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Etiological Spectrum and Diagnostic Yield of Colonoscopy in Patients with Computed Tomography-Detected Colonic Wall Thickening.

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Abstract
Background: Colonic wall thickening detected on computed tomography is a frequent but nonspecific radiological finding. It may reflect transient or benign conditions, inflammatory or infectious disease, ischemia, polyps, or colorectal malignancy. The absence of a uniform diagnostic pathway creates uncertainty regarding the need for colonoscopic evaluation. ***Objective:*** To determine the diagnostic yield and final etiological spectrum among patients with computed tomography-detected colonic wall thickening who underwent colonoscopic evaluation. ***Methods:*** This retrospective cross-sectional study included 119 patients with computed tomography-detected colonic wall thickening who underwent colonoscopic evaluation and subsequent etiological assessment. Demographic characteristics, predominant presenting symptoms, and final diagnoses established through integration of endoscopic findings with available histopathological, microbiological, radiological, and clinical data were analyzed. Continuous data are presented as mean ± standard deviation, and categorical data as number, percentage, and 95% confidence interval. ***Results:*** The mean age was 48.0 ± 5.1 years, and 71 patients (59.7%) were male. Abdominal pain was the most frequent predominant presenting symptom (36, 30.3%), followed by weight loss (29, 24.4%), altered bowel habits (26, 21.8%), per rectal bleeding (17, 14.3%), and nausea (11, 9.2%). A pathological etiology was identified in 80 patients, giving an overall diagnostic yield of 67.2% (95% confidence interval, 58.4%-75.0%). Colonic malignancy was the most common diagnosis (31, 26.1%), followed by inflammatory bowel disease (21, 17.6%), intestinal tuberculosis (17, 14.3%), colonic polyps (5, 4.2%), ischemic colitis (4, 3.4%), and amebic colitis (2, 1.7%). No significant colonic pathology was identified in 39 patients (32.8%). ***Conclusion:*** Computed tomography-detected colonic wall thickening was associated with an identifiable pathological etiology in approximately two-thirds of patients. Colonic malignancy was the single most common diagnosis, while inflammatory bowel disease and intestinal tuberculosis together constituted a substantial proportion of cases. These findings support structured colonoscopic and tissue-based evaluation, interpreted within the clinical and regional epidemiological context.

Keywords: colonic wall thickening; computed tomography; colonoscopy; colorectal malignancy; inflammatory bowel disease; intestinal tuberculosis.

INTRODUCTION
Colonic wall thickening is commonly encountered on abdominal computed tomography and may be focal, segmental, or diffuse. Its interpretation is influenced by luminal distension, the length and symmetry of involvement, enhancement pattern, pericolic changes, and associated clinical findings. Focal irregular or asymmetric thickening raises concern for neoplasia, whereas segmental or diffuse involvement more often reflects inflammatory, infectious, ischemic, or edematous processes; however, substantial overlap exists [1].

Although computed tomography can narrow the differential diagnosis, colonic wall thickening is not itself an etiological diagnosis. Inadequate luminal distension, retained fecal material, transient contraction, and systemic conditions causing bowel wall edema may produce apparent thickening without clinically important mucosal disease. Conversely, early malignancy, inflammatory bowel disease, intestinal infection, and ischemia may be detected initially as mural thickening before a definitive diagnosis is established. This diagnostic ambiguity has led to variable clinical practice and the absence of a universally accepted management algorithm [2].

Several retrospective series have demonstrated clinically important pathology after colonoscopic evaluation of computed tomography-detected colonic wall thickening. Moraitis et al. reported neoplasia in a clinically relevant proportion of patients and emphasized that malignancy may be present even in the absence of gastrointestinal symptoms [3]. Wolff et al. identified new colorectal adenocarcinoma and inflammatory bowel disease among patients investigated for abdominal pain and colonic thickening [4]. Eskaros et al. found concordant abnormal colonoscopic findings in 64% of patients, with particularly strong correlation when a mass lesion accompanied wall thickening on computed tomography [5]. Nicholson et al. similarly reported a high yield of pathology after colonoscopy and biopsy [6]

The etiological distribution is also likely to vary by population. In regions where intestinal tuberculosis remains prevalent, infectious causes may account for a larger proportion of bowel wall thickening than in Western cohorts. A locally relevant description of the diagnostic spectrum is therefore necessary to guide clinical prioritization and counseling. The present study aimed to determine the diagnostic yield of colonoscopic evaluation and describe the final etiological spectrum among patients with computed tomography-detected colonic wall thickening.

MATERIALS AND METHODS

Study design and setting

This was a retrospective cross-sectional observational study conducted in the Department of Medical Gastroenterology, Gandhi Hospital, Secunderabad over the period 10 month period (Jan 2024 – Oct 2024). The manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology recommendations [7].

Study population

The study included 119 eligible patients in whom colonic wall thickening was reported on abdominal computed tomography and who subsequently underwent colonoscopic evaluation with sufficient clinical and diagnostic information to assign a final etiology. The predefined exclusion criterion was acute intestinal obstruction that precluded complete colonoscopic evaluation.

Data collection

Demographic variables included age and sex. The symptom documented as the principal presenting complaint was categorized as abdominal pain, weight loss, altered bowel habits, per rectal bleeding, or nausea. Computed tomography reports were reviewed for the documented presence of colonic wall thickening. Colonoscopy reports, histopathology records, microbiological investigations, radiological findings, and relevant clinical documentation were reviewed to establish the final diagnosis.

Diagnostic classification

Final etiological diagnoses were assigned from the integrated clinical record rather than from colonoscopic appearance alone. Colonic malignancy required histopathological confirmation. Inflammatory bowel disease, intestinal tuberculosis, ischemic colitis, and amebic colitis were classified according to the documented final diagnosis based on the combination of endoscopic, histopathological, microbiological, radiological, and clinical evidence available for each patient. Colonic polyps were categorized from colonoscopic and pathological documentation. Patients without a clinically significant abnormality on colonoscopy and subsequent diagnostic assessment were classified as having no significant colonic pathology.

Outcome measures

The primary outcome was the overall diagnostic yield, defined as the proportion of patients in whom a pathological etiology was established after colonoscopic evaluation and subsequent diagnostic work-up. Secondary outcomes were the distribution of final etiological diagnoses and the demographic and clinical profile of the study population.

Statistical analysis

Age is reported as mean ± standard deviation. Categorical variables are presented as number and percentage. The 95% confidence intervals for proportions were calculated using the Wilson score method. The diagnostic yield was calculated as the number of patients with an identified pathological etiology divided by the total study population. Because only aggregate, unstratified data were available for the present analysis, valid between-group hypothesis tests, regression models, test statistics, and adjusted effect estimates could not be generated without risking spurious inference. Accordingly, proportions with 95% confidence intervals were used as the principal effect estimates. Analyses were performed using Python version 3.13.5 (Python Software Foundation, Wilmington, Delaware, United States), pandas version 2.2.3, SciPy version 1.17.0, statsmodels version 0.14.6, and scikit-learn version 1.8.0.

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was obtained before participation. Patient confidentiality was maintained throughout data extraction and analysis.

RESULTS

Demographic characteristics

A total of 119 patients were included. Their mean age was 48.0 ± 5.1 years. There were 71 males (59.7%; 95% confidence interval, 50.7%-68.0%) and 48 females (40.3%; 95% confidence interval, 32.0%-49.3%). The baseline demographic characteristics are summarized in Table 1.

Table 1. Baseline demographic characteristics of the study population (N = 119)

Characteristic	Value
Age, years	48.0 ± 5.1 (mean ± standard deviation)
Male sex	71 (59.7%)
Female sex	48 (40.3%)

Values are presented as mean ± standard deviation or number (percentage), as appropriate. N denotes the total number of patients.

Clinical presentation

Abdominal pain was the most frequent predominant presenting symptom, reported by 36 patients (30.3%), followed by weight loss in 29 (24.4%), altered bowel habits in 26 (21.8%), per rectal bleeding in 17 (14.3%), and nausea in 11 (9.2%). The distribution of predominant presenting symptoms is shown in Table 2.

Table 2. Predominant clinical presentation of the study population (N = 119)

Predominant presenting symptom	Number	Percentage
Abdominal pain	36	30.3
Weight loss	29	24.4
Altered bowel habits	26	21.8
Per rectal bleeding	17	14.3
Nausea	11	9.2
Total	**119**	**100.0**

Each patient was categorized according to the principal presenting complaint documented at presentation. Percentages use the total cohort as the denominator. Confidence intervals were calculated using the Wilson score method.

Diagnostic yield and etiological spectrum

A pathological etiology was identified in 80 of 119 patients, corresponding to an overall diagnostic yield of 67.2% (95% confidence interval, 58.4%-75.0%). No significant colonic pathology was identified in 39 patients (32.8%). Colonic malignancy was the most common final diagnosis, occurring in 31 patients (26.1%), followed by inflammatory bowel disease in 21 (17.6%) and intestinal tuberculosis in 17 (14.3%). The complete etiological spectrum is presented in Table 3.

Table 3. Etiological spectrum of colonic wall thickening detected on computed tomography (N = 119)

Final etiological diagnosis	Number	Percentage
No significant colonic pathology	39	32.8
Colonic malignancy	31	26.1
Inflammatory bowel disease	21	17.6
Intestinal tuberculosis	17	14.3
Colonic polyp	5	4.2
Ischemic colitis	4	3.4
Amebic colitis	2	1.7
Total	**119**	**100.0**

Final diagnoses were assigned after integration of colonoscopic findings with available histopathological, microbiological, radiological, and clinical evidence; they should not be interpreted as diagnoses made by colonoscopic appearance alone. Percentages use the total cohort as the denominator. Confidence intervals were calculated using the Wilson score method.

DISCUSSION

The present study demonstrates that computed tomography-detected colonic wall thickening frequently represents clinically important disease. A pathological etiology was established in 67.2% of patients, while 32.8% had no significant colonic pathology. Colonic malignancy was the most common single diagnosis, accounting for 26.1% of the cohort. Inflammatory bowel disease and intestinal tuberculosis together accounted for an additional 31.9%, underscoring the broad differential diagnosis and the importance of tissue-based and clinicopathological correlation.

The overall diagnostic yield is closely aligned with the 64% concordance between computed tomography-detected wall thickening and abnormal colonoscopy reported by Eskaros et al. [5]. It is also consistent with the broader literature showing that more than half of patients referred for endoscopic assessment have a relevant abnormality. Uzzaman et al. found endoscopic abnormalities at the corresponding site of bowel wall thickening in 57.6% of patients, including malignant lesions in 21.8% [8]. Al-Khowaiter et al. reported normal colonoscopy in only 24% of symptomatic patients with no previously known gastrointestinal disease [9]. These observations support the clinical relevance of colonic wall thickening when interpreted in an appropriate symptomatic context.

The malignancy rate of 26.1% in the present cohort is particularly important. It is similar to the 28.8% malignancy rate reported by Baş and Pakoz, whose cohort also had a 33.3% rate of normal colonoscopy [11]. Nicholson et al. reported malignancy in 26% and adenoma in 25% of patients undergoing colonoscopy for computed tomography-detected wall thickening [6]. In contrast, Wolff et al. identified colorectal adenocarcinoma in 7.4% of patients [4], and the prospective Indian study by Khairnar et al. reported cancer in approximately 11.7% [10]. Differences among studies likely reflect variation in referral pathways, whether the thickening was incidental or symptom-driven, exclusion of obvious masses, age distribution, anemia prevalence, computed tomography technique, and the threshold used by radiologists to report thickening.

Patient selection also explains why some cohorts report substantially higher or lower neoplasia rates. Karacin et al. studied incidentally detected colonic wall thickening and found neoplastic lesions in 19.6% and adenocarcinoma in 6.5%; focal, asymmetric, and moderate-to-severe thickening independently predicted neoplasia [12]. Akbas et al., in contrast, reported malignancy in 52 of 116 patients selected for indeterminate wall thickening and investigated laboratory predictors of malignancy [13]. Such heterogeneity indicates that a single prevalence estimate should not be applied indiscriminately. The clinical probability of malignancy depends on the imaging phenotype and the pretest risk profile.

More recent work has attempted to refine this risk stratification. Pothoulakis et al. found that localized wall thickening, low hematocrit, and elevated platelet count were independently associated with colorectal carcinoma, whereas younger age and elevated platelet count were associated with inflammatory bowel disease [14]. Baş and Pakoz also found malignancy to be concentrated among older patients and those with marked anemia [11]. The present aggregate dataset did not include hemoglobin, platelet count, segmental distribution, thickness, symmetry, or enhancement characteristics; therefore, predictor analysis was not possible. Incorporating these variables in future studies would allow development of a clinically useful triage model.

The proportion of inflammatory bowel disease in the present study was 17.6%, higher than that reported in several Western cohorts but clinically plausible in a symptom-selected gastroenterology population. Importantly, intestinal tuberculosis accounted for 14.3% of all cases. This finding highlights the regional epidemiological context. In a prospective study from a tropical country, Kumar et al. found tuberculosis in 48% of patients with ileal and/or cecal thickening and Crohn's disease in 20% [15]. Although that study focused on the ileocecal region, it illustrates why tuberculosis must remain prominent in the differential diagnosis of bowel wall thickening in endemic settings. Differentiating intestinal tuberculosis from Crohn's disease requires integration of endoscopic distribution, histology, microbiology, radiology, systemic features, and therapeutic response; colonoscopic appearance alone is insufficient.

Approximately one-third of patients had no significant colonic pathology despite computed tomography-reported thickening. This discordance should not be interpreted as failure of either modality. Computed tomography and colonoscopy evaluate different components of the bowel wall: computed tomography depicts mural and extramural changes, whereas colonoscopy primarily assesses the mucosal surface. Apparent thickening can arise from suboptimal luminal distension, transient peristalsis, fecal residue, or systemic edema, and some inflammatory or ischemic processes may resolve before endoscopic evaluation [1,2]. Conversely, submucosal or extrinsic disease may not produce a conspicuous mucosal abnormality. Clinical follow-up remains important when symptoms persist despite a normal colonoscopic examination.

The symptom distribution in this cohort also deserves attention. Abdominal pain was the most common predominant presentation, but weight loss, altered bowel habits, and per rectal bleeding together accounted for more than half of

presentations. These symptoms overlap with malignancy, inflammatory bowel disease, intestinal infection, and ischemia and therefore cannot independently establish etiology. Nevertheless, the coexistence of alarm features such as weight loss or bleeding should lower the threshold for timely colonoscopy and biopsy, especially when computed tomography shows focal, irregular, or asymmetric thickening.

From a practical perspective, the findings support a structured diagnostic pathway rather than automatic reassurance or indiscriminate emergency intervention. The initial assessment should verify whether the thickening is focal or diffuse, symmetric or asymmetric, and whether it is associated with a mass, lymphadenopathy, obstruction, disproportionate fat stranding, or systemic edema. Clinical prioritization should incorporate age, anemia, platelet count, weight loss, bleeding, and family history. Colonoscopy with targeted biopsy is appropriate in most clinically stable patients with unexplained colonic wall thickening, but final diagnosis must be based on integrated clinicopathological evidence.

Strengths

Use of a clearly defined imaging-based entry criterion, inclusion of a clinically meaningful sample of 119 patients, reporting of the complete etiological spectrum rather than malignancy alone, and separation of final etiological diagnosis from colonoscopic appearance. The study also reflects a setting in which intestinal tuberculosis remains relevant, increasing the applicability of the findings to tropical and resource-constrained regions.

Limitations

Only patients who underwent colonoscopy were included, so the results cannot be generalized to all individuals with incidentally detected wall thickening. The categorization of symptoms as a single predominant complaint may underrepresent coexisting symptoms.

CONCLUSION

Computed tomography-detected colonic wall thickening was associated with an identifiable pathological etiology in approximately two-thirds of patients in this cohort. Colonic malignancy was the most common individual diagnosis, while inflammatory bowel disease and intestinal tuberculosis comprised a substantial additional burden. Colonoscopic evaluation with appropriate biopsy and subsequent clinicopathological correlation is therefore warranted in clinically suitable patients, particularly when alarm symptoms or high-risk imaging features are present. Prospective studies incorporating standardized computed tomography characteristics, laboratory variables, and patient-level outcome data are needed to develop reliable risk-stratification algorithms.

Declarations

Ethics approval and consent to participate: The study was approved by the Institutional Ethics Committee of Gandhi Medical College. Written informed consent was obtained from all participants.

Conflicts of interest: The authors declare no conflicts of interest.

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