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KIM-1 Downregulation and Histological Recovery Following Combined Terminalia chebula and Annona muricata Administration in a Zebrafish Nephrotoxicity Model

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Objectives : Drug-induced nephrotoxicity remains a major barrier to safe antimicrobial therapy. This study evaluated whether a combined extract of Terminalia chebula and Annona muricata (TCAM) mitigates gentamicin induced renal injury in zebrafish using kidney histopathology and KIM-1 expression, alongside phytochemical profiling.
Methods : TCAM phytochemical constituents were profiled by high performance liquid chromatography (HPLC). Seventy two zebrafish (Danio rerio) were allocated into six groups: control, gentamicin only, TCAM only, and gentamicin plus TCAM at 50, 100, or 200 mg/kg. Renal damage was evaluated by kidney histopathology, and KIM-1 gene expression was assessed using gel electrophoresis based analysis.
Results : HPLC identified quercetin, corilagin, gallic acid, chebulagic acid, and ellagic acid in TCAM. Gentamicin induced pronounced tubular injury, whereas TCAM alone showed no observable renal damage. In gentamicin challenged fish, TCAM co-treatment reduced tubular necrosis and improved renal tissue architecture across doses, with greater improvement at higher doses. KIM-1 expression increased in the gentamicin group and was substantially reduced in TCAM treated groups, consistent with attenuated kidney injury.
Conclusions : TCAM confers nephroprotection in a zebrafish gentamicin model, supported by improved renal histology and reduced KIM-1 expression, with phytochemical constituents identified by HPLC. These findings justify further mechanistic studies and translational evaluation of TCAM for mitigating drug-induced nephrotoxicity.