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Altered Expression of Ubiquitin Editing Complex in Ulcerative Colitis

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Abstract : Introduction: Ulcerative Colitis (UC) is an inflammatory disease of the colon resulting from an autoimmune response towards individual's own microbiota. Excessive inflammation is characterized by hyper-activation of NFkB, a transcription factor regulating expression of various pro-inflammatory genes. The ubiquitin editing complex consisting of TNFAIP3, ITCH, RNF11 and TAX1BP1 maintains homeostatic levels of active NFkB through feedback inhibition and assembles in response to various stimuli that activate NFkB. TNFAIP3 deubiquitinates key signaling molecules involved in NFkB activation pathway. ITCH, RNF11 and TAX1BP1 provide substrate specificity, acting as adaptors for TNFAIP3 function. Aim: This study aimed to find expression of members of the ubiquitin editing complex at the transcript level in inflamed colon tissues of UC patients. Materials and Methods: Colonic biopsy samples were collected from 30 UC patients recruited at Department of Gastroenterology, AIIMS (New Delhi). Control group (n= 10) consisted of individuals undergoing examination for functional disorders. Real Time PCR was used to determine relative expression with GAPDH as housekeeping gene. Results: Expression of members of the ubiquitin editing complex was significantly altered during active disease. Expression of TNFAIP3 was upregulated while concomitant decrease in expression of ITCH, RNF11, TAX1BP1 was seen in UC patients. Discussion: This study reveals that increase in expression of TNFAIP3 was unable to control inflammation during active UC. Further, insufficient upregulation of ITCH, RNF11, TAX1BP1 may limit the formation of the ubiquitin complex and contribute to pathogenesis of UC.

Keywords : altered expression, inflammation, ubiquitin editing complex, ulcerative colitis
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