 expression in vitro and in a RCT repair rat model.

Material and Method

The expression of FABP4 under hypoxic conditions was evaluated in C2C12 myoblasts treated with fenofibrate(10 and 100 μM) using quantitative real-time polymerase chain reaction(qRT-PCR). To investigate molecular mechanisms, the expression levels of upstream regulators and adipocyte differentiation–related genes were also measured by qRT-PCR following fenofibrate treatment in the same cell line. For the in vivo study, an RCT model was established, and Fenofibrate(20mg/kg) was locally administered three times over two days following surgery. At 6weeks, supraspinatus muscle were harvested. Fatty infiltration was qualitatively and quantitatively assessed using histological analysis, and the expression of relevant metabolic and adipogenic genes was analyzed by qRT-PCR.

Results

In vitro, fenofibrate significantly downregulated FABP4 expression in a dose-dependent manner under hypoxic conditions (Fenofibrate; 10μM:0.86±0.12, $P<0.01$, 100 μM:0.32±0.06, $P<0.01$, Control; 1.12±0.17). Expression of PPARα(Fenofibrate; 10μM:2.46±0.10, $P<0.01$, 100μM:2.75±0.13, $P<0.01$, Control; 1.00±0.10), an upstream regulator of FABP4, was significantly upregulated following treatment in a similarly dose-dependent fashion. In vivo, fenofibrate-treated shoulders demonstrated marked suppression of fatty infiltration, as confirmed by both qualitative histological evaluation and quantitative fat area analysis (Fenofibrate; 6.66%±10.38%, Control; 46.38%±21.79%, $P<0.01$). Gene expression profiling revealed that PPARα was significantly upregulated (Fenofibrate; 10.65±7.02, Control; 1.00±0.62, $P<0.05$), while FABP4 expression was significantly reduced (Fenofibrate; 2.64±0.91, Control; 5.44±2.00, $P<0.05$) in the fenofibrate group compared to controls.

Conclusions

Fenofibrate reduces muscle fatty infiltration by modulating the PPARα-FABP4 metabolic pathway in the setting of rotator cuff tear. These findings support the potential of fenofibrate as a drug repositioning candidate to prevent muscle degeneration and enhance tissue quality in rotator cuff injuries.