

CASE REPORT

Synchronous Low-Grade Appendiceal Mucinous Neoplasm, Perforated Ascending Colon Diverticulitis, and Mature Ovarian Cystic Teratoma Treated in a Single Operation: A Case Report

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ARTICLE INFO

Article history:

Received: April 19, 2026

Revised: May 22, 2026

Accepted: June 18, 2026

Available Online: June 25, 2026

Published: June 28, 2026

Keywords:

Ascending colon diverticulitis; Low-grade appendiceal mucinous neoplasm; Mature cystic teratoma; Pseudomyxoma peritonei; Right hemicolectomy

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DOI:

<https://doi.org/10.37275/sjs.v9i1.177>

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ABSTRACT

Background: Low-grade appendiceal mucinous neoplasm (LAMN) is found in about 0.13%–0.30% of appendectomy specimens and can cause pseudomyxoma peritonei (PMP) after rupture. Its coexistence with perforated ascending colon diverticulitis and an ovarian mature cystic teratoma complicates diagnosis and surgical planning.

Objective: To describe the presentation, imaging pitfalls, single-stage surgical management, and 90-day outcome of this combination.

Methods: Following the CARE guideline, we report a 52-year-old postmenopausal woman treated at Klungkung Regional General Hospital, Indonesia (protocol No. 2026/0418) and followed from symptom onset (day –30) to postoperative day 90.

Results: On admission she had leukocytosis (white blood cells $19.18 \times 10^3/\mu\text{L}$; neutrophils 91.0%; neutrophil-to-lymphocyte ratio 21.67) and pre-renal azotemia (creatinine 1.40 mg/dL). Ultrasonography and computed tomography showed a $10.20 \times 10.44 \times 10.46$ cm (583.2 cm^3) right ovarian teratoma and a right lower quadrant phlegmon containing a tubular structure that was read as an abscess. At laparotomy, this proved to be a $9.50 \times 3.00 \times 2.50$ cm (37.31 cm^3) mucinous appendix; two ascending colon diverticula had perforated (50.0 mL of pus). Single-stage oncologic right hemicolectomy, end ileostomy, transverse colon stump closure, and total abdominal hysterectomy with bilateral salpingo-oophorectomy achieved R0 resection of a pT4a LAMN. White blood cells ($8.42 \times 10^3/\mu\text{L}$; –56.1%) and creatinine (0.88 mg/dL; –37.1%) normalized by day 5; at day 90, there was no clinical, biochemical, or CT evidence of PMP.

Conclusion: In this patient, a large ovarian teratoma and a diverticular phlegmon obscured a coexisting LAMN on imaging; systematic intraoperative exploration allowed all three lesions to be treated in one operation.

1. Introduction

The vermiform appendix is a narrow, blind-ending, lymphoid-rich tubular organ that arises from the posteromedial wall of the cecum approximately 2.0 cm below the ileocecal valve.¹ Although it was long regarded as a vestigial remnant, microbiome studies suggest that it acts as a reservoir ("safe house") that reseeds commensal colonic flora after diarrheal illness and contributes to intestinal immunoglobulin A production.^{2,3} The appendiceal lumen also gives rise to a heterogeneous spectrum of inflammatory and neoplastic diseases, ranging from acute suppurative appendicitis and acquired diverticulosis to neuroendocrine tumors, goblet cell adenocarcinomas, and mucinous epithelial neoplasms.^{4,5} Within this spectrum, low-grade appendiceal mucinous neoplasm (LAMN) is a distinct entity, defined by the Peritoneal Surface Oncology Group International (PSOGI) and the World Health Organization (WHO) as a proliferation of low-grade mucin-secreting columnar epithelium that replaces the normal appendiceal mucosa.^{3,4,6} It effaces the lamina muscularis mucosae, induces submucosal fibrosis, and expands with a pushing front, without destructive desmoplastic stromal invasion.^{3,6,7}

Primary appendiceal neoplasms account for approximately 0.5% of all gastrointestinal tract neoplasms, and LAMN is found in about 0.13% to 0.30% of appendectomy specimens.^{3–5} The

German national cancer registry recorded LAMN in 0.13% of 108,247 appendectomies in 2018, pooled series totaling 171,341 appendectomies found mucinous neoplasms in 0.28%, and appendiceal neoplasia of any type is found in about 0.7% to 2% of specimens, with a female predominance and a peak in the fifth to seventh decades of life.^{4,5,8,9} When patients present with complicated appendicitis and a periappendicular phlegmon or abscess, the prevalence of an underlying appendiceal neoplasm rises to 14.3%, compared with 1.5% in uncomplicated appendicitis, as shown in the prospective multicenter Peri-Appendicitis Acute Tumor cohort of 370 consecutive patients with periappendicular abscess.¹⁰ Other series report overall neoplasm rates of 0.84% to 6.5% in appendectomy specimens and a rising incidence over time,^{11–15} although a meta-analysis found no significant difference between complicated and uncomplicated appendicitis (3.29% vs. 1.49%).¹⁶ Despite its cytologically bland appearance and the absence of hematogenous or lymphatic spread, the main long-term risk of LAMN is that transmural mucin dissection or frank appendiceal rupture releases extracellular mucin and neoplastic epithelium into the peritoneal cavity, initiating pseudomyxoma peritonei (PMP).^{3–6,8,9} Once established, PMP spreads by the redistribution phenomenon—accumulating at gravitational and fluid-resorption sites such as the rectouterine pouch of Douglas, the right paracolic gutter, the greater omentum, and the

subdiaphragmatic spaces—and requires cytoreductive surgery (CRS) combined with hyperthermic intraperitoneal chemotherapy (HIPEC) at specialized peritoneal oncology centers.^{3,6,8,9,17}

LAMN is difficult to recognize before surgery because fewer than 30% to 40% of patients present with classical right lower quadrant (RLQ) pain mimicking acute appendicitis, while many lesions are discovered incidentally during cross-sectional imaging, endoscopy, or abdominal exploration for presumed gynecologic or colonic disease.^{3,18–22} The classical diagnostic signs—the ultrasonographic onion-skin sign of concentric echogenic mucin layers, the computed tomographic (CT) appearance of a curvilinear (eggshell) calcified tubular cyst more than 15 mm in diameter with internal attenuation below 20 Hounsfield units (HU), and the colonoscopic volcano sign of a smooth mound extruding mucin at the appendiceal orifice—are absent or obscured in more than half of presentations, particularly when the lesion is complicated by acute periappendicular inflammation.^{3,10,18,23,24} In postmenopausal women, an enlarged LAMN that reaches the pelvis is easily mistaken for a primary right adnexal tumor, because the two share contiguous anatomical boundaries in the right iliac fossa and show overlapping cystic–solid imaging features.^{25,26}

When a second or third pathology coexists in the right lower quadrant, cross-sectional imaging becomes harder to interpret.^{10,18,25,26} Right-sided colonic diverticulitis, which accounts for 38% to 75% of diverticulitis in Asian populations compared with only 1.5% to 5% in Western cohorts,^{27–30} produces segmental mural thickening of the ascending colon, pericolic fat stranding, intramural gas, and a localized phlegmon or abscess after perforation; these changes can camouflage an adjacent dilated appendix and create a radiological dilemma, which is followed by a histological one because appendiceal diverticular disease can mimic LAMN.^{30–32} A large ipsilateral mature cystic teratoma (dermoid cyst) of the right ovary, in turn, shows characteristic radiological landmarks—intraleisional sebaceous fat with negative CT attenuation (≤ -20 HU), coarse calcification (> 350 HU), and a Rokitansky protuberance^{33,34}—that may anchor the radiologist's attention and lead to premature diagnostic closure regarding the adjacent periappendicular phlegmon. Furthermore, in secondary and district referral hospitals in low- and middle-income countries (LMICs), surgeons often have to resolve this diagnostic ambiguity intraoperatively, without frozen-section histopathology, rapid serum tumor markers, or on-site CRS-HIPEC facilities.^{35,36}

To our knowledge, the synchronous occurrence of a histopathologically confirmed pT4a LAMN, multifocal perforated ascending colon diverticulitis, and an ipsilateral mature ovarian cystic teratoma in one patient has not been reported. The case shows how a 583.2 cm³ ovarian teratoma and a perforated ascending colon phlegmon concealed a 37.31 cm³ pT4a LAMN on ultrasonography and led to its misinterpretation as an abscess on CT until laparotomy. The objective of this case report is to describe the clinical, hemodynamic, hematological, and biochemical course of a 52-year-old postmenopausal woman with this combination of lesions; the imaging features and histological criteria that separate LAMN from complicated diverticular disease; the intraoperative multidisciplinary decision-making that converted a presumed adnexal exploration into a single-stage oncologic right hemicolectomy with end ileostomy, transverse colon stump closure, and total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH-BSO); and the five-year PMP surveillance planned in line

with current PSOGI and American Society of Colon and Rectal Surgeons (ASCRS) guidance.

2. Methods

Case design and reporting framework

This report describes a single patient managed by the Department of Digestive Surgery and the Department of Obstetrics and Gynecology, Klungkung Regional General Hospital, Semarapura, Bali, Indonesia, and follows the CARE guideline for case reports.^{37,38} The observation period spanned 120 consecutive days: the 30-day preoperative symptomatic period (day –30 to day 0), including 72 hours of inpatient treatment before referral at Gema Santi Nusa Penida Regional General Hospital (day –3 to day 0); the 5-day index surgical admission with intensive perioperative monitoring (day 0 to postoperative day [POD] 5); and outpatient oncological and stoma follow-up to POD 90.

Case setting and patient information

The patient was an adult woman referred to the emergency surgical service with an acute-on-chronic right lower quadrant and pelvic mass syndrome. Intraoperative exploration and histopathological examination established three distinct pathological entities: (1) a primary appendiceal mucinous epithelial neoplasm meeting the 2016 PSOGI modified Delphi consensus criteria and the 2019 WHO 5th edition classification for LAMN;^{3,4,6} (2) complicated ascending colon diverticular disease with macroscopic and microscopic transmural perforation;^{29,30,32} and (3) a histologically verified ipsilateral ovarian germ cell neoplasm (mature cystic teratoma).³³ Secondary mucinous carcinomatosis from an extra-appendiceal gastrointestinal primary, isolated uncomplicated acute appendicitis, and reactive appendiceal mucosal hyperplasia secondary to appendiceal diverticulitis without neoplastic architectural effacement were excluded.³¹

Data collection and diagnostic assessment

Clinical, hemodynamic, hematological, biochemical, and radiological parameters were recorded at nine time points: day –30 (symptom onset); day –3 (admission at Gema Santi Nusa Penida Regional General Hospital); day 0 (arrival at the emergency department, multimodal imaging, and emergency midline laparotomy at Klungkung Regional General Hospital); POD 1 (24-hour intensive care unit [ICU] hemodynamic and metabolic stabilization); POD 2 (48-hour transfer to the digestive surgery ward and start of ileostomy function); POD 3 (72-hour diet escalation from clear liquids to soft food); POD 4 (96-hour removal of the closed-suction peritoneal drain and confirmation of ambulation); POD 5 (120-hour discharge assessment); and POD 90 (3-month outpatient oncological, tumor-marker, and CT surveillance before planned ileostomy reversal). Preoperative imaging on day 0 used a 3.5–4.0 MHz convex transabdominal transducer with color Doppler (depth 15.0 cm, gain 61 dB), followed by dual-phase contrast-enhanced 64-slice multidetector abdominopelvic CT reconstructed in 3.0 mm axial and coronal planes, with region-of-interest densitometry in Hounsfield units. The resected specimens (en bloc right hemicolectomy specimen, greater omentum, and TAH-BSO specimen) were fixed in 10% neutral buffered formalin, sectioned serially at 3.0 mm intervals across the appendiceal base, body, tip, mesoappendix, ascending colon diverticular perforations, and Rokitansky tubercle, embedded in paraffin, stained with hematoxylin and eosin (H&E), and examined by brightfield microscopy ($\times 40$, $\times 100$, and $\times 400$).

Reference values and published cases

To place the diagnostic pitfalls and operative decisions in context without inferential claims, the patient's clinical, radiological, and pathological measurements were compared with three sets of references: (1) physiological and anatomical reference values for adult and postmenopausal women (normal adult appendiceal volume 1.13 cm³ and postmenopausal ovarian volume 2.50 cm³);^{39,40} (2) the histological criteria of Lowes et al. separating LAMN from complicated appendiceal and colonic diverticular disease,³¹ together with the PSOGI classification, the American Joint Committee on Cancer (AJCC) 8th edition staging categories (pTis, pT3, pT4a, pT4b, M1a-c), and recent LAMN stage-outcome data;^{4,6,41} and (3) nine published cohorts and case reports from 2021 to 2026 on appendiceal tumor prevalence in periappendicular abscess, diagnostic uncertainty in LAMN, adnexal mimicry, and diverticular differentiation (Table 5).

Measurements and analysis

Because only one patient was studied, no inferential statistical tests (such as t-tests, analysis of variance, Mann-Whitney U tests, or χ^2 tests) were used, and the findings are reported descriptively. Organ and tumor volumes were calculated with the ellipsoid formula $V = (\pi/6) \times L \times W \times H \approx 0.5236 \times L \times W \times H$, where L, W, and H are the orthogonal craniocaudal (longitudinal), laterolateral, and anteroposterior diameters in centimeters. Relative changes in hematological and biochemical variables from day 0 were expressed as $\Delta\% = [(X_t - X_{\text{day } 0}) / X_{\text{day } 0}] \times 100\%$. Systemic inflammation was described with four composite ratios derived from the complete blood count: the neutrophil-to-lymphocyte ratio (NLR = neutrophils [%]/lymphocytes [%]), the platelet-to-lymphocyte ratio (PLR = platelets [$\times 10^3/\mu\text{L}$]/absolute lymphocyte count [ALC, $\times 10^3/\mu\text{L}$]), the systemic immune-inflammation index (SII = platelets $\times$ NLR, $\times 10^3/\mu\text{L}$),⁴² and the systemic inflammation response index (SIRI = [absolute neutrophil count $\times$ absolute monocyte count]/ALC, $\times 10^3/\mu\text{L}$).⁴³ Hemodynamic status and renal function were assessed with the mean arterial pressure (MAP = DBP + [SBP – DBP]/3), the shock index (SI = HR/SBP), blood urea nitrogen (BUN = urea/2.14), and the 2021 race-free Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation for the estimated glomerular filtration rate (eGFR, mL/min/1.73 m²).⁴⁴ Diagnostic probability, peritoneal contamination, peritoneal disease, and postoperative morbidity were classified with the Alvarado score (0–10),⁴⁵ the Appendicitis Inflammatory Response (AIR) score (0–12),⁴⁶ the modified Hinchey classification (stages 0–IV),⁴⁷ the Sugarbaker Peritoneal Cancer Index (PCI, 0–39), and the Clavien–Dindo classification (grades I–V).⁴⁸

Ethical approval and consent

Ethical approval for this case report was granted by CMHC Research Center Ethics Committee, Indonesia (protocol no. 2026/0418), with institutional clinical governance authorization from Klungkung Regional General Hospital. The work was conducted in accordance with the Declaration of Helsinki (2013 Fortaleza revision) and the International Committee of Medical Journal Editors (ICMJE) Recommendations for the Protection of Research Participants. Written informed consent was obtained directly from the patient before surgery and before manuscript preparation, both for the extended multidisciplinary operation (oncologic right hemicolectomy, end ileostomy, transverse colon stump closure, and concomitant TAH-BSO) and for publication of de-identified clinical records, serial laboratory results, diagnostic images, and photomicrographs. Embedded DICOM headers, registration numbers, institutional timestamps, and personal identifiers

were removed from the ultrasound, CT, and histopathology images before figure assembly, in accordance with ICMJE patient-privacy standards.

3. Results

Patient information and preoperative course

A 52-year-old multiparous (P3A0, three spontaneous vaginal deliveries) postmenopausal Indonesian woman (menarche at 13 years, natural menopause at 50 years) presented to the emergency department of Klungkung Regional General Hospital (day 0) with a 30-day history of right lower quadrant (RLQ) pain that had worsened acutely over the preceding 48 hours. At symptom onset one month before admission (day –30), she had intermittent, dull epigastric discomfort that gradually moved to the right iliac fossa over several weeks—a subacute course that differs from the 12-to-24-hour periumbilical-to-McBurney migration of uncomplicated acute appendicitis. A primary-care consultation in the second week of illness led to empirical oral analgesia, which did not relieve the pain. Seventy-two hours before referral (day –3), the pain changed from intermittent colic to severe, constant, sharp RLQ pain with anorexia, persistent nausea, and three episodes of non-bilious, non-bloody vomiting. She was admitted to Gema Santi Nusa Penida Regional General Hospital, where she received 72 hours of conservative treatment with intravenous Ringer's lactate, intravenous ceftriaxone 2.0 g every 24 hours, oral ibuprofen 400 mg three times daily, and intravenous omeprazole 40 mg twice daily. Despite full adherence to this treatment, her RLQ peritoneal signs and leukocytosis progressed (white blood cells [WBC] from $17.60 \times 10^3/\mu\text{L}$ on day –3 to $19.18 \times 10^3/\mu\text{L}$ on day 0), prompting urgent referral by sea and road to our surgical service. She denied fever, chills, unintended weight loss, hematemesis, melena, hematochezia, dysuria, hematuria, postmenopausal vaginal bleeding, or leukorrhea, and reported normal passage of flatus and stool. She had no personal or family history of colorectal, ovarian, or endometrial malignancy, tuberculosis, viral hepatitis, diabetes mellitus, hypertension, or coagulopathy, and denied regular non-steroidal anti-inflammatory drug use, tobacco smoking, alcohol consumption, or herbal remedies.

On arrival at our emergency department (day 0), the patient was alert and fully oriented, with a Glasgow Coma Scale score of 15 (E4V5M6). Her blood pressure was 120/70 mmHg (calculated mean arterial pressure [MAP] 86.7 mmHg), heart rate 89 beats/min and regular, giving a shock index (SI = HR/SBP) of 0.74 (slightly above the 0.50–0.70 reference range⁴⁹), respiratory rate 20 breaths/min, axillary temperature 36.0 °C, and peripheral oxygen saturation (SpO₂) 98.0% on room air (FiO₂ 0.21). The conjunctivae were not pale and the sclerae were anicteric; cardiopulmonary auscultation revealed vesicular breath sounds without rales and regular heart sounds without murmurs. Abdominal inspection showed mild asymmetric lower abdominal distension without visible peristalsis, caput medusae, or surgical scars, and auscultation revealed hypoactive bowel sounds (3–4 per minute). Palpation elicited marked localized tenderness over McBurney's point with involuntary and voluntary muscular guarding, a positive Rovsing sign on palpation of the left iliac fossa, and a firm, ill-defined, tender deep mass occupying the RLQ and right hemipelvis and measuring approximately 10 cm in maximal clinical dimension. Digital rectal examination revealed normal sphincter tone, a smooth rectal vault without intraluminal masses or melena, and mild right anterolateral cul-de-sac tenderness; bimanual vaginal examination showed a non-tender cervix without cervical motion tenderness, no vaginal bleeding,

and an extrauterine 10-cm right adnexal–pelvic mass tethered to the right iliac fossa.

The admission hematological, leukocyte differential, inflammatory, and biochemical results (day 0) are given in Table 1. The complete blood count showed normochromic normocytic red cell indices (hemoglobin 15.4 g/dL, red blood cells $5.20 \times 10^6/\mu\text{L}$, hematocrit 44.7%, mean corpuscular volume [MCV] 85.4 fL, mean corpuscular hemoglobin [MCH] 29.5 pg, mean corpuscular hemoglobin concentration [MCHC] 34.5%, red cell distribution width [RDW-CV] 12.8%) and a lower-normal platelet count of $155 \times 10^3/\mu\text{L}$ (mean platelet volume [MPV] 7.62 fL). In contrast, there was marked leukocytosis of $19.18 \times 10^3/\mu\text{L}$ (+91.8% above the upper reference limit [URL] of $10.00 \times 10^3/\mu\text{L}$) with neutrophilia of 91.0% (+23.5% above the relative URL of 73.7%), corresponding to an absolute neutrophil count (ANC) of $17.45 \times 10^3/\mu\text{L}$ (+132.7% above the URL of $7.50 \times 10^3/\mu\text{L}$). There was also marked relative and absolute lymphopenia, consistent with acute stress (4.2%; absolute lymphocyte count [ALC] $0.81 \times 10^3/\mu\text{L}$, –19.0% below the lower reference limit [LRL] of $1.00 \times 10^3/\mu\text{L}$), and eosinopenia (0.04%, –93.3% below the relative LRL; absolute count $0.008 \times 10^3/\mu\text{L}$, –60.0% below the absolute LRL), whereas monocytes (4.4%; absolute monocyte count [AMC] $0.84 \times 10^3/\mu\text{L}$) and basophils (0.11%; $0.021 \times 10^3/\mu\text{L}$) were within reference limits.

Consequently, all four composite inflammatory indices were elevated: the neutrophil-to-lymphocyte ratio (NLR) was 21.67 (7.22 times the URL of 3.00; +622.3%), the platelet-to-lymphocyte ratio (PLR) 191.36 (+27.6% above the URL of 150.0), the systemic immune-inflammation index (SII) $3,358.85 \times 10^3/\mu\text{L}$ (5.60 times the URL of $600.0 \times 10^3/\mu\text{L}$; +459.8%), and the systemic inflammation response index (SIRI) $18.10 \times 10^3/\mu\text{L}$ (15.08 times the URL of $1.20 \times 10^3/\mu\text{L}$; +1,408.3%). Hepatic transaminases were normal (aspartate aminotransferase [AST] 35 U/L, alanine aminotransferase [ALT] 31 U/L, De Ritis ratio 1.13), whereas serum creatinine was elevated at 1.40 mg/dL (+16.7% above the URL of 1.20 mg/dL), with serum urea 36.0 mg/dL (calculated BUN 16.82 mg/dL; urea-to-creatinine ratio 25.71) and a reduced CKD-EPI eGFR of 46.8 mL/min/1.73 m², consistent with pre-renal acute kidney injury secondary to vomiting-induced hypovolemia and third-space peritoneal sequestration. Serum sodium was upper-normal (Na⁺ 145 mmol/L), potassium lower-normal (K⁺ 3.5 mmol/L), chloride normal (Cl[–] 103 mmol/L), and random blood glucose 121 mg/dL. The Alvarado score was 9/10 (high probability) and the Appendicitis Inflammatory Response (AIR) score 8/12 (indeterminate probability; C-reactive protein was not measured), together indicating an acute surgical abdomen (Table 1).

Table 1. Baseline hematology, leukocyte differential, derived inflammatory indices, hepatic and renal biochemistry, and clinical scores at index presentation (day 0).

Domain	Parameter	Value (day 0)	Unit	Reference interval	Deviation / interpretation
Hematology	Hemoglobin	15.4	g/dL	10.8–16.5	Within reference interval (normochromic normocytic)
Hematology	White blood cells (WBC)	19.18	$\times 10^3/\mu\text{L}$	3.50–10.00	+91.8% above URL; marked leukocytosis
Hematology	Red blood cells	5.20	$\times 10^6/\mu\text{L}$	3.50–5.50	Within reference interval
Hematology	Hematocrit	44.7	%	35.0–55.0	Within reference interval; mild hemoconcentration
Hematology	Platelets	155	$\times 10^3/\mu\text{L}$	145–450	Lower-normal
Leukocyte differential	Neutrophils (relative / ANC)	91.0 / 17.45	% / $\times 10^3/\mu\text{L}$	39.3–73.7 / 1.50–7.50	+23.5% (relative) / +132.7% (ANC) above URL; neutrophilia
Leukocyte differential	Lymphocytes (relative / ALC)	4.2 / 0.81	% / $\times 10^3/\mu\text{L}$	18.0–48.3 / 1.00–4.00	–76.7% (relative) / –19.0% (ALC) below LRL; stress lymphopenia
Leukocyte differential	Monocytes (relative / AMC)	4.4 / 0.84	% / $\times 10^3/\mu\text{L}$	4.4–12.7 / 0.20–1.00	At lower relative limit; absolute count preserved
Leukocyte differential	Eosinophils (relative / AEC)	0.04 / 0.008	% / $\times 10^3/\mu\text{L}$	0.60–7.30 / 0.02–0.50	–93.3% (relative) / –60.0% (AEC) below LRL; eosinopenia
Leukocyte differential	Basophils (relative / ABC)	0.11 / 0.021	% / $\times 10^3/\mu\text{L}$	0.00–1.70 / 0.00–0.20	Within reference interval
Derived ratios	Neutrophil-to-lymphocyte ratio (NLR)	21.67	Ratio	1.00–3.00	+622.3% above URL (7.22 $\times$); markedly elevated
Derived ratios	Platelet-to-lymphocyte ratio (PLR)	191.36	Ratio	90.0–150.0	+27.6% above URL; mildly elevated
Derived ratios	Systemic immune-inflammation index (SII)	3,358.85	$\times 10^3/\mu\text{L}$	300.0–600.0	+459.8% above URL (5.60 $\times$); markedly elevated
Derived ratios	Systemic inflammation response index (SIRI)	18.10	$\times 10^3/\mu\text{L}$	0.30–1.20	+1,408.3% above URL (15.08 $\times$); markedly elevated
Erythrocyte and platelet indices	MCV / MCH / MCHC	85.4 / 29.5 / 34.5	fL / pg / %	81.1–96.0 / 27.0–31.2 / 31.5–35.0	All within reference intervals
Erythrocyte and platelet indices	RDW-CV / MPV	12.8 / 7.62	% / fL	11.5–14.5 / 6.90–10.60	Normal anisocytosis and platelet volume
Hepatic biochemistry	AST / ALT / De Ritis ratio	35 / 31 / 1.13	U/L / U/L / ratio	8–37 / 13–42 / 0.80–1.20	Hepatocellular enzymes normal
Renal function	Serum urea / calculated BUN	36.0 / 16.82	mg/dL	10.0–50.0 / 7.0–20.0	Within reference interval
Renal function	Serum creatinine / CKD-EPI eGFR	1.40 / 46.8	mg/dL / mL/min/1.73 m ²	0.60–1.20 / ≥ 90.0	+16.7% above URL; consistent with pre-renal azotemia (emesis, peritonitis)
Electrolytes and glucose	Na ⁺ / K ⁺ / Cl [–] / random glucose	145 / 3.5 / 103 / 121	mmol/L ($\times 3$) / mg/dL	135–145 / 3.5–4.5 / 95–105 / 80–200	Upper-normal Na ⁺ and lower-normal K ⁺ after 48 h of vomiting
Clinical scores	Alvarado / AIR score	9/10 / 8/12	Score	Alvarado ≥ 7 high; AIR 0–4 low, 5–8 indeterminate, 9–12 high	High (Alvarado) and indeterminate (AIR) probability of appendicitis; CRP not measured

Abbreviations: ABC, absolute basophil count; AEC, absolute eosinophil count; AIR, Appendicitis Inflammatory Response; ALC, absolute lymphocyte count; ALT, alanine aminotransferase; AMC, absolute monocyte count; ANC, absolute neutrophil count; AST, aspartate aminotransferase; BUN, blood urea nitrogen (urea/2.14); CRP, C-reactive protein; eGFR, estimated glomerular filtration rate (2021 CKD-EPI creatinine equation); LRL, lower reference limit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPV, mean platelet volume; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio;

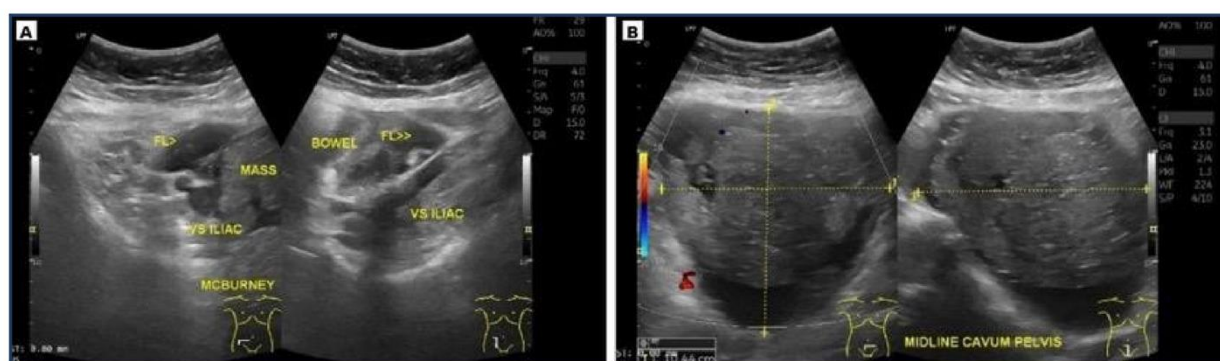
RDW-CV, red cell distribution width-coefficient of variation; SII, systemic immune-inflammation index (platelets $\times$ NLR); SIRI, systemic inflammation response index (ANC $\times$ AMC/ALC); URL, upper reference limit. Score references: Alvarado,⁴⁵ AIR.⁴⁶

Imaging findings

Bedside transabdominal ultrasonography on day 0 showed a large, lobulated, predominantly hyperechoic mass with thickened walls (4.2–5.8 mm), regular outer margins, and well-defined borders occupying the midline and right hemipelvis (Figure 1). Orthogonal caliper measurements were anteroposterior (AP) 10.20 cm $\times$ laterolateral (LL) 10.44 cm $\times$ craniocaudal (CC) 10.46 cm, corresponding to an ellipsoidal volume of $V = (\pi/6) \times 10.20 \times 10.44 \times 10.46 = 583.2 \text{ cm}^3$, or 233.3 times the volume of a normal postmenopausal ovary (2.50 cm^3). Color Doppler showed scant peripheral mural vascularity

without central chaotic neovascularization. Although the transducer was swept systematically across McBurney's point and the right iliac vessels (labeled VS ILIAC and BOWEL in Figure 1), the concentric echogenic onion-skin layering of an appendiceal mucocoele could not be visualized, probably because dense posterior acoustic attenuation from the sebaceous-keratinous pelvic mass and overlying gas in paralytic bowel loops obscured the retrocecal-paracolic space. The provisional ultrasonographic impression was therefore limited to a large pelvic abscess or a complex adnexal tumor of indeterminate origin (Figure 1).

(a) Transabdominal & McBurney-Point Gray-Scale (A) and Color Doppler (B) Ultrasonography at Index Admission (T0)



SONOGRAPHIC MORPHOMETRY (PANEL B)

- Anteroposterior (AP): 10.20 cm
- Laterolateral (LL): 10.44 cm
- Craniocaudal (CC): 10.46 cm
- Ellipsoidal Volume: $V = 583.2 \text{ cm}^3$ (233.3 $\times$ normal)

ACOUSTIC & DOPPLER ARCHITECTURE

- Echogenicity: Lobulated hyperechoic pelvic mass
- Wall profile: Thickened capsule (4.2–5.8 mm)
- Color Doppler: Mild peripheral mural flow
- Topography: Abuts RLQ bowel & right iliac vessels

ACOUSTIC MASKING MECHANISM

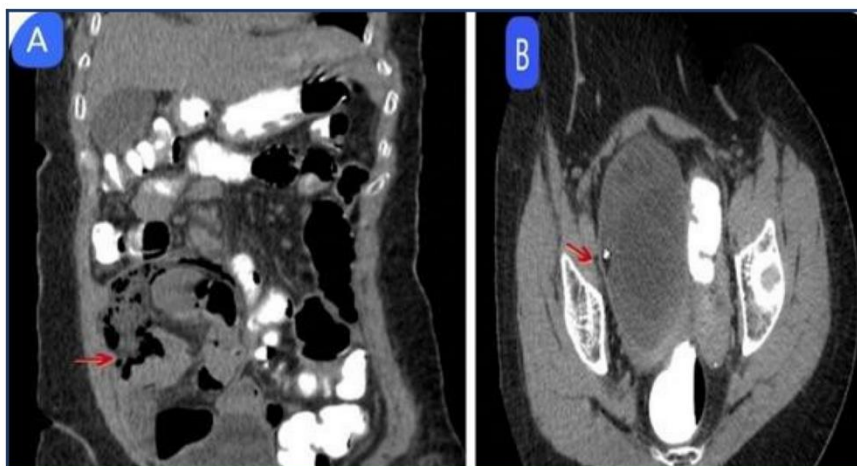
- Intracystic sebaceous lipid & keratin debris cast dense posterior acoustic attenuation shadows
- Overlying paralytic ileus bowel gas obscured the retrocecal 9.50 cm (37.31 cm^3) LAMN onion-skin sign

Figure 1. Preoperative transabdominal and McBurney-point ultrasonography at admission (day 0). Gray-scale (A) and color Doppler (B) images show a well-defined, thick-walled, heterogeneous, hyperechoic lobulated mass in the pelvis and right lower quadrant measuring AP 10.20 $\times$ LL 10.44 $\times$ CC 10.46 cm (ellipsoidal volume 583.2 cm^3), abutting adjacent bowel loops (BOWEL) and the right iliac vessels (VS ILIAC), with mild peripheral mural Doppler flow. The summary boxes give the sonographic measurements and the authors' interpretation of how posterior acoustic attenuation from the sebaceous contents and overlying bowel gas obscured the onion-skin sign of the 9.50-cm retrocecal low-grade appendiceal mucinous neoplasm (LAMN). AP, anteroposterior; CC, craniocaudal; LL, laterolateral; RLQ, right lower quadrant.

To resolve this ambiguity, dual-phase contrast-enhanced abdominopelvic computed tomography (CECT) was performed before laparotomy (Figure 2). Coronal and axial reconstructions showed two distinct but contiguous pathological foci in the right lower quadrant and pelvis. First, in the right iliac fossa (Panel A of Figure 2), there was marked focal mural densification and asymmetric wall thickening (8.5–11.0 mm) of the cecum and proximal ascending colon, surrounded by coarse pericolic fat stranding, extraluminal and intramural air bubbles, and a 9.50 $\times$ 3.00 cm enhancing tubular structure (internal fluid attenuation 12.0–18.0 HU) embedded in a coalescent inflammatory phlegmon. Because curvilinear eggshell mural calcification was absent and the periappendiceal fat planes were obliterated by pericolic gas and purulent exudate, this focus was interpreted as a complicated periappendiceal inflammatory mass with a

localized abscess. Second, in the right hemipelvis and midline pelvic cavity (Panel B of Figure 2), CECT showed a well-circumscribed, septated, heterogeneous solid-cystic adnexal mass measuring 10.46 cm in maximal diameter, containing intralésional fat-attenuating sebaceous material (–45.0 to –22.0 HU), a dependent fluid-debris interface, and a densely calcified mural Rokitsky protuberance (> 350.0 HU). This conspicuous appearance anchored the preoperative working diagnosis as (1) a right ovarian mature cystic teratoma, (2) a periappendiceal inflammatory phlegmon with a suspected abscess, and (3) an ascending colon inflammatory mass of indeterminate cause. The appendiceal lesion was thus visible as a tubular structure but was interpreted as an abscess, next to a teratoma 15.6 times larger (583.2 cm^3 vs. 37.31 cm^3) and within the diverticular phlegmon (Figure 2).

(a) Contrast-Enhanced Abdominopelvic CT: Coronal (A) & Axial (B)



(b) Quantitative Hounsfield Unit (HU) & Masking Profile

CORONAL RLQ PHLEGMON (PANEL A — RED ARROW)

- Cecal & ascending colon wall thickening (8.5–11.0 mm)
- Pericolic fat stranding & extraluminal/intramural air
- Enhancing tubular LAMN (9.50 × 3.00 cm; 37.31 cm³)
- **Intraluminal mucin attenuation: 12.0 to 18.0 HU**
- Phlegmon camouflaged LAMN as periappendiceal abscess

AXIAL PELVIC DERMOID CYST (PANEL B — RED ARROW)

- Septated solid-cystic right ovarian mass (10.46 cm)
- Volumetric dominance: 583.2 cm³ (15.6× LAMN volume)
- **Intralesional sebaceous lipid: −45.0 to −22.0 HU**
- **Calcified mural Rokitansky protuberance: >350.0 HU**
- Stereotyped dermoid signs triggered anchoring bias

Figure 2. Preoperative contrast-enhanced abdominopelvic computed tomography (CECT) at day 0. (A) Coronal image (red arrow) showing focal cecal and ascending colon wall thickening (8.5–11.0 mm) with pericolic fat stranding, extraluminal and intramural gas, and an enhancing 9.50 × 3.00 cm tubular structure (12.0–18.0 HU) within a right lower quadrant inflammatory phlegmon. (B) Axial pelvic image (red arrow) showing a septated solid-cystic right adnexal mass (10.46 cm; 583.2 cm³) with intralesional fat (−45.0 to −22.0 HU) and mural Rokitansky calcification (> 350.0 HU), consistent with a mature cystic teratoma. Panel (b) summarizes the attenuation values and the radiological interpretation. HU, Hounsfield units; LAMN, low-grade appendiceal mucinous neoplasm; RLQ, right lower quadrant.

Operative findings and single-stage surgical management

After intravenous crystalloid resuscitation (1,500 mL of Ringer's lactate to correct the pre-renal azotemia), broad-spectrum empirical antimicrobial coverage of enteric Gram-negative bacilli and obligate anaerobes (intravenous ceftriaxone 2.0 g every 24 hours plus intravenous metronidazole 500 mg every 8 hours), and multidisciplinary preoperative counseling of the patient and her family about the possible need for colonic resection, stoma diversion, and bilateral adnexectomy, emergency exploratory laparotomy was performed through an infraumbilical-to-supraumbilical midline incision by a combined digestive surgery and obstetrics-gynecology team. On entering the peritoneal cavity, 50.0 mL of frank, malodorous purulent fluid was aspirated from the right paracolic gutter and the rectouterine cul-de-sac and sent for microbiological culture (about 10 times the normal peritoneal fluid volume). Systematic four-quadrant exploration gave a Sugarbaker Peritoneal Cancer Index (PCI) of 0/39 for macroscopic mucinous implants: there were no pools of gelatinous ascites, no omental cake, and no peritoneal surface nodules over the hemidiaphragms, the Glisson capsule, or the pelvic peritoneum.

However, dense, vascularized fibropurulent adhesions obliterated the native tissue planes across four contiguous compartments: (1) between the hepatic flexure/ascending colon and the inferior surface of the right hepatic lobe; (2) between the gallbladder fundus and the transverse mesocolon; (3) between the cecum, appendix, and right lateral parietal peritoneum; and (4) between the right tubo-ovarian complex

and the ileocecal phlegmon. Careful sharp and blunt adhesiolysis under direct vision exposed the three lesions. First, the vermiform appendix was distended into a tense, sausage-shaped, thick-walled cystic mass measuring 9.50 × 3.00 × 2.50 cm (ellipsoidal volume 37.31 cm³, 33.02 times the normal appendiceal volume), with a congested serosa coated by fibrinous exudate and focal glistening acellular mucin-like extrusion into the mesoappendix, which immediately raised suspicion of an appendiceal mucinous neoplasm. Second, dissection of the ascending colon proximal to the periappendiceal phlegmon revealed multifocal inflamed diverticula, two of which had perforated through the full thickness of the wall (3.5 mm and 4.0 mm defects); they were partly walled off by adherent greater omentum and terminal ileal loops but were discharging purulent exudate along the right paracolic gutter (modified Hinchey stage IIb/III). Third, the right ovary was replaced by a 10.46 cm (583.2 cm³) thick-walled cystic tumor filled with sebaceous material and hair; the left ovary and fallopian tube were macroscopically atrophic and uninvolved, and the uterine corpus showed mild intramural leiomyomatous nodularity.

Intraoperative consultation between the digestive and gynecologic surgeons addressed three simultaneous needs in the absence of frozen-section pathology: avoiding iatrogenic rupture of the suspected LAMN and peritoneal seeding; achieving definitive source control of the two perforated ascending colon diverticula while avoiding an anastomosis in a purulent field; and achieving complete oncological clearance of the 10.46 cm postmenopausal ovarian neoplasm. The digestive

surgery team therefore performed a formal oncologic right hemicolectomy with a no-touch technique and high central ligation of the ileocolic and right colic arterial and venous pedicles at their superior mesenteric origins, with en bloc resection of the terminal 15 cm of ileum, the cecum, the 9.50 cm appendiceal mass, the ascending colon, the hepatic flexure, the complete mesocolic lymphovascular envelope, and the adherent greater omentum. Because the right retroperitoneal and paracolic bed was heavily contaminated by 50.0 mL of pus from the perforated diverticula—with an anastomotic leak probability exceeding 15%—primary ileo-transverse anastomosis was deferred in favor of fecal diversion: the distal transverse colon resection line was closed as a Hartmann-like transverse colon stump/mucous fistula, and a well-vascularized end ileostomy was constructed in the right lower abdominal wall. Through the same midline incision, the obstetrics and gynecology team then performed a total abdominal hysterectomy and bilateral salpingo-oophorectomy (TAH-BSO). The peritoneal cavity was irrigated with 4,000 mL of warm (37 °C) sterile 0.9% sodium chloride solution to clear cellular or mucinous debris, a closed-suction pelvic/paracolic silicone drain was placed, and the linea alba was closed in layers.

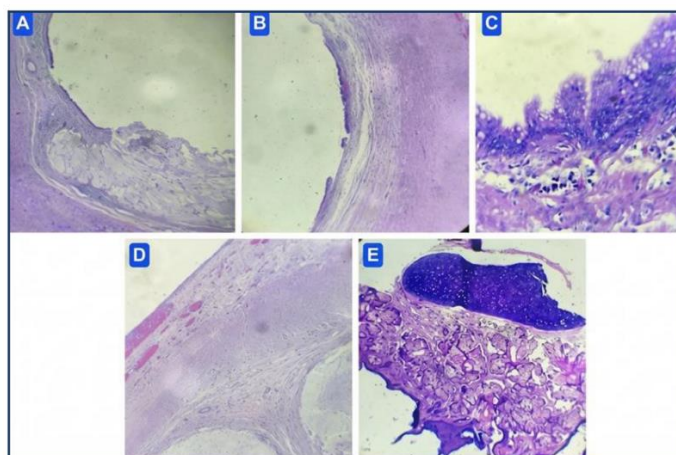
Histopathology and staging

Gross and microscopic examination of the en bloc right hemicolectomy, omentectomy, and TAH-BSO specimens established the definitive diagnosis of the three lesions (Figure 3). Macroscopically, the appendix measured 9.50 cm in length × 3.00 cm in maximal transverse width × 2.50 cm in anteroposterior thickness (ellipsoidal volume 37.31 cm³, 33.02 times the normal adult appendiceal volume of 1.13 cm³). Longitudinal sectioning showed a markedly dilated lumen distended by thick, tenacious, translucent-to-opalescent gelatinous mucin, a fibrotic and attenuated wall, and focal

glistening mucinous exudate extending onto the periappendiceal serosa and mesoappendix.

Microscopic examination of H&E-stained sections of the appendiceal body and tip (Panels A–C of Figure 3) showed a dilated lumen filled with abundant extracellular basophilic mucin (Panel A, ×40) and lined by an undulating, villous, and focally flattened monolayer of tall columnar mucin-producing epithelial cells with prominent apical cytoplasmic mucin vacuoles (Panel C, ×400). The neoplastic cells had hyperchromatic, elongated, basally oriented nuclei with low-grade cytological atypia (WHO grade 1; 0–1 mitoses per 10 high-power fields), without full-thickness nuclear stratification, cribriform or micropapillary architecture, signet-ring cells, or infiltrative tumor budding. Architecturally (Panel B of Figure 3, ×100), normal colonic-type crypts and submucosal lymphoid follicles were completely effaced, the lamina muscularis mucosae was obliterated around the whole neoplastic circumference, and the submucosa was replaced by dense, hyalinized collagenous fibrosis with a broad pushing-type front. Sections of the appendiceal subserosa and mesoappendix (Panel D of Figure 3, ×100) showed dense polymorphonuclear and mononuclear infiltrates, congested ectatic capillaries, and focal dissection of acellular mucin extruding through the visceral peritoneum into the periappendicular fibrofatty tissue, without extra-appendiceal neoplastic epithelial cells in 12 step sections. The right adnexectomy specimen (Panel E of Figure 3, ×100) showed a thick-walled fibrotic ovarian cyst lined by mature keratinizing stratified squamous epithelium with pilosebaceous units (sebaceous glands and hair follicles), mature univacuolar adipose lobules, and islands of mature hyaline cartilage, confirming a benign mature cystic teratoma without immature neuroectodermal tissue, germ cell neoplasia in situ, or squamous cell carcinoma (Figure 3).

(a) Hematoxylin-Eosin (H&E) Photomicrographs of Appendix (A–D) & Right Ovarian Dermoid Cyst (E)



(b) Histomorphometric & PSOGI / AJCC 8th Edition Staging Key

PANEL A (H&E ×40): APPENDICEAL LUMEN Dilated lumen (30.0 mm) distended by extracellular mucin and lined by flattened single-layer mucinous epithelium	PANEL D (H&E ×100): SUBSEROAL MUCIN EXTRUSION Focal acellular mucin dissecting into mesoappendix/serosa with PMN/MN infiltrate → PSOGI Stage pT4a pN0 M0 (R0)
PANEL B (H&E ×100): MURAL ARCHITECTURE 100% effacement of muscularis mucosae, lymphoid atrophy, loss of normal crypts & hyalinized submucosal fibrosis	PANEL E (H&E ×100): RIGHT OVARIAN DERMOID CYST - Keratinized stratified squamous epithelium, sebaceous glands, mature adipocytes & hyaline cartilage islands - Benign Mature Cystic Teratoma (0% immature / 0% SCC)
PANEL C (H&E ×400): EPITHELIAL CYTOMORPHOLOGY Undulating villous tall columnar mucinous cells with low-grade atypia (WHO G1; 0–1 mitoses/10 HPF; 0% budding)	

Figure 3. Histopathology of the right hemicolectomy and right adnexectomy specimens (hematoxylin and eosin [H&E]). (A, ×40) Distended appendiceal lumen (30.0 mm) filled with extracellular mucin and lined by flattened mucinous epithelium. (B, ×100) Mucosal denudation

with loss of colonic crypts, atrophy of submucosal lymphoid tissue, effacement of the lamina muscularis mucosae, and dense hyalinized submucosal fibrosis. (C, $\times 400$) Undulating villous monolayer of tall columnar mucinous cells with low-grade cytological atypia (G1, 0–1 mitoses/10 HPF) without infiltrative invasion. (D, $\times 100$) Periapendiceal subserosa and mesoappendix with dense polymorphonuclear and mononuclear infiltrates and focal acellular mucin, reported as AJCC 8th edition pT4a. (E, $\times 100$) Right ovarian cyst wall lined by keratinized stratified squamous epithelium with sebaceous glands, mature adipose tissue, and hyaline cartilage, consistent with a benign mature cystic teratoma. Panel (b) is the histological key. AJCC, American Joint Committee on Cancer; HPF, high-power field; PSOGI, Peritoneal Surface Oncology Group International.

The macroscopic, radiological, and histological measurements of the three lesions are compared with normal adult reference values in Table 2. All proximal ileal, distal transverse colonic, and radial mesenteric resection margins were microscopically free of neoplasia and mucin (R0 resection; 0/18 regional lymph nodes positive), giving a diagnosis of low-grade appendiceal mucinous neoplasm, AJCC 8th edition pT4a pN0 M0 (stage IIB), classified according to PSOGI. Adjacent segments of the

ascending colon confirmed multifocal true and false diverticulosis with two transmural diverticular perforations, peridiverticular microabscesses, and widespread serosal fibropurulent peritonitis (modified Hinchey stage IIB/III). Routine sections of the left ovary, both fallopian tubes, myometrium (mild benign leiomyoma), endometrium, and ectocervix were unremarkable (Table 2).

Table 2. Macroscopic, radiological, and histological characteristics of the three lesions compared with normal anatomical reference values.

Organ and lesion	Parameter	Patient measurement	Normal adult / postmenopausal reference	Ratio / deviation	Clinical and staging significance
Appendix (LAMN, pT4a)	Macroscopic dimensions (L $\times$ W $\times$ H) and volume	9.50 $\times$ 3.00 $\times$ 2.50 cm (V = 37.31 cm ³)	6.00 $\times$ 0.60 $\times$ 0.60 cm (V = 1.13 cm ³)	33.02 $\times$ volume (+3,201.8%)	Intraluminal mucin retention causing mural tension and localized serosal extrusion
Appendix (LAMN, pT4a)	CT attenuation and ultrasound profile	12–18 HU tubular cyst within phlegmon; onion-skin sign not seen	< 6.0 mm compressible lumen with air or fluid ³⁹	5.00 $\times$ outer diameter (30.0 mm)	Within the pericolic phlegmon and read as an abscess, so LAMN was not recognized preoperatively
Appendix (LAMN, pT4a)	Epithelial and mural histology (H&E)	Villous/flat G1 mucinous epithelium; muscularis mucosae effaced (100%); submucosal fibrosis	Colonic-type crypts, intact muscularis mucosae, abundant lymphoid follicles	0.0% crypt retention; 0–1 mitoses/10 HPF	Fulfills the PSOGI and Lowes et al. criteria for LAMN; excludes appendiceal diverticulosis
Appendix (LAMN, pT4a)	Extra-appendiceal mucin and resection margins	Focal acellular mucin in mesoappendix/serosa; no extra-appendiceal epithelium; R0 margins	Intact visceral serosa without mucin	pT4a pN0 M0 (AJCC 8th edition stage IIB)	No progression in pT4a M0 tumors in a 192-patient series; ⁴¹ 5-year imaging and tumor-marker follow-up planned
Ascending colon (perforated diverticulitis)	Diverticular morphology and perforation	Multifocal inflamed diverticula; 2 perforations sealed by omentum and ileum	Intact colonic wall (1.5–3.0 mm) without outpouching	2 transmural perforations (modified Hinchey IIB/III)	Cause of the acute 48-h peritonitic deterioration and dense visceral adhesions
Ascending colon (perforated diverticulitis)	Peritoneal contamination	50.0 mL purulent fluid along the right paracolic gutter	< 5.0 mL sterile serous peritoneal fluid	10.00 $\times$ fluid volume (+900.0%)	Primary ileo-transverse anastomosis deferred (> 15% leak risk); end ileostomy
Right ovary (mature cystic teratoma)	Ultrasound and gross dimensions and volume	AP 10.20 $\times$ LL 10.44 $\times$ CC 10.46 cm (V = 583.2 cm ³)	2.00 $\times$ 1.50 $\times$ 1.60 cm (V = 2.50 cm ³ , postmenopausal)	233.3 $\times$ volume	15.6 $\times$ larger than the LAMN-bearing appendix; dominated the imaging field and may have contributed to anchoring bias
Right ovary (mature cystic teratoma)	CT attenuation profile	Sebaceous lipid –45 to –22 HU; Rokitansky calcification > 350 HU; septation	Homogeneous ovarian stroma (25–45 HU)	Fat–calcium attenuation spread > 395 HU	Characteristic dermoid appearance; TAH-BSO chosen given the 1%–3% risk of malignant transformation after menopause ^{33,50}
Right ovary (mature cystic teratoma)	Histology and contralateral adnexa	Keratinized stratified squamous epithelium, sebaceous glands, adipocytes, hyaline cartilage	Atrophic postmenopausal ovarian cortex with corpora albicantia	No immature neuroepithelium or SCC; complete TAH-BSO	Benign mature cystic teratoma; uterus (mild leiomyoma) and left adnexa unremarkable

Volumes calculated with $V = (\pi/6) \times L \times W \times H$. Abbreviations: AJCC, American Joint Committee on Cancer; AP, anteroposterior; CC, craniocaudal; CT, computed tomography; G1, grade 1 (low grade); H&E, hematoxylin and eosin; HPF, high-power field; HU, Hounsfield units; LAMN, low-grade appendiceal mucinous neoplasm; LL, laterolateral; PSOGI, Peritoneal Surface Oncology Group International; R0, microscopically margin-negative resection; SCC, squamous cell carcinoma; TAH-BSO, total abdominal hysterectomy with bilateral salpingo-oophorectomy.

Postoperative course and 90-day follow-up

After surgery, the patient was monitored in the intensive care unit for 24 hours (POD 1) and then on the digestive surgery ward for 4 more days (POD 2 to POD 5), receiving intravenous ceftriaxone 2.0 g every 24 hours, intravenous metronidazole 500 mg every 8 hours, multimodal analgesia (intravenous paracetamol 1,000 mg every 8 hours with rescue intramuscular ketorolac 30 mg), intravenous omeprazole 40 mg twice daily, and standard pharmacological and mechanical venous thromboprophylaxis. The hemodynamic, hematological, biochemical, and gastrointestinal values at all nine time points are given in Table 3. After en bloc removal of the perforated

colon and the distended appendix, systemic inflammatory markers fell steadily: the leukocyte count decreased from $19.18 \times 10^3/\mu\text{L}$ on day 0 to $14.80 \times 10^3/\mu\text{L}$ on POD 1 ($\Delta\% = -22.8\%$), $12.10 \times 10^3/\mu\text{L}$ on POD 2 ($\Delta\% = -36.9\%$), $9.85 \times 10^3/\mu\text{L}$ on POD 3 ($\Delta\% = -48.6\%$, below the upper reference limit of $10.00 \times 10^3/\mu\text{L}$ within 72 hours), $9.10 \times 10^3/\mu\text{L}$ on POD 4 ($\Delta\% = -52.6\%$), and $8.42 \times 10^3/\mu\text{L}$ at discharge on POD 5 ($\Delta\% = -56.1\%$). Over the same five days, the neutrophil fraction fell from 91.0% to 68.0% ($\Delta\% = -25.3\%$; ANC $5.73 \times 10^3/\mu\text{L}$) and the lymphocyte fraction recovered from 4.2% to 22.5% (+435.7%; ALC $1.89 \times 10^3/\mu\text{L}$), so the NLR came down from 21.67 to 3.02 (–86.1%) by POD 5 (Table 3).

Table 3. Clinical, hemodynamic, hematological, biochemical, and functional course from symptom onset to the 3-month follow-up.

Time point	WBC ($\times 10^3/\mu\text{L}$) and $\Delta\%$ vs. day 0	Neutrophils (%) / NLR and $\Delta\%$ vs. day 0	Creatinine (mg/dL) / eGFR and $\Delta\%$ vs. day 0	BP (mmHg) / HR (bpm) / temp ($^{\circ}\text{C}$)	Gastrointestinal, stoma, and wound status	Key diagnostic or therapeutic step
Day -30 (symptom onset)	NR (outpatient)	NR	NR	122/74 / 78 / 36.4	Intermittent epigastric ache migrating to the RLQ; normal stool and flatus	Outpatient primary-care visit; oral analgesia without relief
Day -3 (outside admission)	17.60 (-8.2%)	88.5% / 16.40 (-24.3%)	1.28 / 52.1 (-8.6%)	118/72 / 86 / 36.5	Constant RLQ pain, nausea, 3 episodes of non-bilious vomiting	Admission to Gema Santi Nusa Penida Hospital: IV Ringer's lactate, ceftriaxone 2.0 g q24h, ibuprofen 400 mg TID, omeprazole 40 mg BID
Day 0 (referral and laparotomy)	19.18 (baseline)	91.0% / 21.67 (baseline)	1.40 / 46.8 (baseline)	120/70 (MAP 86.7) / 89 (SI 0.74) / 36.0	McBurney guarding, Rovsing sign, 10.46-cm RLQ-pelvic mass; 50.0 mL paracolic pus	Ultrasound and CECT; emergency midline laparotomy: oncologic right hemicolectomy, end ileostomy, transverse colon stump, TAH-BSO
POD 1 (ICU)	14.80 (-22.8%)	84.0% / 11.20 (-48.3%)	1.15 / 59.4 (-17.9% / +26.9%)	118/74 (MAP 88.7) / 82 / 36.6	Nil by mouth to sips of water; viable stoma; serosanguineous drain output 65 mL/24 h	ICU monitoring; IV ceftriaxone 2.0 g q24h + metronidazole 500 mg q8h; IV paracetamol + rescue IM ketorolac; thromboprophylaxis
POD 2 (ward)	12.10 (-36.9%)	78.0% / 6.85 (-68.4%)	1.02 / 68.2 (-27.1% / +45.7%)	120/76 (MAP 90.7) / 78 / 36.5	End ileostomy functioning (320 mL/24 h bilious effluent); clear fluids tolerated	Transfer from ICU to ward; stoma education and mobilization
POD 3 (diet escalation)	9.85 (-48.6%)	72.0% / 4.20 (-80.6%)	0.95 / 73.8 (-32.1% / +57.7%)	116/74 (MAP 88.0) / 76 / 36.4	Soft diet; stoma output 480 mL/24 h; drain < 25 mL/24 h	Leukocyte count ($< 10.00 \times 10^3/\mu\text{L}$) and creatinine (< 1.20 mg/dL) normalized; oral analgesia
POD 4 (drain removal)	9.10 (-52.6%)	70.0% / 3.50 (-83.8%)	0.90 / 77.6 (-35.7% / +65.8%)	118/72 (MAP 87.3) / 74 / 36.5	Drain removed; wound healing by primary intention	Independent stoma care by patient and family; no surgical site infection
POD 5 (discharge)	8.42 (-56.1%)	68.0% / 3.02 (-86.1%)	0.88 / 79.4 (-37.1% / +69.7%)	120/76 (MAP 90.7) / 74 / 36.5	Well-perfused end ileostomy (450 mL/24 h); Clavien-Dindo grade I course	Discharged on oral cefixime 200 mg BID (7 days), paracetamol 500 mg TID, ibuprofen 400 mg PRN, omeprazole 20 mg BID
POD 90 (month 3)	6.80 (-64.5%)	58.5% / 1.95 (-91.0%)	0.82 / 84.2 (-41.4% / +79.9%)	120/78 (MAP 92.0) / 72 / 36.5	Mature midline scar; good nutritional status; PCI 0/39 on CT	Abdominopelvic CECT negative for PMP; CEA 1.8 ng/mL, CA 19-9 14.2 U/mL, CA-125 11.6 U/mL; cleared for ileostomy reversal

Percentage change calculated as $\Delta\% = [(X_t - X_{\text{day 0}})/X_{\text{day 0}}] \times 100\%$. Abbreviations: BID, twice daily; BP, blood pressure; CA 19-9, carbohydrate antigen 19-9; CA-125, cancer antigen 125; CEA, carcinoembryonic antigen; CECT, contrast-enhanced computed tomography; eGFR, estimated glomerular filtration rate ($\text{mL}/\text{min}/1.73 \text{ m}^2$); HR, heart rate; ICU, intensive care unit; IM, intramuscular; IV, intravenous; MAP, mean arterial pressure; NLR, neutrophil-to-lymphocyte ratio; NR, not recorded; PCI, Peritoneal Cancer Index; PMP, pseudomyxoma peritonei; POD, postoperative day; PRN, as needed; RLQ, right lower quadrant; SI, shock index (HR/SBP); TAH-BSO, total abdominal hysterectomy with bilateral salpingo-oophorectomy; TID, three times daily; WBC, white blood cells.

The perioperative and 90-day course of inflammation and renal function and the relative volumes of the three lesions are shown in Figure 4. As shown in Panel c of Figure 4, perioperative crystalloid resuscitation and source control reversed the pre-renal azotemia: serum creatinine fell from 1.40 mg/dL on day 0 to 1.15 mg/dL on POD 1 ($\Delta\% = -17.9\%$, within the reference range after 24 hours), 1.02 mg/dL on POD 2 ($\Delta\% = -27.1\%$), 0.95 mg/dL on POD 3 ($\Delta\% = -32.1\%$), 0.90 mg/dL on POD 4 ($\Delta\% = -35.7\%$), and 0.88 mg/dL on POD 5 ($\Delta\% = -37.1\%$), and the CKD-EPI eGFR rose by 69.7%, from 46.8 mL/min/ 1.73 m^2 on day 0 to 79.4 mL/min/ 1.73 m^2 at discharge. The end ileostomy began functioning on POD 2 with passage of flatus and 320 mL/24 h of bilious effluent and stabilized at 450–480 mL/24 h from POD 3 to POD 5 as oral intake advanced from clear liquids to a soft diet. The pelvic-paracolic drain was removed on POD 4 after its 24-hour serous output had fallen below 25 mL, and the midline wound healed by primary intention without surgical

site infection (Clavien-Dindo grade I course). The patient was discharged on POD 5 with oral cefixime 200 mg twice daily for 7 days, oral paracetamol 500 mg three times daily, oral ibuprofen 400 mg as needed, and oral omeprazole 20 mg twice daily. At the 90-day (month 3) oncological follow-up visit, she was asymptomatic and well nourished, with a normal leukocyte count ($6.80 \times 10^3/\mu\text{L}$; $\Delta\% = -64.5\%$), a normal NLR (1.95; $\Delta\% = -91.0\%$), normal renal function (creatinine 0.82 mg/dL, $\Delta\% = -41.4\%$; eGFR 84.2 mL/min/ 1.73 m^2), normal serum tumor markers (carcinoembryonic antigen [CEA] 1.8 ng/mL [reference < 5.0 ng/mL], carbohydrate antigen 19-9 [CA 19-9] 14.2 U/mL [reference < 37.0 U/mL], and cancer antigen 125 [CA-125] 11.6 U/mL [reference < 35.0 U/mL]), and a negative contrast-enhanced abdominopelvic CT scan (PCI 0/39, no mucinous ascites); she was cleared for elective restoration of ileo-transverse continuity (Figure 4).

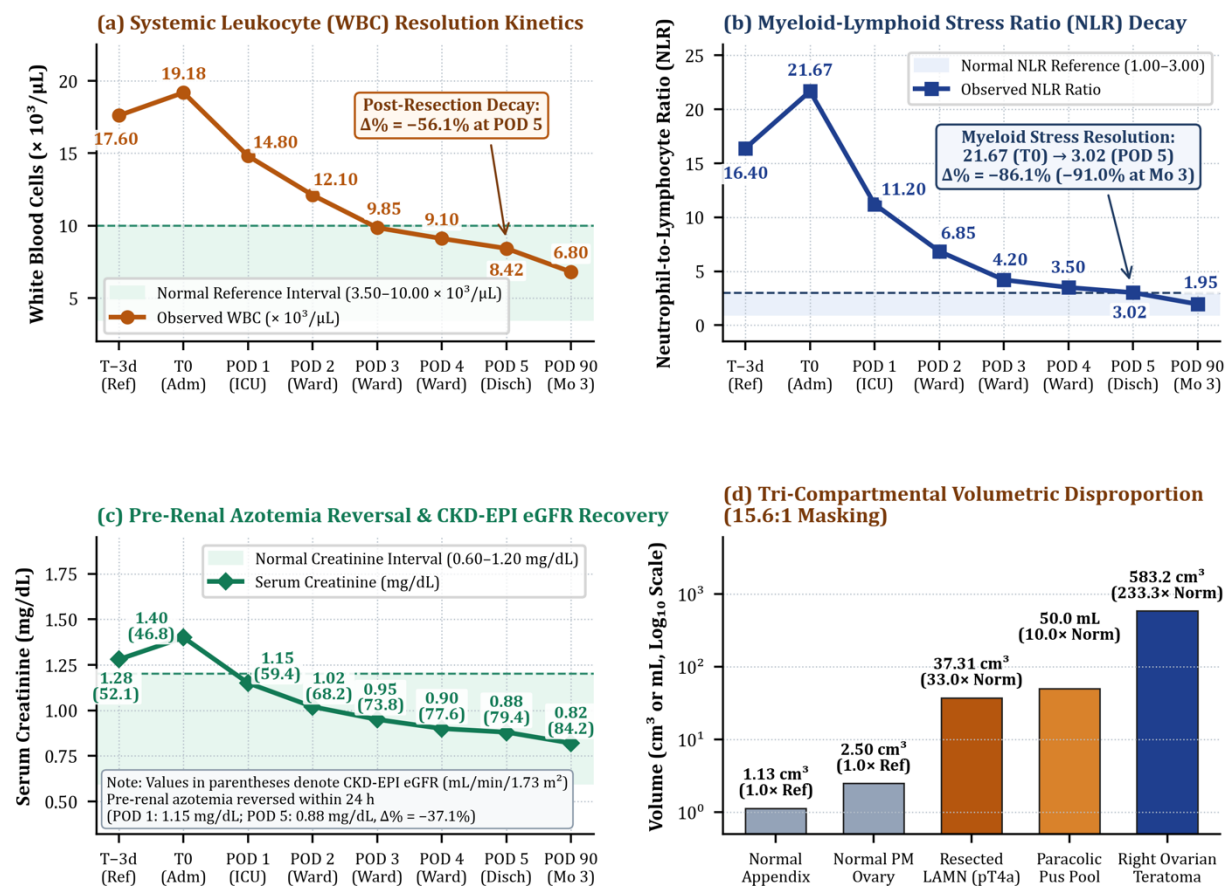


Figure 4. Perioperative and 90-day course and lesion volumes. (a) White blood cell count (WBC; $19.18 \times 10^3/\mu\text{L}$ at day 0, $8.42 \times 10^3/\mu\text{L}$ at POD 5 [$\Delta\% = -56.1\%$], and $6.80 \times 10^3/\mu\text{L}$ at POD 90 [$\Delta\% = -64.5\%$]). (b) Neutrophil-to-lymphocyte ratio (NLR; 21.67 at day 0, 3.02 at POD 5 [$\Delta\% = -86.1\%$], and 1.95 at POD 90 [$\Delta\% = -91.0\%$]). (c) Serum creatinine (1.40 mg/dL at day 0, 0.88 mg/dL at POD 5 [$\Delta\% = -37.1\%$], and 0.82 mg/dL at POD 90 [$\Delta\% = -41.4\%$]) with CKD-EPI eGFR in parentheses (46.8 to 84.2 mL/min/1.73 m²). (d) Logarithmic comparison of the volumes of the right ovarian mature cystic teratoma (583.2 cm³; 233.3× the normal postmenopausal ovary), the LAMN-bearing appendix (37.31 cm³; 33.0× the normal appendix; ratio 15.6:1), and the 50.0 mL of paracolic purulent fluid. eGFR, estimated glomerular filtration rate; LAMN, low-grade appendiceal mucinous neoplasm; POD, postoperative day.

Clinical scores and comparison with published cases

The clinical scores, pathological stage, and postoperative course are summarized in Table 4. In the authors' clinical interpretation, the pT4a LAMN accounted for the insidious 30-day prodrome of epigastric-to-RLQ discomfort due to visceral distension; the two perforated ascending colon diverticula (modified Hinchey stage IIb/III) accounted for the acute 48-hour deterioration, the failure of 72 hours of antibiotic treatment before referral, the 50.0 mL right paracolic purulent collection, and the marked inflammatory indices (WBC $19.18 \times$

$10^3/\mu\text{L}$, NLR 21.67, SII $3,358.85 \times 10^3/\mu\text{L}$, SIRI $18.10 \times 10^3/\mu\text{L}$); and the 583.2 cm³ right ovarian teratoma produced the palpable 10.46 cm pelvi-abdominal mass and the fat-calcium CT appearance that drew attention away from the LAMN. The baseline creatinine elevation (1.40 mg/dL; eGFR 46.8 mL/min/1.73 m²) was attributed to pre-renal hemodynamic causes (vomiting and peritonitis) rather than to cephalosporin or non-steroidal anti-inflammatory drug nephrotoxicity, because it normalized to 0.88 mg/dL ($\Delta\% = -37.1\%$) during continued antibiotic treatment (Table 4).

Table 4. Clinical scores, pathological staging, and postoperative course.

Instrument	Criterion assessed	Patient score / classification	Scale or threshold	Interpretation
Alvarado score ⁴⁵	Migratory RLQ pain (1), anorexia (1), nausea/vomiting (1), RLQ tenderness (2), guarding (1), WBC $> 10 \times 10^3/\mu\text{L}$ (2), neutrophils $> 75\%$ (1), afebrile (0)	9/10 points	0–10 (≥ 7 high probability)	Acute surgical abdomen requiring urgent exploration despite the atypical 30-day prodrome
Appendicitis Inflammatory Response (AIR) score ⁴⁶	Vomiting (1), RLQ pain (1), medium muscular defense (2), temperature $< 38.5^\circ\text{C}$ (0), PMN $\geq 85\%$ (2), WBC $\geq 15.0 \times 10^3/\mu\text{L}$ (2); CRP not measured	8/12 points	0–12 (0–4 low, 5–8 indeterminate, 9–12 high)	Indeterminate probability; marked inflammatory response consistent with the perforated diverticulitis
Modified Hinchey classification ⁴⁷	Multifocal ascending colon diverticulitis with 2 perforations, pericolic phlegmon, and 50.0 mL of right paracolic purulent fluid	Stage IIb / III	Stages 0, Ia, Ib, II, III, IV	Explains the failure of 72 h of IV ceftriaxone at the referring hospital
PSOGI / AJCC 8th edition staging ⁴¹	LAMN with acellular mucin in the periappendiceal serosa/mesoappendix; 0/18 nodes; no intraperitoneal cellular mucin	pT4a pN0 M0 (stage IIB)	pTis, pT3, pT4a, pT4b, M1a–c	R0 resection achieved by right hemicolectomy

Instrument	Criterion assessed	Patient score / classification	Scale or threshold	Interpretation
Peritoneal Cancer Index (PCI)	Macroscopic peritoneal mucinous implants at laparotomy and on month-3 CT	PCI 0/39	PCI 0–39	No peritoneal mucinous implants at laparotomy or on month-3 CT
Clavien–Dindo classification ⁴⁸	5-day hospitalization (1 day ICU, 4 days ward), ileostomy functioning on POD 2, drain removal on POD 4, primary wound healing	Grade I (no major morbidity)	Grades I–V	Uneventful recovery after single-stage resection in a contaminated field

Abbreviations: AJCC, American Joint Committee on Cancer; CRP, C-reactive protein; ICU, intensive care unit; IV, intravenous; LAMN, low-grade appendiceal mucinous neoplasm; PMN, polymorphonuclear leukocytes; POD, postoperative day; PSOGI, Peritoneal Surface Oncology Group International; RLQ, right lower quadrant; WBC, white blood cells.

Table 5 compares the present case with nine cohorts and case reports published between 2021 and 2026. In the prospective Finnish multicenter Peri-Appendicitis Acuta Tumor cohort, Salminen et al. found an appendiceal tumor in 14.3% of patients with periappendicular abscess, and CT raised suspicion of a tumor in only 8 of 52 patients with tumors (15.4%).¹⁰ The other publications—by Lu et al.,⁵¹ Kindie et al.,³⁵ Perivoliotis et al.,⁵² Vidović et al.,⁵³ Ma et al.,²³ Wang et al.,⁵⁴ Matias-García et al.,⁵⁵ and Arrieta Navarro et al.³⁶—describe primary appendiceal lesions (LAMN, HAMN, mucinous adenocarcinoma, or

appendiceal diverticulitis), with disseminated ovarian and peritoneal disease in one case (Kindie et al.) and no neoplasm in another (Vidović et al.); none describes three independent pathologies in the same anatomical quadrant, as in our patient. As shown in Panel d of Figure 4 and in Table 2, the ovarian teratoma (583.2 cm³) was 15.6 times larger than the LAMN-bearing appendix (37.31 cm³), which, together with the 50.0 mL of purulent diverticular fluid, may explain why the appendiceal neoplasm was not recognized on preoperative ultrasonography and CECT.

Table 5. Present case and published reports of appendiceal mucinous neoplasms with diagnostic mimicry, appendiceal diverticular disease, or adnexal misdiagnosis (2021–2026).

Study / year (country)	Patient(s)	Presentation and laboratory findings	Preoperative imaging and diagnostic pitfall	Operation	Pathology, stage, and outcome
Present case, Sandika and Yustina, 2026 (Indonesia)	52 / F (postmenopausal; n = 1)	30-day RLQ pain with acute worsening; WBC 19.18 × 10 ³ /μL, neutrophils 91.0%, NLR 21.67, creatinine 1.40 mg/dL	USG: 10.46-cm (583.2 cm ³) pelvic mass; CECT: fat-containing calcified ovarian teratoma and RLQ phlegmon; the 9.50-cm LAMN was read as an abscess	Oncologic right hemicolectomy, end ileostomy, transverse colon stump, TAH-BSO (digestive surgery and OB-GYN teams)	LAMN (pT4a, 37.31 cm ³) + perforated ascending colon diverticulitis (Hinchey IIb/III) + right ovarian mature cystic teratoma; LOS 5 days; month 3 PCI 0
Salminen et al., ¹⁰ 2025 (Finland)	Prospective multicenter cohort (n = 370 periappendicular abscesses; median age 58 years)	Acute appendicitis with imaging-confirmed periappendicular abscess	CT raised tumor suspicion in only 8 of 52 patients with tumors (15.4%); CT cannot separate inflammation from tumor	Interval or emergency appendectomy, ileocecal resection, or right hemicolectomy	Tumor rate 14.3% (53/370: 21 LAMN, 20 adenocarcinoma, 8 adenoma, 5 NET); age ≥ 35 years, sensitivity 98.1%
Lu et al., ⁵¹ 2024 (USA)	41 / M (n = 1; case report and literature review)	3 days of acute RLQ pain	CT: two fluid collections read as perforated appendicitis with abscess	Exploratory laparotomy and right hemicolectomy	Perforated LAMN with acellular mucin; margins and 42 nodes negative; follow-up 4 months
Kindie et al., ³⁵ 2025 (Ethiopia)	37 / F (n = 1)	6-month abdominal swelling, 20-week-size adnexal mass; CA-125 189 IU/mL, CEA 10 ng/mL	Ultrasound only (no CT): bilateral complex cystic ovarian masses read as ovarian malignancy	TAH, bilateral salpingo-oophorectomy, omentectomy, and appendectomy	LAMN pT4b NXM1c with ovarian, omental, and peritoneal involvement; FOLFOX chemotherapy
Perivoliotis et al., ⁵² 2021 (Greece)	71 / F (n = 1)	1 month of vague lower abdominal pain; normal tumor markers	Transvaginal ultrasound and MDCT: 3.3 × 6.5 cm cystic lesion read as a right ovarian tumor	Right hemicolectomy (tumor excised unruptured)	LAMN with a normal right ovary; disease-free at 6 months
Vidović et al., ⁵³ 2025 (Croatia)	53 / M (n = 1; + systematic review)	Acute right-sided abdominal pain since the same morning; WBC 20.9 × 10 ³ /μL, CRP 22.4 mg/L	Ultrasound only: 25-mm non-compressible tubular structure suggesting appendicitis	Laparoscopic appendectomy	Uncomplicated acquired appendiceal diverticulitis (no neoplasm); uneventful at 3 months
Ma et al., ²³ 2025 (China)	39 / F (n = 1)	9-day RLQ pain and distension, palpable 15 × 6 cm mass; WBC 9.25 × 10 ³ /μL; CEA 65 ng/mL, CA 19-9 329 U/mL, CA-125 85 U/mL	CT: 13 × 5 cm eggplant-like calcified appendiceal cyst; colonoscopy: smooth spherical lesion protruding from the appendiceal orifice	3D laparoscopic appendectomy with ultrasonic division of the mesoappendix after anti-inflammatory treatment	Giant LAMN (13 × 6 × 5 cm; CK20+/MUC2+/CK7–) without serosal invasion; no recurrence during follow-up
Wang et al., ⁵⁴ 2022 (USA)	Case series (n = 5; 45–79 years)	LUQ pain, constipation with weight loss, microscopic hematuria, or RLQ/flank pain	CT; diagnosis established only at pathology	Appendectomy in all patients	LAMN pTis (4) and pT3 (1); no recurrence on CT at 1 year
Matias-García et al., ⁵⁵ 2021 (Spain)	Retrospective cohort (n = 24 mucinous lesions: 20 LAMN, 1 HAMN, 3 mucinous adenocarcinomas)	Chronic abdominal pain 37.5%, acute right iliac fossa pain 29.2%, incidental 29.2%	Diagnosis established by CT in 50%	Appendectomy 62.5%, right colectomy 8 patients, CRS-HIPEC 4 patients	No recurrence at a median follow-up of 15 months
Arrieta Navarro et al., ³⁶ 2026 (Peru)	68 / F (n = 1)	Chronic abdominal pain and distension, abnormal vaginal bleeding; CEA 10.5 ng/mL, creatinine 1.89 mg/dL	TVUS and CT/MRI (13.1 × 7.7 × 4.4 cm calcified pelvic mass) read as bilateral ovarian neoplasm	Planned TAH-BSO converted to oncologic right hemicolectomy (18 × 8 × 5 cm specimen)	High-grade appendiceal mucinous neoplasm (HAMN) sparing the ovaries; referred for oncologic follow-up

Abbreviations: CA, carbohydrate antigen (CA 19-9) or cancer antigen (CA-125); CEA, carcinoembryonic antigen; CECT, contrast-enhanced computed tomography; CK, cytokeratin; CRP, C-reactive protein; CRS-HIPEC, cytoreductive surgery with hyperthermic intraperitoneal chemotherapy; F, female; FOLFOX, folinic acid, fluorouracil, and oxaliplatin; HAMN, high-grade appendiceal mucinous neoplasm; LAMN, low-grade appendiceal mucinous neoplasm; LOS, length of hospital stay; LUQ, left upper quadrant; M, male; MDCT, multidetector computed tomography; MRI, magnetic resonance imaging; MUC2, mucin 2; NET, neuroendocrine tumor; NLR, neutrophil-to-lymphocyte ratio; OB-GYN, obstetrics and gynecology; PCI, Peritoneal Cancer Index; RLQ, right lower quadrant; TAH-BSO, total abdominal hysterectomy with bilateral salpingo-oophorectomy; TVUS, transvaginal ultrasonography; USG, ultrasonography; WBC, white blood cells.

4. Discussion

Three coexisting lesions in the right lower quadrant

In this 52-year-old postmenopausal woman, a histologically verified pT4a LAMN, multifocal perforated ascending colon

diverticulitis, and a 10.46 cm ipsilateral mature cystic teratoma of the ovary were found together in the right lower quadrant. Each lesion, on its own, has an established diagnostic and surgical pathway. Here they coexisted, and each carried a different hazard. The indolent epithelial neoplasm threatened

late peritoneal mucinous dissemination (PMP).^{3-6,8,9} The transmural colonic perforation caused immediate Gram-negative and anaerobic peritonitis.³² The large postmenopausal germ cell tumor carried an occult risk of malignant transformation into squamous cell carcinoma.³³ Taken together, the three lesions illustrate pitfalls in diagnostic imaging and in intraoperative decision-making in a resource-limited regional hospital (Figure 5).

From a pathophysiological standpoint (Tier 1 of Figure 5), the three lesions arise along independent biological pathways. Axis I—the appendiceal LAMN—is typically driven by somatic activating mutations in KRAS (predominantly codons 12 and 13) and in GNAS (codon 201, R201C/R201H), which were not tested in this patient; KRAS mutations were found in 94% and GNAS mutations in 50% of 32 LAMNs in one series⁵⁶ and in 98.1% and 55.8%, respectively, of 52 LAMN and PMP cases in another, and KRAS–GNAS co-mutation is reported in 65% to 85% of LAMNs.^{3,7} Constitutive activation of the GNAS–cyclic adenosine monophosphate–protein kinase A (cAMP–PKA) cascade and of the KRAS–mitogen-activated protein kinase (MAPK) pathway increases the production of the gel-forming mucins MUC2 and MUC5AC by appendiceal goblet and columnar cells.^{3,7,57} Because the appendix is a narrow blind-ending tube, continuous intraluminal accumulation of viscous mucin produces progressive hydrostatic distension (to 37.31 cm³ in our patient), which compresses the lamina propria, causes ischemic atrophy of the submucosal lymphoid follicles, effaces the muscularis mucosae, and leads to hyalinized submucosal fibrosis with a pushing front.^{3,4,6,7,23} When intraluminal pressure exceeds the tensile strength of the thinned wall, acellular mucin dissects

through the muscularis propria and may reach the serosa and mesoappendix (Panel D of Figure 3 and Table 2); under the AJCC 8th edition, mucin confined to the subserosa or mesoappendix is classified as pT3, and mucin involving the serosal surface as pT4a.^{4,41}

Axis II—perforated ascending colon diverticulitis—reflects the behavior of right-sided colonic diverticular disease, which predominates in Asian populations and in which true and false diverticula are both described along the vasa recta penetrations of the ascending colon.^{27,30,32,58} Stasis of inspissated fecaliths within the diverticula is thought to trigger mucosal erosion, anaerobic and Gram-negative bacterial proliferation, and transmural necrotizing inflammation; in our patient, this culminated in two perforations (modified Hinchey stage IIb/III) with 50.0 mL of pus along the right paracolic gutter. The contiguity between the perforated ascending colon and the LAMN-bearing appendix may have created a bidirectional inflammatory loop (Tier 2A of Figure 5): pericolic fibropurulent exudate tethered the ascending colon to the right hepatic lobe, gallbladder fundus, cecum, and right adnexa, and the extrinsic phlegmon may have further impaired appendiceal venous drainage and accelerated serosal mucin extravasation. Axis III—the right ovarian mature cystic teratoma—is thought to arise by parthenogenetic development of a germ cell after the first meiotic division (homozygous 46,XX genotype), with differentiation along ectodermal (keratinized stratified squamous epithelium and sebaceous glands), mesodermal (mature adipose tissue and hyaline cartilage), and endodermal lineages;³³ in this patient it formed a 583.2 cm³ pelvi-abdominal mass (Tiers 1–4 of Figure 5).

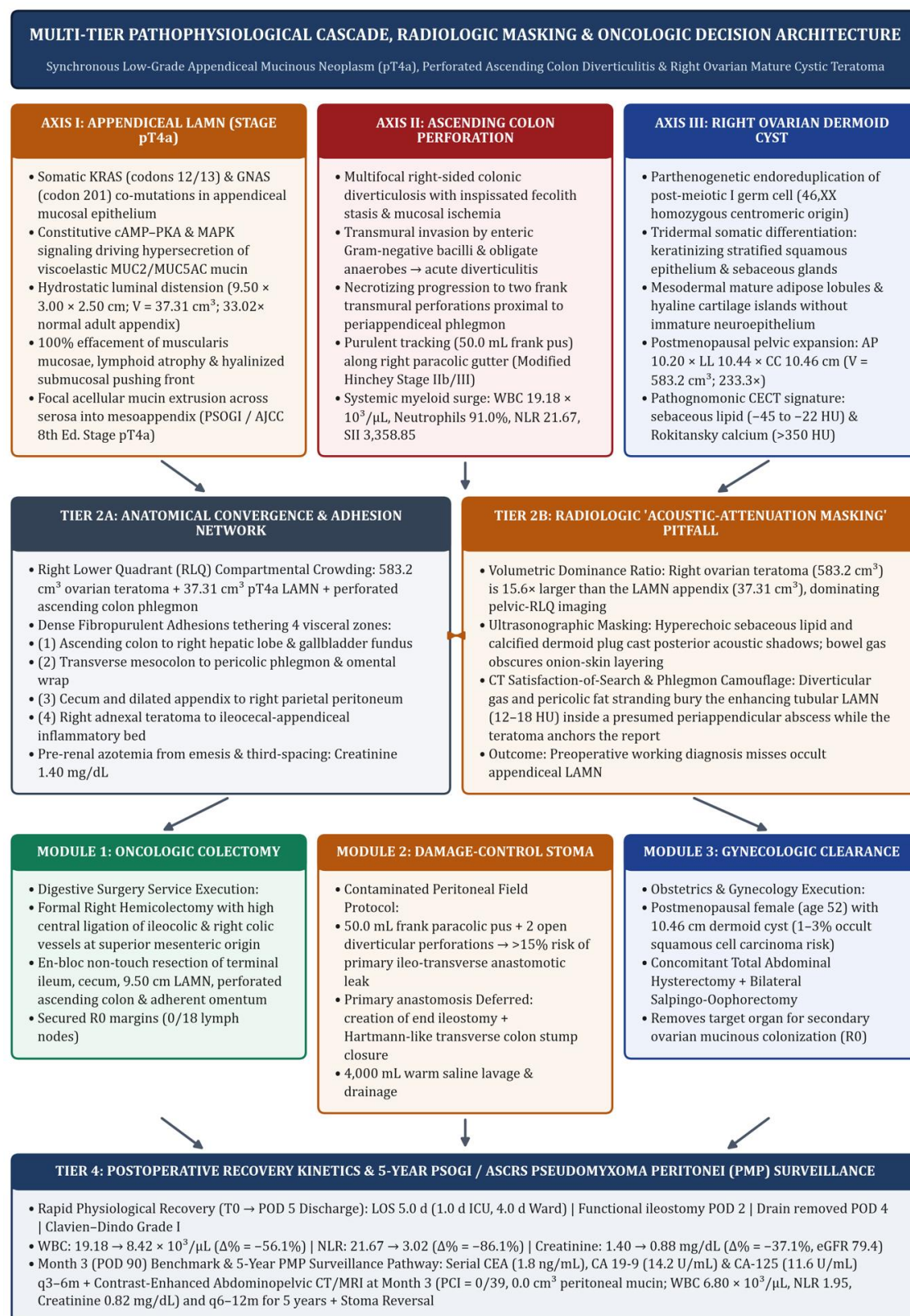


Figure 5. Schematic summary of the three lesions, the imaging pitfall, and the operative strategy. Tier 1 shows the three unrelated disease processes; the molecular statements (KRAS/GNAS-driven MUC2/MUC5AC hypersecretion, parthenogenetic origin of the teratoma) are background from the literature and were not tested in this patient. Tier 2 shows the anatomical convergence in the right lower quadrant and the 15.6:1 size difference that, in the authors' interpretation, contributed to the LAMN being missed. Tier 3 shows the three operative components (oncologic right hemicolectomy with high ileocolic and right colic ligation, end ileostomy with transverse colon stump, and concomitant TAH-BSO). Tier 4 summarizes the 5-day recovery and the planned 5-year surveillance for pseudomyxoma peritonei (PMP). LAMN, low-grade appendiceal mucinous neoplasm; TAH-BSO, total abdominal hysterectomy with bilateral salpingo-oophorectomy.

Imaging pitfalls, histological differentiation, and oncological clearance

The main clinical lesson of this case is that a large dermoid cyst and a diverticular phlegmon can obscure or mimic an adjacent

appendiceal lesion (Tier 2B of Figure 5), so that LAMN was not recognized before surgery on either ultrasonography or contrast-enhanced CT.^{10,18,25,26,35,51,52} In uncomplicated appendiceal mucocoeles, transabdominal ultrasonography

typically shows an encapsulated tubular mass with concentric echogenic mucinous layers (the onion-skin sign), and CT shows a thin-walled cystic structure of low attenuation (below 20 HU in most reports),^{18,23,24} with mural calcification in 38.7% to 55% of cases.^{18,24,59} In our patient, two factors probably removed these cues. First, on ultrasonography (Figure 1), the 583.2 cm³ right ovarian teratoma, filled with echogenic sebaceous material, keratin debris, and hair, produced posterior acoustic shadowing that obscured the retrocecal–paracolic space,³³ while gas in the adjacent paralytic bowel loops scattered the ultrasound beam. Second, on dual-phase CECT (Figure 2), the two perforated diverticula produced a dense pericolic phlegmon with extraluminal air and purulent exudate that enveloped the 9.50 × 3.00 × 2.50 cm appendix; the hypoattenuating appendiceal lumen (12.0–18.0 HU) was therefore read as a loculated periappendicular abscess, whereas the striking fat–calcium appearance of the 10.46 cm teratoma (fat –45.0 to –22.0 HU; Rokitansky calcification > 350.0 HU) drew diagnostic attention.^{10,18,33,60} This observation is consistent with the prospective data of Salminen et al., in whom CT raised suspicion of a tumor in only 8 of 52 patients (15.4%) with an appendiceal tumor in a periappendicular abscess, and an age of 35 years or older identified patients with tumors with a sensitivity of 98.1%.¹⁰

After resection, the second diagnostic challenge was to distinguish a true LAMN from reactive mucosal changes secondary to complicated appendiceal or colonic diverticulitis.^{6,7,31,53,61,62} In the comparative pathology study of Lowes et al., complicated appendiceal diverticular disease closely mimicked LAMN, and ruptured appendiceal diverticula may extrude acellular mucin onto the serosa.³¹ LAMN is characterized by the following features, all of which were present in our patient's specimen (Figure 3 and Table 2): (1) replacement of the normal colonic-type crypts by tall, hypermucinous, filiform or flat villous epithelium with elongated, basally oriented nuclei; (2) effacement of the lamina muscularis mucosae; (3) atrophy and loss of the submucosal lymphoid follicles; (4) dense, hyalinized collagenous fibrosis of the submucosa; and (5) a broad pushing-type front rather than herniation of intact mucosa through a vascular hiatus in the muscularis propria.^{3,4,6,7,31} Histological examination also excluded high-grade appendiceal mucinous neoplasm (HAMN), which is defined by high-grade cytological atypia (micropapillary or cribriform growth occurs only occasionally),^{6,63} and invasive mucinous adenocarcinoma, which is characterized by destructive desmoplastic stromal invasion or signet-ring cells.^{6,60,64}

In the operating room, the multidisciplinary team faced a complex decision without intraoperative frozen-section histopathology, which is commonly unavailable in secondary referral hospitals in Indonesia. According to current PSOGI/EURACAN and ASCRS guidance, an uncomplicated LAMN confined to the appendix can be managed by appendectomy with complete mesoappendiceal excision, provided that a microscopically negative (R0) cecal margin is achieved without iatrogenic rupture.^{3,8,9,54,65} Right hemicolectomy is considered when the base of the appendix is involved,^{3,9} whereas supplemental hemicolectomy offers no advantage for LAMN confined to the appendix.^{8,54} In our patient, three findings made simple appendectomy or limited ileocecal resection unsafe (Tier 3 of Figure 5): (1) the 9.50 cm tense, mucin-distended appendix was fused with the cecum into a single inflammatory mass, so that stapling across the appendiceal base risked iatrogenic rupture and peritoneal seeding; (2) the proximal and mid-ascending colon contained multifocal diverticulitis with two

active perforations extending toward the hepatic flexure; and (3) without frozen section, a mucinous adenocarcinoma could not be excluded. A formal oncologic right hemicolectomy with high ligation of the ileocolic and right colic vessels achieved R0 margins (0/18 nodes), removed the perforated ascending colon segment, and cleared the adherent greater omentum. Because primary ileo-transverse anastomosis in a purulent field (modified Hinchey stage IIb/III) carries an anastomotic leak risk of more than 15%, an end ileostomy with a Hartmann-like transverse colon stump diverted the fecal stream and was followed by rapid recovery (WBC –56.1%, NLR –86.1%, and creatinine –37.1% by POD 5; Table 3 and Figure 4).

Performing a concomitant TAH-BSO during the same laparotomy addressed two oncological concerns in a 52-year-old postmenopausal patient.^{25,26,33} First, whereas ovary-sparing cystectomy is standard for benign mature cystic teratomas in women of reproductive age, postmenopausal women with dermoid cysts of 10 cm or more face a 1% to 3% risk of occult malignant transformation,^{33,50,66} 80% to 85% of which are squamous cell carcinomas,^{33,67} associated with TP53 and PIK3CA mutations^{66,67} and with poor survival when the tumor is spilled intraoperatively, as described in the reports of Cong et al.,³³ Luo et al.,⁶⁷ Palomba et al.,⁵⁰ and Li et al.⁶⁶ Second, in women with appendiceal mucinous neoplasms, the ovaries are a frequent site of synchronous or metachronous secondary mucinous involvement (about half of female patients), which is often misdiagnosed as primary ovarian mucinous carcinoma before immunophenotyping (CK20+/CDX2+/SATB2+/MUC2+/CK7–/PAX8–).^{3,25,26,35,36,52} Bilateral salpingo-oophorectomy with hysterectomy removed both the risk of occult teratomatous malignancy and the target organ for secondary ovarian mucinous implantation, with a single anesthetic and a single incision.

Finally, our patient's pathological stage—pT4a pN0 M0 LAMN with focal acellular extra-appendiceal mucin in the mesoappendix/serosa—warrants structured surveillance for at least five years (Tier 4 of Figure 5).^{3,41} In the series of Ballentine et al., none of the 74 tumors confined to the appendix (pM0, including 12 pT4a M0 tumors) progressed, whereas 22.2% of tumors with peritoneal acellular mucin (pM1a) and 70.3% of those with intraperitoneal neoplastic cells (pM1b) did;⁴¹ in another series, peritoneal recurrence occurred in 7 of 46 perforated and in none of 76 non-perforated LAMNs,³ and smaller series and reviews report similarly low recurrence after complete resection.^{4,8,55} Moreover, in the 2025 meta-analysis of 6,612 PMP patients by Wei et al., elevated serum CA 19-9 (risk ratio [RR] 2.30), CA-125 (RR 3.39), and CEA (RR 3.16), a high Peritoneal Cancer Index (PCI > 20; RR 1.05), and incomplete cytoreduction (CC-2/3; RR 2.10) were associated with worse long-term survival after CRS-HIPEC.⁶⁸ Our pragmatic district-hospital surveillance plan comprises clinical evaluation and serum CEA, CA 19-9, and CA-125 every 3 to 6 months, with contrast-enhanced abdominopelvic CT or magnetic resonance imaging (MRI) at month 3 (CEA 1.8 ng/mL, CA 19-9 14.2 U/mL, CA-125 11.6 U/mL, and PCI 0/39 in our patient) and then every 6 to 12 months for 5 years, so that early peritoneal recurrence may be detected while the PCI is low and the patient can be referred promptly to a tertiary CRS-HIPEC center; published follow-up schedules vary, and no universal standard exists.^{3,8,68}

This report has several limitations. First, it describes a single patient with a rare combination of lesions, so its findings cannot be generalized. Second, preoperative serum tumor markers (CEA, CA 19-9, and CA-125) and intraoperative frozen-section histopathology were unavailable during the emergency admission at our regional referral hospital, so operative

escalation relied on macroscopic surgical judgment; the postoperative tumor markers and the permanent sections (12 step sections) supported the intraoperative strategy. Third, extended immunohistochemistry (CK20, CK7, CDX2, SATB2, MUC2, PAX8) and next-generation sequencing for somatic KRAS, GNAS, and TP53 mutations were not performed because of regional resource constraints, although the H&E morphology was diagnostic under both PSOGI and WHO 5th edition criteria.^{3,4,6}

Molecular background of LAMN and peritoneal spread

The molecular pathobiology of low-grade appendiceal mucinous neoplasms may explain why lesions with acellular extra-appendiceal mucin occupy an intermediate position between benign adenomas and infiltrative adenocarcinomas.^{3,7,41} Unlike conventional colorectal adenocarcinomas, which evolve through the APC–KRAS–SMAD4–TP53 chromosomal instability sequence and show destructive desmoplastic stromal invasion, tumor budding, and lymphovascular metastasis, LAMNs are characterized by co-occurring activating mutations in KRAS (codons 12, 13, and 61) and GNAS (codons 201 and 227), with wild-type TP53 and preserved mismatch-repair proficiency in most low-grade tumors.^{3,7,56} Genomic studies report KRAS mutations in 50% to 98.1% of LAMNs and GNAS mutations in about half, with KRAS–GNAS co-mutation in 65% to 85%.^{3,7,56} The mutant Gs α subunit encoded by GNAS R201C or R201H has impaired intrinsic GTPase activity, locking adenylyl cyclase in a constitutively active state that raises intracellular cyclic adenosine monophosphate (cAMP) and activates protein kinase A (PKA) phosphorylation of the cAMP-response element-binding protein (CREB).⁵⁷ This cAMP–PKA signaling acts together with KRAS-mediated MAPK/ERK and PI3K/AKT signaling to increase the production of secreted gel-forming mucins, principally MUC2 and MUC5AC.^{3,7}

Biophysically, MUC2 is a large, heavily O-glycosylated polymer that forms net-like hydrogels and expands more than 1,000-fold upon secretion.⁶⁹ Because the appendiceal orifice is narrow—and in our patient may also have been compressed by the pericolic phlegmon of the perforated diverticulitis—continuous secretion of viscoelastic mucin converts the appendix into a closed, high-pressure chamber.^{3,23} As intraluminal pressure exceeds the capillary perfusion pressure (25.0–30.0 mmHg) in the lamina propria, the mucosal crypts undergo pressure atrophy, the lymphoid follicles involute, and the lamina muscularis mucosae fragments and disappears, leaving a flat or undulating monolayer of mucin-producing epithelium on a densely hyalinized collagenous submucosa (Panels A–C of Figure 3 and Table 2).^{3,6,7,31} When acellular mucin dissects through the vascular hiatuses of the muscularis propria to reach the subserosa and mesoappendix (Panel D of Figure 3), the extruded mucin gel resists rapid macrophage clearance and elicits a foreign-body-like polymorphonuclear and mononuclear serosal reaction.

If intact strips of low-grade neoplastic epithelium escape into the free peritoneal cavity before or during surgical manipulation, they do not invade the peritoneal mesothelium directly through matrix metalloproteinases; rather, they float passively within the mucin along physiological peritoneal fluid currents.^{3,6,17} Carried by the circulation of peritoneal fluid and by negative subdiaphragmatic inspiratory pressure, mucinous aggregates are swept toward sites of peritoneal fluid resorption—the greater omentum, the right subphrenic space, the retrohepatic vena caval recess, and the gravity-dependent pelvic cul-de-sac—whereas the mobile small-bowel surfaces are relatively spared by peristalsis until late-stage disease (the Sugarbaker redistribution phenomenon).^{6,17} By recognizing the

tense 9.50 × 3.00 × 2.50 cm appendix intraoperatively and performing an en bloc no-touch right hemicolectomy with omentectomy and 4,000 mL of warm saline lavage before free rupture, the surgical team removed the lesion while the mucin was still localized, and the PCI was 0/39 both at laparotomy and on the 90-day CT.

Hemodynamic, microbiological, and anastomotic considerations in perforated right-sided diverticulitis

The second component of the presentation—multifocal ascending colon diverticulitis with two perforations and 50.0 mL of purulent fluid along the right paracolic gutter—illustrates the clinical divergence between uncomplicated and complicated right-sided diverticular disease.^{29,30,32} In Southeast Asian and East Asian populations, right-sided diverticulitis accounts for most colonic diverticulitis admissions and predominantly affects patients in their fourth to sixth decades of life.^{27,28} Solitary congenital cecal diverticula are classically described as containing all four layers of the bowel wall (mucosa, submucosa, muscularis propria, and serosa), whereas multifocal ascending colon diverticula, as in our patient, often represent false (pulsion) diverticula formed by herniation of mucosa and submucosa where the vasa recta penetrate the circular muscle along the mesenteric border; one review considers most right-sided diverticula to be false.^{30,58,70} When inspissated fecaliths occlude the narrow necks of these diverticula, bacterial overgrowth of organisms such as *Escherichia coli*, *Klebsiella pneumoniae*, and *Bacteroides fragilis* can cause necrotizing mucosal ulceration and microperforation.^{32,71}

In our patient, 72 hours of empirical intravenous ceftriaxone at the referring hospital did not halt disease progression, because two diverticula had already perforated (3.5 mm and 4.0 mm defects), creating an undrained 50.0 mL purulent collection along the right paracolic gutter (modified Hinchey stage IIb/III), and because ceftriaxone lacks reliable activity against anaerobes such as *Bacteroides* species; metronidazole was added only after referral.^{32,72} The systemic effect of this uncontained purulent peritonitis was reflected in the marked shift from lymphoid to myeloid cells (Table 1): emergency granulopoiesis raised neutrophils to 91.0% (ANC 17.45 × 10³/μL), while lymphocytes fell to 4.2% (ALC 0.81 × 10³/μL) and eosinophils to 0.04%, giving an NLR of 21.67, an SII of 3,358.85 × 10³/μL, and a SIRI of 18.10 × 10³/μL. In sepsis, lymphopenia is attributed mainly to lymphocyte apoptosis and redistribution,⁷³ here probably triggered by toll-like receptor 4 (TLR4) lipopolysaccharide signaling and an endogenous cortisol surge with splenic lymphocyte sequestration, and eosinopenia reflects the adrenal glucocorticoid and epinephrine stress response.⁷⁴ At the same time, interleukin-1 β , interleukin-6, and tumor necrosis factor- α mediate splanchnic vasodilation and capillary leak, which, combined with 48 hours of vomiting, reduced the effective arterial blood volume and lowered the CKD-EPI eGFR to 46.8 mL/min/1.73 m² (serum creatinine 1.40 mg/dL).

From a surgical perspective, the purulent peritonitis (modified Hinchey stage IIb/III), the pre-renal azotemia, and the dense fibropurulent adhesions in four compartments led the team to choose fecal diversion over primary ileo-transverse anastomosis after right hemicolectomy. In a heavily contaminated peritoneal bed with 50.0 mL of frank pus, neutrophil elastase, host matrix metalloproteinases (MMP-1, MMP-8, and MMP-9), and bacterial collagenases degrade submucosal type I and type III collagen along a newly constructed suture or staple line during the critical lag phase of anastomotic healing^{75,76} (72 to 120 hours), increasing anastomotic leak rates beyond 15% to 20%. Moreover, in a patient with a concurrent pT4a LAMN, an anastomotic leak and

relaparotomy would carry a high septic mortality and could disrupt the retroperitoneal and peritoneal tissue planes, potentially facilitating retroperitoneal mucinous implantation.⁸ An end ileostomy with a closed transverse colon stump avoided the risk of an anastomotic leak, allowed early enteral feeding by POD 2–3, was followed by a 56.1% fall in the leukocyte count and a 37.1% fall in creatinine within 120 hours (discharge on POD 5), and preserved healthy, uninflamed bowel ends for elective restoration of continuity at month 3.

Mature cystic teratoma after menopause and lessons for district hospitals

The third component—a 10.20 × 10.44 × 10.46 cm (583.2 cm³) right ovarian mature cystic teratoma—shows the importance of age- and size-based gynecological decisions during complex abdominopelvic surgery.^{33,50,66,67} Mature cystic teratomas account for 69% of ovarian germ cell tumors and behave benignly in young women of reproductive age,³³ but their risk profile changes in older and postmenopausal women with tumors larger than 10 cm.^{50,66,67} The review by Cong et al.,³³ the case reports of Luo et al. (two patients)⁶⁷ and Palomba et al. (one patient),⁵⁰ and the 30-year series of 22 cases by Li et al.⁶⁶ report malignant transformation in about 1% to 3% of mature cystic teratomas, with older age, tumor diameter of 10 cm or more, and solid wall thickening as the main clinical predictors. In 80% to 85% of transformed cases, the ectodermal squamous lining develops invasive squamous cell carcinoma (MCT-SCC; 59.1% in the series of Li et al.), with recurrent TP53, PIK3CA, and CDKN2A alterations, whereas adenocarcinoma, melanoma, and carcinoid tumors account for most of the remainder.^{33,66,67} Because preoperative CT and ultrasound cannot reliably exclude microscopic squamous cell carcinoma within the Rokitansky protuberance of a 10.46 cm postmenopausal dermoid cyst, and because intraoperative rupture spills sebaceous irritants and potentially malignant cells across the peritoneum, oophorectomy is the standard operation for mature cystic teratoma in postmenopausal women,^{33,77} and total abdominal hysterectomy with bilateral salpingo-oophorectomy was performed as the oncological procedure in this patient.⁵⁰

Furthermore, the diagnostic overlap between appendiceal mucinous neoplasms and right adnexal masses is one of the most consequential pitfalls in female pelvic surgery.^{25,26,35,36,52} As reported by Yu et al. in a cohort²⁵ and reviewed by Kawecka et al.,²⁶ primary appendiceal mucinous tumors often extend into the right hemipelvis or metastasize to the ovaries, mimicking primary ovarian tumors on ultrasonography and CT; in our patient, conversely, a primary ovarian teratoma (583.2 cm³) obscured a primary appendiceal LAMN (37.31 cm³) on imaging. When a postmenopausal woman undergoes surgery for a presumed adnexal mass without systematic inspection and palpation of the whole appendix and cecum, an occult LAMN or HAMN can be left in situ or lacerated during adnexal adhesiolysis, leading to delayed presentation with pseudomyxoma peritonei.^{25,26,35,36,52}

The experience at Klungkung Regional General Hospital suggests practical steps for secondary and district referral hospitals in Indonesia and other low- and middle-income countries.^{35,36} First, when cross-sectional imaging in a woman aged 50 years or older shows a dominant right adnexal mass with ipsilateral pericolic fat stranding or a periappendicular phlegmon, preoperative preparation may include baseline serum CEA, CA 19-9, and CA-125,^{3,26} mechanical and antibiotic bowel preparation where feasible, stoma site marking, and a joint pre-incision briefing between the digestive surgery and obstetrics-gynecology teams. Second, during laparotomy for any right lower quadrant phlegmon or pelvic mass, the appendix

and ascending colon should be inspected and palpated before closure;¹⁰ if a tense, mucin-distended appendix coexists with cecal or ascending colon disease, or cannot be resected with a negative cecal margin in the absence of frozen section, formal en bloc oncologic right hemicolectomy is a reasonable option.³ Third, histological sampling of the entire appendiceal circumference and mesoappendix,⁶ together with structured five-year tumor-marker and CT/MRI surveillance,^{3,4} helps patients treated in regional hospitals receive care consistent with PSOGI and ASCRS guidance.

5. Conclusion

The coexistence of a pT4a low-grade appendiceal mucinous neoplasm (9.50 × 3.00 × 2.50 cm; 37.31 cm³), multifocal perforated ascending colon diverticulitis (modified Hinchey stage IIb/III with 50.0 mL of purulent paracolic fluid), and an ipsilateral mature ovarian cystic teratoma (10.20 × 10.44 × 10.46 cm; 583.2 cm³) in a 52-year-old postmenopausal woman made the appendiceal neoplasm difficult to recognize before surgery: the 15.6-fold larger dermoid cyst and the pericolic diverticular phlegmon obscured it, and the appendiceal lesion seen on CT was read as an abscess. In a regional referral hospital without intraoperative frozen-section histopathology, systematic four-quadrant exploration and coordinated multidisciplinary surgery—en bloc oncologic right hemicolectomy with high vascular ligation, end ileostomy, and transverse colon stump closure, combined with total abdominal hysterectomy and bilateral salpingo-oophorectomy—removed all three lesions in one operation without mucin spillage or anastomotic complications, and the patient recovered within 5 days (WBC –56.1%, NLR –86.1%, serum creatinine –37.1%). Complete histological staging combined with five-year surveillance using serum tumor markers (CEA, CA 19-9, CA-125) and CT or MRI is planned for the early detection of pseudomyxoma peritonei; at 90 days there was no evidence of peritoneal disease (PCI 0/39), and the patient was ready for restoration of intestinal continuity.

6. Declarations

Ethics Approval and Consent to Participate

This case report was reviewed and approved by CMHC Research Center Ethics Committee, Indonesia (Protocol No. 2026/0418), and received institutional clinical authorization from Klungkung Regional General Hospital. All procedures were conducted in accordance with the Declaration of Helsinki (2013 Fortaleza revision). Written informed consent to participate and for the surgical procedures was obtained directly from the patient.

Consent for Publication

Written informed consent for the publication of de-identified clinical data, laboratory results, diagnostic radiological images, and histopathological photomicrographs was obtained from the patient before manuscript submission. All diagnostic images were stripped of identifying metadata in accordance with ICMJE privacy standards.

Availability of Data and Materials

All clinical, biochemical, morphometric, radiological, and histopathological data supporting the findings of this report are presented in the manuscript, its tables (Tables 1–5), and its figures (Figures 1–5). De-identified source records are available from the corresponding author on reasonable request.

Artificial Intelligence Use Statement

None.

Author Contribution Statement

I.M.A.S.: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing. M.Y.: Conceptualization, Investigation, Methodology, Supervision, Validation, Writing – review & editing. Both authors read and approved the final version of the manuscript.

Funding Statement

None.

Conflict of Interest / Competing Interests

The authors declare that they have no financial or non-financial competing interests related to this work.

Acknowledgements

The authors thank the nursing, anesthesiology, radiology, anatomical pathology, and intensive care unit staff of Klungkung Regional General Hospital, Semarapura, Bali, Indonesia, for their multidisciplinary support during the perioperative care and follow-up of the patient.

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