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VCAM-1Analysis of Basic Experimental Studies and Nursing Implications

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ABSTRACT

Background: Appendicitis is an acute inflammatory disorder driven by leukocyte infiltration and vascular dysfunction, and vascular cell adhesion molecule-1 (VCAM-1)—a key adhesion molecule—mediates leukocyte recruitment and amplifies inflammatory responses.

Objective: To synthesize basic experimental evidence on VCAM-1's role in appendicitis and explore its nursing relevance.

Methods: Retrospective analysis of PubMed (2019–2024) using keywords "Appendicitis[MeSH] AND VCAM-1[MeSH] AND Basic Research[Filter]". Eligible studies were animal/cell models focusing on VCAM-1 in appendicitis.

Results: Eleven studies were included. VCAM-1 expression was upregulated in appendiceal vascular endothelium of animal models (mouse/rat) and LPS-stimulated endothelial cells, correlating with increased leukocyte infiltration and vascular permeability. VCAM-1 inhibition alleviated appendiceal inflammation and tissue damage.

Conclusion: VCAM-1 is critical for inflammatory progression in appendicitis, providing a molecular basis for nursing strategies in inflammation monitoring and infection prevention.

Keywords: Appendicitis; Endothelial cells; Vascular permeability; Leukocyte infiltration

INTRODUCTION

Appendicitis affects 7–15 per 100,000 individuals annually, with untreated cases leading to perforation (20–35%) and sepsis (5–12%)[1]. Leukocyte recruitment to the appendiceal wall—mediated by adhesion molecules—

is a core pathogenic step, and VCAM-1 (expressed on vascular endothelium) specifically binds to leukocyte integrins (e.g., VLA-4) to drive neutrophil/monocyte infiltration[2]. While VCAM-1's role in inflammatory diseases (e.g., sepsis, inflammatory bowel disease) is well documented, its dynamic expression and regulatory effects in appendicitis remain fragmented in basic research, and translation to nursing practice (e.g., inflammation severity assessment, pain management) is unaddressed. This analysis aimed to: (1) summarize VCAM-1-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 July 2024 (to include recent findings).

Search Strategy

Search string: ("Appendicitis"[MeSH Terms] OR "Appendicitis"[All Fields]) AND ("VCAM-1"[MeSH Terms] OR "Vascular Cell Adhesion Molecule-1"[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: HUVECs, EA.hy926 endothelial cells, RAW264.7 macrophages); (2) studies investigating VCAM-1 expression, regulation, or intervention in appendicitis; (3) outcomes including leukocyte infiltration, vascular permeability, or inflammation.
- **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, VCAM-1 detection methods [immunohistochemistry (IHC), Western blot, qPCR, flow cytometry], key results, nursing-related findings) using a standardized form. Discrepancies were resolved by a third reviewer.

RESULTS

Literature Retrieval Outcomes

Initial search yielded 48 articles. After removing duplicates (n=10) and screening titles/abstracts (n=20 excluded for non-basic research), 18 full-texts were assessed. Seven were excluded (3 reviews, 4 off-topic), resulting in **11 eligible studies**[3-13].

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=3: LPS-stimulated HUVECs/EA.hy926 cells). VCAM-1 was detected via IHC (n=9, localizing vascular endothelium), Western blot (n=8, measuring protein expression), qPCR (n=7, measuring mRNA levels), and flow cytometry (n=4, quantifying endothelial VCAM-1).

VCAM-1 Expression in Appendicitis

In animal models, VCAM-1 expression increased 4–8 hours post-appendicitis induction, peaked at 24 hours (appendiceal VCAM-1 mRNA: 2.3–4.1-fold increase vs. control; protein: 1.9–3.5-fold increase), and gradually declined by 36 hours[3,5,7]. IHC showed VCAM-1 upregulation in appendiceal submucosal vascular endothelium, with co-localization of adherent neutrophils (3.2–5.7-fold increase vs. control)[4,6]. In LPS-stimulated endothelial cells, VCAM-1 expression increased in a dose-dependent manner (LPS 0.1–10 μg/mL), with maximum upregulation at 12 hours[11,12].

VCAM-1-Mediated Mechanisms

Nine studies linked VCAM-1 upregulation to inflammatory progression: VCAM-1 overexpression correlated with increased leukocyte infiltration (neutrophils: 2.8–4.5-fold increase, monocytes: 2.1–3.3-fold increase) and vascular permeability (assessed via Evans blue extravasation: 1.8–3.1-fold increase vs. control)[3,5,8-10]. Seven studies reported that VCAM-1 enhanced inflammation amplification: higher VCAM-1 levels were associated with elevated pro-inflammatory cytokines (TNF-α: 1.7–3.0-fold increase, IL-6: 1.5–2.8-fold increase) via activating leukocyte-derived NF-κB[4,6,9,11].

VCAM-1 Intervention Effects

Four studies tested VCAM-1 modulators: (1) anti-VCAM-1 neutralizing antibody (10–20 μg/kg) reduced leukocyte adhesion by 40–55% and appendiceal edema by 35–50%[5,9]; (2) VCAM-1 siRNA transfection in HUVECs suppressed LPS-induced VCAM-1 expression by 60–70% and reduced monocyte adhesion[13]; (3) VCAM-1 knockout mice showed 3.2-fold lower neutrophil infiltration and 2.7-fold lower TNF-α levels vs. wild-type appendicitis models[7]; (4) statins (atorvastatin, a VCAM-1 inhibitor) downregulated VCAM-1 by 2.1-fold and alleviated pain-related behaviors in rats[10].

Nursing-Relevant Implications

Three studies provided nursing insights: VCAM-1 upregulation correlated with appendiceal inflammation severity (higher VCAM-1 = increased perforation risk)[9]; anti-VCAM-1 antibody reduced bacterial translocation

(a sepsis risk factor)[8]; statin-mediated VCAM-1 inhibition alleviated abdominal pain, supporting targeted pain management1[0]—all guiding nursing focus on inflammation monitoring, sepsis prevention, and pain assessment.

DISCUSSION

This analysis confirms VCAM-1 as a key mediator of leukocyte recruitment and inflammation amplification in appendicitis basic models. Consistent findings show VCAM-1 upregulation drives tissue damage, while VCAM-1 inhibition mitigates these effects.

Translation to Nursing

VCAM-1's correlation with inflammation severity[9] highlights nursing need for monitoring surrogate markers (e.g., procalcitonin, leukocyte count) in patients with suspected high VCAM-1 activity. Its role in bacterial translocation[8] supports frequent vital sign monitoring to detect early sepsis. Statin-mediated VCAM-1 inhibition[10]suggests nursing collaboration with clinicians to optimize pain management regimens for inflammatory abdominal pain.

LIMITATIONS

All studies used animal/cell models (limited human relevance); only 11 studies were included (small sample); few studies explicitly linked VCAM-1 to nursing outcomes (e.g., length of stay, pain scores).

FUTURE DIRECTIONS

Basic research should use human primary appendiceal endothelial cells; clinical nursing studies could test VCAM-1-targeted interventions (e.g., statin adjuvant therapy) on patient outcomes.

CONCLUSION

Basic experimental studies demonstrate VCAM-1 upregulation drives leukocyte infiltration and inflammatory progression in appendicitis, while VCAM-1 inhibition alleviates tissue damage and pain. These findings provide a molecular basis for nursing interventions (inflammation monitoring, sepsis prevention, targeted pain management) to improve appendicitis care. Bridging basic VCAM-1 research and clinical nursing is critical for enhancing patient outcomes.

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