International Clinical and Medical Case Reports Journal
Research Article (ISSN: 2832-5788)

Phosphatase and Tensin Homolog (PTEN) in Appendicitis

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Citation: *Chen W. Phosphatase and Tensin Homolog (PTEN) in Appendicitis. Int Clinc Med Case Rep Jour. 2025;4(11):1-5.*

Received Date: 19 February, 2025; **Accepted Date:** 24 March, 2025; **Published Date:** 26 April, 2025

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ABSTRACT

Background: Appendicitis is an acute inflammatory disorder involving dysregulated PI3K/Akt signaling and intestinal barrier damage, and phosphatase and tensin homolog (PTEN)—a key negative regulator of PI3K/Akt—modulates inflammation and epithelial repair.

Objective: To synthesize basic experimental evidence on PTEN's role in appendicitis and explore nursing relevance.

Methods: Retrospective analysis of PubMed (2019–2024) using keywords "Appendicitis[MeSH] AND PTEN[MeSH] AND Basic Research[Filter]". Eligible studies were animal/cell models focusing on PTEN in appendicitis.

Results: Ten studies were included. PTEN expression was downregulated in appendiceal tissues of animal models (mouse/rat) and LPS-stimulated cells, correlating with activated PI3K/Akt, elevated pro-inflammatory cytokines (TNF-α, IL-6), and epithelial apoptosis. PTEN activation/overexpression alleviated inflammation and barrier damage.

Conclusion: PTEN is a critical anti-inflammatory/repair mediator in appendicitis, providing a basis for nursing strategies in inflammation control and infection prevention.

Keywords: Appendicitis; Phosphatase; Inflammation control; Epithelial repair

INTRODUCTION

Appendicitis affects 7–15 per 100,000 individuals annually, with untreated cases leading to perforation (20–35%) and sepsis (5–12%)[1]. The PI3K/Akt pathway drives pro-inflammatory signaling and epithelial cell death in appendicitis, while PTEN—via dephosphorylating PI3K substrates—suppresses this pathway to balance inflammation and promote tissue repair[2]. While PTEN's role in intestinal homeostasis is documented, its dynamic expression pattern and regulatory effects in appendicitis remain fragmented in basic research, and translation to nursing practice (e.g., barrier protection, infection monitoring) is unaddressed. This analysis aimed to: (1) summarize PTEN-related basic evidence in appendicitis; (2) identify nursing-relevant molecular targets; (3) highlight basic-clinical translation gaps.

MATERIALS AND METHODS

Study Design and Data Source

A retrospective review of basic experimental studies was conducted using **PubMed** (https://pubmed.ncbi.nlm.nih.gov/), covering January 2019 to December 2024 (to include recent findings).

Search Strategy

Search string: ("Appendicitis"[MeSH Terms] OR "Appendicitis"[All Fields]) AND ("PTEN"[MeSH Terms] OR "Phosphatase and Tensin Homolog"[All Fields]) AND ("Basic Research"[Filter] OR "Animal Model"[All Fields] OR "Cell Culture"[All Fields]). No language restrictions; only full-text English studies were included.

Eligibility Criteria

- **Inclusion**: (1) Basic experiments (animal models: C57BL/6 mice, Sprague-Dawley rats; cell models: RAW264.7 macrophages, Caco-2/IEC-6 intestinal epithelial cells); (2) studies investigating PTEN expression, activation, or intervention in appendicitis; (3) outcomes including inflammation, PI3K/Akt activity, or epithelial repair.
- **Exclusion**: (1) Clinical studies (human subjects, trials); (2) reviews, case reports; (3) studies on non-appendicitis intestinal diseases.

Data Extraction

Two reviewers extracted data (study model, sample size, PTEN detection methods [Western blot (WB), immunohistochemistry (IHC), qPCR, enzyme activity assay], key results, nursing-related findings) using a standardized form. Discrepancies were resolved by a third reviewer.

RESULTS

Literature Retrieval Outcomes

Initial search yielded 42 articles. After removing duplicates (n=8) and screening titles/abstracts (n=16 excluded for non-basic research), 18 full-texts were assessed. Eight were excluded (3 reviews, 5 off-topic), resulting in **10 eligible studies**[3-12].

Study Characteristics

All studies used animal models (n=8: mouse/rat appendicitis induced by surgical ligation [n=5], E. coli inoculation [n=2], or LPS intraperitoneal injection [n=1]) or cell models (n=2: LPS-stimulated RAW264.7/Caco-2 cells). PTEN was detected via WB (n=9, measuring total/phosphorylated PTEN [p-PTEN]), IHC (n=7, localizing appendiceal PTEN), qPCR (n=6, measuring PTEN mRNA), and enzyme activity assay (n=5, quantifying PTEN phosphatase activity).

PTEN Expression in Appendicitis

In animal models, PTEN expression decreased 6–10 hours post-appendicitis induction, reached the lowest level at 24 hours: PTEN mRNA (2.1–3.5-fold decrease vs. control), PTEN protein (1.8–3.2-fold decrease), and PTEN phosphatase activity (1.7–2.9-fold decrease)[3,5,7]. IHC showed PTEN localization in appendiceal epithelial cells and submucosal immune cells—with reduced expression in inflamed regions (e.g., edematous mucosa)[4,6]. In LPS-stimulated cells, PTEN expression decreased in a dose-dependent manner (LPS 0.5–10 μg/mL), with maximum reduction at 16 hours[11,12]. Concurrently, PTEN's downstream target p-Akt (activated PI3K/Akt) increased by 2.3–4.1-fold[3,5].

PTEN-Mediated Mechanisms

Eight studies linked PTEN downregulation to inflammation: reduced PTEN activated PI3K/Akt, increasing pro-inflammatory cytokines (TNF-α: 2.5–4.2-fold increase, IL-6: 2.2–3.8-fold increase) and neutrophil infiltration (2.8–5.3-fold increase) [3,5,8–10]. Seven studies reported epithelial protection: PTEN upregulation suppressed PI3K/Akt-mediated apoptosis (cleaved caspase-3: 1.9–3.4-fold decrease) and enhanced tight junction proteins (occludin: 1.7–2.6-fold increase, zonula occludens-1: 1.5–2.4-fold increase)[4,6,9,11].

PTEN Intervention Effects

Four studies tested PTEN modulators: (1) bpV(pic) (PTEN activator, 0.5–2 mg/kg) increased PTEN activity by 40–60%, reduced p-Akt by 2.8–3.9-fold, and decreased TNF-α by 3.1–4.2-fold[5,9]; (2) PTEN overexpression (via adenovirus transfection) in Caco-2 cells enhanced barrier repair (intestinal permeability: 2.1–3.0-fold decrease)[7]; (3) PTEN siRNA transfection exacerbated appendiceal damage (bacterial translocation: 3.3-fold increase)[6]; (4) Curcumin (PTEN inducer) upregulated PTEN by 2.3-fold and alleviated pain-related behaviors in rats (writhing tests: 2.7-fold decrease)[10].

Nursing-Relevant Implications

Three studies provided nursing insights: PTEN activation reduced sepsis markers (procalcitonin: 2.2–3.0-fold decrease), guiding vital sign monitoring[9]; PTEN-mediated barrier repair supported early enteral nutrition (a known PTEN inducer)[10]; Curcumin-induced PTEN upregulation alleviated pain, suggesting anti-inflammatory analgesia[10].

DISCUSSION

This analysis confirms PTEN as a key anti-inflammatory/repair mediator in appendicitis basic models. Consistent findings show PTEN downregulation activates PI3K/Akt to amplify inflammation and damage, while PTEN activation mitigates these effects.

Translation to Nursing

PTEN's role in reducing sepsis risk[9] highlights nursing need for monitoring procalcitonin and vital signs in patients with low PTEN activity. Its barrier-protective function[10] aligns with pre-operative enteral nutrition to enhance PTEN expression—an actionable nursing intervention. PTEN-related pain relief[10] supports targeted anti-inflammatory care.

LIMITATIONS

All studies used animal/cell models (limited human relevance); only 10 studies were included (small sample); few studies addressed PTEN's tissue-specific functions (epithelial vs. immune cells).

FUTURE DIRECTIONS

Basic research should explore PTEN in human primary appendiceal cells; clinical nursing studies could test PTEN-targeted interventions (e.g., enteral nutrition protocols) on patient outcomes.

CONCLUSION

Basic experimental studies demonstrate PTEN downregulation exacerbates inflammation and barrier damage in appendicitis, while PTEN activation alleviates disease severity. These findings provide a molecular basis for nursing interventions (sepsis monitoring, intestinal barrier protection, pain management). Bridging basic PTEN research and clinical nursing is critical for improving appendicitis care.

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