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CONTENTS

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1. Title page
2. Abstract (Structured)
3. Introduction
4. Materials and Methods
5. Results (With tables ar figures)
6. Discussion
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Editorial

Breath Holding Attack

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Breath holding attacks are common among healthy young infant and children from 6 months to 5 years[1]. Family history may be present among 20-30% cases. Incidence is more among male children[2]. Pattern of mode of inheritance is automat dominant. Recurrent, early onset and severe breath holdingspells are associated with several genetic syndromes such as 16p 11, 2 micro deletion syndrome and Riley day syndrome[3]. Etiopathogenisis is unknown but its thoughts most likely multifactorial. Inability to control the autonomic nervous system play significant role in the pathogenisis[4]. During frustration and anger overactivity of respiratory muscle associated with intense cortical activity occur. To control the over activity of the cortex and respiratory muscle children hold the breath up to the point of cerebral hypoxia. Para-sympathetic hyperactivity vigil inhibition is also responsible for pathogenesis of pallid breath holding spell[1]. In some studies found delayed myelination associated with breath holding attack among preschool children[5]. It assumes that breath holding spelling may be due to imbalance between oxidant and anti-oxidant system of the body[6]. Breath holding attack is also associated with Iron deficiency anemia. Some thoughts that deregulation of autonomic nervous system is responsible for breath holding attack which occurs due to iron deficiency anemia[7]. The characteristics of breath holding attack are stay less than 1minute, associated with bradycardia and normal EEG findings[2]. There are many differential diagnoses of breath holding attack but top of most is epilepsy. Epilepsy, Sudden breath holdingduring sleep, Sepsis, Hyperplasia (Stiff body syndrome or startle disease), Shuddering, Congenital laryengeal stridor, Laryengospasm, and Whooping cough can be considered as differential diagnoses. That all can be differentiated by characteristics clinicalfeatures[8]. there is no role of investigation for diagnosis. Complete blood count and serum iron should be done if anemia is present[9]. ECG should be done to excude prolong QT syndrome. Though It is benign condition[10]. But it may causes anxiety, depression and psychosocial problem among parents of severe breath holding attack children[11]. Affected children have normal intelligence and normal neurological devlopment[12]. Main crucial part of the treatment is to explain the disease pattern and also to give the reassurance to the parent of child[13].Treatment of iron deficiency anemia by iron reduces the frequencies of attack[14]. I n many studies found that Glycopyrotol a synthetic quaternary ammonium compound effective in the treatment of children suffering from severe pallid breath holding spells[15]. Fluoxetine also found effective in the treatment of Pallid breath holding spells of children[16]. Cardiac pacemaker implantation is effective treatment in cyanotic breath holding spells associated with prolonged a systole[17]. The result of many studies were infavor that Pierceton is effective in the treatment of breath holding attack[18]. Theophylline is also effective in the treatment of breath holding attack[19]. It disappear spontaneously before school going age. Prognosis is excellent. So, cornerstone of treatment is to give confidential reassurance and counseling to the family members.

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Original Article

Depression and Anxiety in Patients with Chronic Kidney Disease: Prevalence, Risk Factors and Impact on Quality of Life

***Hasan MJ[1], Pathan MAS[2]**

Abstract

Background*: Chronic kidney disease (CKD) represents a significant challenge to public health, often accompanied by significant psychological distress, including depression and anxiety. These psychological disorders can negatively influence patients' quality of life (QoL) and adherence to treatment.*

Objective*: The present study sought to determine the prevalence of depression and anxiety in individuals with CKD, examine related risk factors and assess their effect on QoL.*

Methods and Materials*: A cross-sectional study was carried out at the Department of Nephrology in collaboration with the Department of Psychiatry at Community-Based Medical College, Bangladesh, between January 2023 and December 2024.*

Results*: A total of 83 CKD patients were recruited using purposive sampling. Data were collected through structured questionnaires, including validated scales for depression, anxiety and QoL assessment. Demographic, clinical and psychosocial factors were analyzedto determine their association with mental health outcomes. Data were analyzed using SPSS version 23.0, employing both descriptive and inferential statistical methods to examine the associations between variables. Among 83 CKD patients, depression prevalence was 38.6% and anxiety prevalence was 31.3%. Dialysis dependency (OR: 2.45 for depression, 2.12 for anxiety), low socioeconomic status (OR: 1.89 for depression, 1.76 for anxiety) and comorbidities were significant risk factors. Depression correlated with reduced physical health (r=-0.52) and emotional well-being (r =-0.61), while anxiety showed similar negative correlations (r =-0.48 and r =-0.56, respectively).*

Conclusions*: Depression and anxiety are common among individuals with CKD and substantially reduce their quality of life. Prompt screening and the incorporation of mental health interventions are crucial for alleviating psychological distress and enhancing patient outcomes. Future studies should prioritize longitudinal research and tailored strategies to lessen the mental health impact on this group.*

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Keywords: *Diabetic Retinopathy, Young-Onset Diabetes, Late-Onset Diabetes, Disease Progression, Vision Loss, Hyperglycemia*

Introduction

Chronic kidney disease (CKD) poses a major worldwide health challenge, impacting more than 850 million individuals globally, with its occurrence expected to increase alongside rising rates of diabetes, hypertension and an aging population[1]. CKD is a gradually advancing disorder that frequently progresses to end-stage renal disease (ESRD), requiring dialysis or kidney transplantation to sustain life[2]. In addition to its physical effects, CKD is increasingly acknowledged for its significant psychological burden, with depression and anxiety ranking among the most common mental health issues in affected individuals[3]. Depression and anxiety are widespread among CKD patients, with research considerably greater than those seen in the general population, highlighting the distinct psychological indicating that depression affects between 20.0% and 40.0% of patients, while anxiety impacts roughly 25.0% to 30.0% of this population[4,5]. These figures are challenges experienced by individuals with CKD[6]. Depression and anxiety not only reduce patients' quality of life but also lead to adverse clinical outcomes, such as poortreatment adherence, faster disease progression andhigher mortality rates[7,8]. For instance, depressive symptoms

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have been linked to misseddialysis sessions and poor dietary compliance, while anxiety is associated with heightened perceptions of symptom severity and reducedengagementin self-care[9,10]. The development of depression and anxiety in CKD patients is influenced by multiple factors, encompassing biological, psychological and social dimensions. Disease-specific elements, including advanced CKD stages,reliance on dialysis and coexisting conditions such as cardiovascular disease and diabetes, play a major role in triggering psychological distress[11]. Additionally, psychosocial factors, including low socioeconomic status, limited social support and financial strain, further exacerbate mental health challenges in this population[12]. The burden of managing a chronic illness, coupled with the demands of dialysis or transplantation, often leads to feelings of helplessness and hopelessness, which can precipitate or worsen mental health conditions[13]. Despite their high prevalence and harmful effects, depression and anxiety in CKD patients remain largely underdiagnosed and inadequately treated[14]. This care gap is particularly concerning, as accumulating evidence links untreated mental health disorders with worse clinical outcomes, including higher hospitalization rates and lower survival[15]. Quality of life (QoL), a multi-dimensional concept encompassing physical, emotional and social well-being, serves as a vital outcome measure in CKD patients, reflecting the overall impact of the disease and its treatment on daily life[16]. This study seeks to fill these gaps by evaluating the prevalence of depression and anxiety in CKD patients, identifying contributing risk factors and assessing their effects on QoL. By highlighting the psychological burden associated with CKD, the research aims to guide the development of targeted interventions that incorporate mental health care into standard CKD management, ultimately enhancing patient outcomes and quality of life.

Methods

This cross-sectional study was conducted in the Department of Nephrology in collaboration with the Department of Psychiatry outpatients in the Community-Based Medical College, Bangladesh, from January 2023 to December 2024. A total of 83 CKD patients wererecruited using purposive sampling. Inclusion criteria included patients aged 18 years or older diagnosed with CKD stages 3-5, including those on dialysis. Patients with severe cognitive impairment or acute psychiatric conditions were excluded. Data were collected through structured questionnaires, which included validated tools such as the Patient Health Questionnaire-9 (PHQ-9) for depression, the Generalized Anxiety Disorder-7 (GAD-7) scale for anxiety and the Kidney Disease Quality of Life-36 (KDQOL-36) for QoL assessment. Demographic, clinical and psychosocial data, including age, gender, socioeconomic status, comorbidities and dialysis dependency, were also recorded. Data analysis was performed using SPSS version 23.0. Descriptive statistics were used to summarize demographic and clinical characteristics. Prevalence rates of depression and anxiety were calculated and inferential statistics, including chi-square tests and logistic regression, were employed to identify risk factors associated with psychological distress. Pearson's correlation and multiple regression analyses were conducted to evaluate the impact of depression and anxiety on QoL. Ethical approval was obtained from the institutional review board and written informed consent was secured from all participants.

Results

The study enrolled 83 CKD patients, with a mean age of 52.4±12.3 years. Of these, 54.2% were male and 45.8% were female. Most participants (62.7%) were undergoing dialysis, while 37.3% were in the pre-dialysis stage. Hypertension (73.5%) and diabetes (56.6%) were the most frequently reported comorbidities. The prevalence of depression and anxiety, assessed using the PHQ-9 and GAD-7 scales, was 38.6% and 31.3%, respectively. Dialysis dependency, low socioeconomic status and the presence of comorbidities emerged as significant risk factors. Patients receiving dialysis had 2.45 times higher odds of depression (95% CI: 1.32-4.56) and 2.12 times higher odds of anxiety (95% CI: 1.18-3.81). Similarly, low socioeconomic status increased the likelihood of depression (OR: 1.89, 95% CI: 1.05-3.41) and anxiety (OR: 1.76, 95% CI: 1.02-3.04). Comorbidities, especially diabetes and hypertension, also significantly contributed to psychological distress. Depression was strongly negatively correlated with physical health ($r = -0.52$, $p<0.05$), emotional well-being ($r = -0.61$, $p<0.05$) and social functioning ($r = -0.45$, $p<0.05$).

Table I: Demographic characteristics of participants

Variable	Frequency (n)	Percentage (%)
Age (Mean±SD)	52.4±12.3	
Gender		
Male	45	54.20
Female	38	45.80
Education		
Primary	25	30.10
Secondary	35	42.20
Graduate & above	23	27.70

Likewise, anxiety showed negative correlations with physical health ($r = -0.48$, $p<0.05$), emotional well-being ($r = -0.56$, $p<0.05$) and social functioning ($r = -0.41$, $p<0.05$). These results highlight the substantial adverse impact of psychological distress on multiple aspects of quality of life in CKD patients.

Table II: Clinical characteristics of participants

Variable	Number (n)	Percentage (%)
CKD stages		
CKD stage 3	22	26.50
CKD stage 4	28	33.70
CKD stage 5	33	39.80
Dialysis dependency	52	62.70
Comorbidities		
Diabetes	47	56.60
Hypertension	61	73.50

Table III: Risk factors for depression and anxiety

Factors	Depression (OR, 95% CI)	Anxiety (OR, 95% CI)
DD	2.45 (1.32-4.56)	2.12 (1.18-3.81)
LSS	1.89 (1.05-3.41)	1.76 (1.02-3.04)
PC	2.10 (1.20-3.68)	1.95 (1.10-3.45)

DD: Dialysis dependency, LSS: Low socioeconomic status, PC: Presence of comorbidities

Table IV: Correlation between depression and QoL domains

Qol Domain	Correlation (r)	p value
Physical health	-0.52	<0.05
Emotional well-being	-0.61	<0.05
Social functioning	-0.45	<0.05

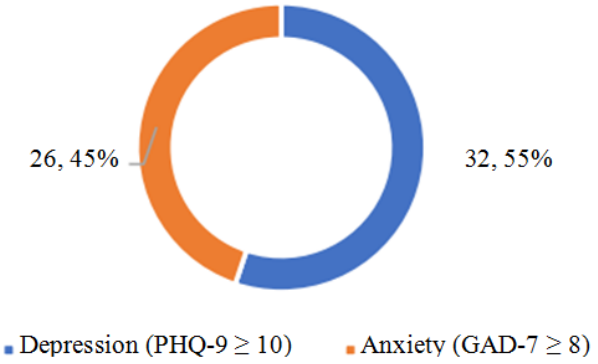

Figure 1: Prevalence of depression and anxiety

Table V: Correlation between anxiety and QoL domains

Qol Domain	Correlation (r)	p value
Physical health	-0.48	<0.05
Emotional well-being	-0.56	<0.05
Social functioning	-0041	<0.05

Discussion

The results of this study indicate a high prevalence of depression (38.6%) and anxiety (31.3%) among CKD patients, in line with previous research demonstrating elevated psychological distress in this population[17]. These rates far exceed those observed in the general population, highlighting the distinct mental health challenges faced by individuals with CKD[6]. The findings are consistent with global evidence, including a meta-analysis by Palmer et al., which reported depression prevalence of up to 40.0% in CKD patients[3]. and a study by Webster et al., which found anxiety affecting roughly 30.0% of this group[2]. Dialysis dependency was identified as a major risk factor for both depression and anxiety, with patients undergoing dialysis being 2.45 times more likely to experience depression and 2.12 times more likely to experience anxiety compared to those in the pre-dialysis stage. This finding is consistent with prior studies that have highlighted the psychological burden of dialysis, including its time-consuming nature, physical discomfort and impact on daily functioning[18,19]. Additionally, low socioeconomic status and the presence of comorbidities, particularly diabetes and hypertension, were strongly associated with psychological distress. These findings are supported by Cohen et al., who identified socioeconomic challenges and comorbid conditions as key contributors to mental health issues in CKD patients[6]. The negative impact of depression and anxiety on QoL was profound, with strong correlations observed across physical health, emotional well-being and social functioning domains. These results are consistent with studies by Perlman et al.[20] and Mujais et al.[16]. which demonstrated that psychological distress significantly impairs QoL in CKD patients. The physical health domain was particularly affected, likely due to the overlapping symptoms of CKD and mental health disorders, such as fatigue, sleep disturbances and reduced mobility. Emotional well-being was also severely impacted, reflecting the emotional toll of living with a chronic illness and the associated treatment burden. The findings of this study have important clinical implications. First, they highlight the need for routine screening for depression and anxiety in CKD patients, particularly those on dialysis or with low socioeconomic status. Early identification of psychological distress can facilitate timely interventions, potentially improving both mental health outcomes and QoL. Second, integrated care models that incorporate mental health services into CKD management should be prioritized. For example, collaborative care approaches involving nephrologists, psychologists and social workers have shown promise in addressing the complex needs of CKD patients[21] . Depression and anxiety are common among CKD patients and substantially

compromise their quality of life. Implementing early screening, integrated care approaches and targeted interventions is crucial for enhancing patient outcomes and overall well-being. However, this study's cross-sectional design limits the ability to infer causality. Additionally, the small sample size, single-center recruitment and reliance on self-reported measures may reduce generalizability and introduce bias. Future research should utilize longitudinal designs with larger and more diverse populations to confirm these findings and examine causal relationships.

Conclusion

Depression and anxiety are common in CKD patients, markedly impacting their quality of life, especially in physical and emotional aspects. Key risk factors include dialysis dependency, low socioeconomic status and comorbidities. Early screening and integrated mental health interventions are essential to address these challenges. Future studies should prioritize longitudinal research and the development of targeted interventions to reduce the psychological burden in this population.

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Original Article

Comparison of Progression of Diabetic Retinopathy among Young-Onset and Late-Onset Diabetes Mellitus

***Siddiqi MH[1], Khan Y[2], Kadir A[3]**

Abstract

Background*: Diabetic retinopathy (DR) is a major microvascular complication of diabetes mellitus (DM) and a leading cause of vision loss globally. The rate of progression of DR may differ between individuals diagnosed with diabetes at a younger age (young-onset diabetes mellitus, YODM) and those diagnosed later in life (late-onset diabetes mellitus, LODM).*

Objective*: This study aims to compare the progression patterns of DR in YODM and LODM patients, evaluating differences in severity, risk factors and clinical outcomes.*

Methods and Materials*: This retrospective cohort study analyzed medical records of patients diagnosed with diabetes for at least five years at Vitreo Retina Department of National Institute of Ophthalmology & Hospital, Dhaka in 2017 & 2018. Participants were categorized into two groups: YODM: Diagnosed before the age of 40 years & LODM: Diagnosed at or after the age of 40 years. Demographic and clinical data, including age at diagnosis, diabetes duration, HbA1c levels, blood pressure, lipid profile and treatment history, were collected. Comparisons between YODM and LODM groups were performed using chi-square tests for categorical variables and t-tests for continuous variables.*

Results*: 45.0% of YODM patients had progressed to PDR compared to 27.0% of LODM patients ($p<0.001$). DME was observed in 38.0% of YODM cases versus 22.0% in LODM cases ($p=0.002$). YODM patients exhibited higher mean HbA1c levels (8.9±1.3%) than LODM patients (7.8±1.1%, $p=0.015$). Poor glycemic control was strongly correlated with DR severity in both groups. LODM patients had a higher prevalence of hypertension (68.0% vs. 45.0%, $p<0.001$) and dyslipidemia (52.0% vs. 40.0%, $p=0.012$), which were associated with DR progression. However, despite more comorbidities, DR severity was lower in LODM, possibly due to shorter disease duration. Intravitreal anti-VEGF injections were required in 35.0% of YODM patients versus 20.0% of LODM patients. Laser photocoagulation was more frequentlyneeded in YODM (28.0%) than in LODM (15.0%).*

Conclusions*: This study highlights that DR progresses more aggressively in YODM patients despite similar diabetes duration when compared to LODM patients. The increased risk appears to be driven primarily by prolonged hyperglycemic exposure and poorer metabolic control. In contrast, LODM patients, despite a higher burden of systemic comorbidities, exhibit slower DR progression.*

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Keywords: *Diabetic Retinopathy, Young-Onset Diabetes, Late-Onset Diabetes, Disease Progression, Vision Loss, Hyperglycemia*

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by prolonged hyper-glycemia, leading to micro-vascular and macro-vascular complications. One of the most significant microvascular complications is diabetic retinopathy (DR), which affects nearly one-third of all individuals with diabetes and remains a leading cause of vision loss worldwide[1,2]. The progression of DR is influenced by several factors, including glycemic control, blood pressure, lipid levels and the duration of diabetes[3,4]. The age at onset of diabetes plays a crucial role in determining the disease burden and the severity of its complications Patients diagnosed at a younger age (young-onset diabetes mellitus, YODM) experiencea longer cumulative exposure to hyperglycemia,which increasestheir risk for more aggressive micro-vascular damage and early progression to sight-threatening DR[5,6]. Several studies indicatethat YODM patients exhibit higher rates of diabetic

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complications, including nephropathy, neuropathy and retinopathy, due to prolonged disease duration and often poorer glycemic control[7,8]. Conversely, late-onset diabetes mellitus (LODM) is commonly associated with a higher prevalence of systemic comorbidities, such as hypertension, dyslipidemia and cardiovascular disease, which can influence DR progression via mechanisms like endothelial dysfunction and increased vascular permeability[9,10]. While LODM patients generally have a shorter duration of hyperglycemia exposure, the presence of additional risk factors, such as insulin resistance and systemic inflammation, mayaccelerate micro-vasculardamage[11]. Despite the growing body of evidence on DR risk factors, comparative data on the progression of DR in YODM versus LODM remains limited. Understanding how DR progresses in these two distinct patient populations is critical for optimizing screening protocols, tailoring management strategies and preventing vision loss. This study aims to compare the severity and progression of DR in YODM and LODM patients, focusing on the impact of glycemic control, comorbidities and therapeutic interventions to guide evidence-based clinical practice.

Methods

This retrospective cohort study analyzed medical records of 2017 & 2018 patients diagnosed with diabetes for at least five years at Vitreo Retina Department of National Institute of Ophthalmology & Hospital, Dhaka. Participants were categorized into two groups:

•YODM: Diagnosed before the age of 40 years.

•LODM: Diagnosed at or after the age of 40 years.

Inclusion criteria included confirmed diagnosis of DR through fundus photography and optical coherence tomography (OCT). Patients with secondary diabetes or other retinal diseases were excluded. Demographic and clinical data, including age at diagnosis, diabetes duration, HbA1c levels, blood pressure, lipid profile and treatment history, were collected. DR severity was classified using the International Clinical Diabetic Retinopathy Severity Scale into mild non-proliferative DR (NPDR), moderate NPDR, severe NPDR and proliferative DR (PDR). Diabetic macular edema (DME) presence was also assessed. Comparisons between YODM and LODM groups were performed using chi-square tests for categorical variables and t-tests for continuous variables. Multivariate logistic regression was conducted to identify independent risk factors for DR progression.

Results

A total of 400 patients (200 YODM, 200 LODM) were included. The mean age at diabetes diagnosis was 32.5±4.8 years in YODM and 55.7±6.2 years in LODM. The mean duration of diabetes at study entry was similar between groups (15.2±3.9 years for YODM vs.14.8±4.1 years for LODM, p=0.

Table I: Baseline Characteristics

Characteristic	YODM (n=200)	LODM (n=200)	p value
Mean age at diagnosis (years)	32.5±4.8	55.7±6.2	
Mean diabetes duration (years)	15.2±3.9	14.8±4.1	0.42

Table II: Diabetic Retinopathy Severity and Progression

Severity/progession	YODM (%)	LODM (%)	p value
Progression to PDR	45.0	27.0	<0.001
Presence of DME	38.0	22.0	0.002

Table III: Association with Glycemic Control

Glycemin control factor	YODM	LODM	p value
Mean HbA1c (%)	8.9±1.3	7.8±1.1	0.015
Correlation with DR severity	Strong	Strong	-

Table IV: Impact of Comorbidities

Comorbidity	YODM (%)	LODM (%)	p value
Hypertension	45.0	68.0	<0.001
Dyslipidemia	40.0	52.0	0.012

Table V: Therapeutic Interventions

Intervention	YODM (%)	LODM (%)
Intravitreal anti-VEGF	35.0	20.0
Laser photocoagulation	28.0	15.0

Faster Progression in YODM:
•45% of YODM patients had progressed to PDR compared to 27% of LODM patients (p<0.001).
•DME was observed in 38% of YODM cases versus 22% in LODM cases (p=0.002).
Association with Glycemic Control:
•YODM patients exhibited higher mean HbA1c levels (8.9±1.3%) than LODM patients (7.8±1.1%, p=0.015).
•Poor glycemic control was strongly correlated with DR severity in both groups.
Impact of Comorbidities:
•LODM patients had a higher prevalence of hypertension (68.0% vs. 45.0%, p<0.001) and
•dyslipidemia (52.0% vs. 40.0%, p=0.012), which were associated with DR progression.
•However, despite more comorbidities, DR severity was lower in LODM, possibly due to shorter disease duration.
Therapeutic Interventions:
•Intravitreal anti-VEGF injections were required in 35% of YODM patients versus 20% of LODM patients.
•Laser photocoagulation was more frequently needed in YODM (28%) than in LODM (15%).

Discussion

Our study highlights significant differences in the progression of diabetic retinopathy (DR) between young-onset diabetes mellitus (YODM) and late-onset diabetes mellitus (LODM) patients. Despite a similar duration of diabetes at study entry, YODM patientsexhibited a more rapid and severe progression of DR, with a higher prevalence of proliferative diabetic retinopathy (PDR) and diabetic acular edema (DME). Several key factors, including glycemic control, comorbidities and therapeutic interventions, werefound to influence these outcomes. Our results demonstrate that 45.0% of YODM patients progressed to PDR, compared to only 27.0% of LODM patients (p<0.001). Similarly, DME was more common in YODM (38.0% vs. 22.0%, p=0.002). These findings are consistent with previous studies indicating that patients diagnosed with diabetes at a younger age tend to experience longer cumulative exposure to hyperglycemia, which accelerates microvascular complications[5,11]. The prolonged exposure to elevated glucose levels may contribute to increased oxidative stress, inflammation and retinal microvascular damage6. A key finding in our study was that YODM patients had higher mean HbA1c levels (8.9±1.3%) compared to LODM patients (7.8±1.1%, p=0.015). Poor glycemic control was strongly correlated with DR severity in both groups. This is in line with findings from the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS), both of which demonstrated that higher HbA1c levels are associated with increased DR progression isk[6,9]. Intensive glycemic control has been shown to significantly reduce the progression of DR by limiting endothelial dysfunction and reducing retinal vascular permeability[3]. Interestingly, while LODM patients exhibited a higher prevalence of hypertension (68.0% vs. 45.0%, p<0.001) and dyslipidemia (52.0% vs. 40.0%, p=0.012), DR severity was lower in this group. This may be due to the shorter duration of hyperglycemia exposure before DR onset. Studies suggest that hypertension and dyslipidemia contribute to DR progression by impairing retinal autoregulation and promoting vascular leakage[10]. However, in LODM, the relatively shorter disease duration may offset these risk factors, leading to less aggressive DR progression compared to YODM. This study also found that YODM patients required more aggressive treatment strategies, with 35.0% receivingintravitreal anti-VEGF therapy compared to 20.0% of LODM patients and 28.0% requiring laser photocoagulation compared to 15.0% in LODM. This aligns with previous studies that report increased treatment demand in younger patients due to the more aggressive disease course[12]. The need for earlier and more frequent interventions in YODM highlights the importance of strict metabolic control and regular ophthalmologic monitoring. Early and More Frequent Screening for YODM Patients: Given their increased risk of rapid progression to PDR and DME, YODM patients should undergo more intensive ophthalmologic surveillance. Current guidelines recommend annual retinal examinations, but our findings suggest that high-risk YODM individuals may benefit frombiannual screening[2].

1.Glycemic Control Strategies:
Since hyperglycemia is a major driver of DR progression, personalized HbA1c targets should be set for YODM patients to mitigate long-term complications.
2.Comorbidity Management in LODM:
Although DR progression is slower in LODM, hypertension and dyslipidemia remain significant contributors. Strict blood pressure and lipid control are essential to prevent future complications.

Conclusion

This study highlights that DR progresses more aggressively in YODM patients despite similar diabetes duration when compared to LODM patients. The increased risk appears to be driven primarily by prolonged hyperglycemic exposure and poorer metabolic control. In contrast, LODM patients, despite a higher burden of systemic comorbidities, exhibit slower DR progression, likely due to their shorter cumulative exposure to hyperglycemia. These findings emphasize the need for early screening, stricter metabolic control and tailored treatment strategies based on age at diabetes onset.

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Original Article

Diagnostic Role of Ultrasonography in Severe Acute Appendicitis and Correlation with Age

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Abstract

Background*: The appendix is a narrow, tube-like organ extending from the caecal section of the colon, exhibiting considerable anatomical variation. It contributes to intestinal flora balance, immunological function and the connection between the brain and the digestive system. Appendicitis, inflammation of the appendix, causespain usually starting around the naveland moving to the lower abdomen, worsening withinflammation sometimes develops severe acute condition. The incidence is about 233 per 100,000 annually, peaking in late teens. Acute appendicitis often necessitates emergency surgery, with appendectomy being the preferred treatment. Diagnosis can be challenging without classic symptoms. Diagnosis is supported by ultrasound, complete blood count, CRP testing and computed tomography, with ultrasound preferred due to its safety.*

Objective*: This research aims to analyze the utility of sonography in identifying severe acute appendicitis and to examine its relationship with patient age.*

Methods and Materials*: A cross-sectional study evaluated the role of ultrasonography in detecting acute and severe appendicitis and its correlations with patient age at Community Medical College and Hospital, Mymensingh, Bangladesh, over a one-year period (September 2023 to August 2024). It involved 150 patients, with 113 diagnosed with severe acute appendicitis. Diagnosis relied on clinical assessments and pelvic ultrasonography using high-resolution probes. Histological examination confirmed appendicitisin 100 of 113 cases. Ethical approval and informed consent were obtained. Inclusion criteria included patients below 35 years. Exclusion criteria covered moribund, complicated, unwilling, non-consenting and pregnant patients. Statistical analysis was conducted utilizing SPSS software, version 26, applying descriptive statistical methods.*

Results*: This study involved 150 patients with appendicitis, of whom 113 had acute appendicitis, confirmed in 100 cases by histopathology. The 16-25 age group had the highest incidence of severe acute appendicitis (44% of cases). The majority of patients were male(60%). Ultrasound (USG) showed the appendix in 95% of cases, with a target sign in 95% and 100% had sonographic McBurney's tenderness. USG accuracy included a sensitivity of 94.24% and a specificity of 91.7%. Among 14 USG-negative cases, five were histopathology-positive and nine were negative. Overall, USG demonstrated a diagnostic accuracy of 92.1%.*

Conclusions*: The study confirms ultrasonography (USG) as an effective, non-invasive and cost-efficient tool for diagnosing acute appendicitis. USG aligns well with histopathological findings, showing high sensitivity and specificity. It helps reduce unnecessary surgeries and aids in timely decision-making. Younger males are more frequently affected by appendicitis.*

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Keywords: *Diagnostic, Ultrasonography, Severe Acute Appendicitis, Correlation*

Introduction

The appendix is a slender tube originating from the cecum of the colon[1]. Adult appendices are pencil-shaped structures[2]. The appendix extends from the inner rear wall of the cecum and its location can vary[3]. This organ plays a key part in managing the intestinal microbiome. It is crucial for supporting immune function and the neurological connection with the gu[4]. Appendicitis refers to the inflammatory condition of this organ[5]. Pain in the lower right abdomen is most frequently linked to appendicitis. Typically, discomfort begins near the belly button before migrating to the lower abdomen. Symptoms commonly intensify as the inflammation progresses[6]. The incidence of rate of

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appendicitis at 1,349.8 cases per 100,000 population[7]. The incidence of appendicitis gradually rises from birth, peaks in the late teens and declines in the geriatric years[8]. Severe acute appendicitis represents grossly increased wall thickness and irregularity, luminal fecoliths associated with dense collection, likely pus beside the appendix. Appendicitis most frequently occurs in younger individuals. With the current rise in life expectancy, its incidence seems to be increasing within this population[8]. Acute inflammation of the appendix represents the predominant reason for urgent surgical intervention within the abdomen. It ranks among the primary origins of an acute surgical abdomen[9]. Surgical removal of the appendixremains the standard therapy for both acute appendicitis (AA) and its severe form (SAA), carrying a complication rate of 3.1%. When associated with issues such as perforation, this rate can escalate significantly, potentially reaching 47.2%. These adverseoutcomes often result from delayed medical consultation and treatment initiation, alongside individual patient characteristics. AA and SAA pose significant hazards because of their potentially fatal consequences. Therefore, meticulous evaluation is essential in surgical practice to reduce avoidable complications from appendicitis[10]. Typical clinical features are evident in merely 60.0-70.0% of cases[11]. Diagnosis can be challenging when the classic symptom pattern- pain beginning near the navel and shifting to the lower right abdomen- is absent. Conditions related to gynecology can further complicate the diagnostic process[9]. Diagnostic accuracy is enhanced by specific tests, including a complete blood count, CRP level, abdominal ultrasound and helical CT scanning[12]. Ultrasonography has demonstrated high diagnostic accuracy not only for acute appendicitis but also for other pathologies causing right lower quadrant pain[13] .The clear advantages of ultrasound include the absence of ionizing radiation, its non-invasive nature, minimal patient discomfort, ease of acquisition, portability, repeatability and lack of required special preparation. Consequently, in numerous medical facilities, ultrasound has become the preferred initial imaging modality for evaluating suspected acute appendicitis with unclear clinical signs, especially in children and women of reproductive age[9]. Thus, this research seeks to analyze the diagnostic utility of ultrasound in identifying severe acute appendicitis and to determine its relationshipwith the age of patients.

Methods

We performed a cross-sectional study to evaluate the diagnostic role of ultrasonography in detecting severe acute appendicitis and assess the correlation with patient age, conducted at the Department of Imaging in Community Medical College and ospital, Mymensingh, Bangladesh. The duration of the study was 1 year between September 2023 and August 2024. The study involved 150 patients diagnosed with appendicitis, of which 113 were identified as having severe acute appendicitis. The diagnosis of appendicitis and the decision to operate depends mainly on the clinical picture and investigations, such as pelvic ultrasonography. Ultrasound apparatus with high-resolution probes was used for our study. Standard histological examination was conducted for all patientswith acute appendicitis. Histopathological examination confirmed appendicitis in 100 of those 113 cases. Prior to the study, ethical approval was obtained from the institutional ethical committee. Informed consent was obtained from all the patients involved in the study and confidentiality was maintained.

Inclusion Criteria

•All individuals under the age of 35, regardless of gender, who present with clinical suspicion of appendicitis.

Exclusion Criteria

•Patients in a terminal state are unsuitable for surgical intervention.

•Individuals with appendiceal complications such as an abscess or mass.

•Cases where patients decline additional treatment.

•Patients who do not provide consent.

•Pregnant individuals.

•Standardsforassessmentand operational definitions

The detection of an inflamed appendix or a surrounding abscess on ultrasound was classified as a positive finding for severe acute appendicitis. A non-visualized appendix or one measuring under 5mm was documented as a negative finding. When visible, the inflamed appendix's maximal outer diameter was measured with on-screen calipers. The final diagnosis was confirmed by histopathological analysis. For patients not undergoing surgery, the diagnosis was established by reviewing all clinical assessments and subsequent follow-up data. All relevant data were

organized into suitable tables or charts, with explanatory text to ensure clarity. The statistical evaluation was carried out using SPSS, version 26.0. Results are summarized as means ± standard deviations for parametric data, medians with interquartile ranges for non-parametric data and frequencies (percentages) for categorical variables.

Results

This study includes 150 patients diagnosed with appendicitis. Among all, 113 participants had acute appendicitis. Of the 113 patients, 100 cases were confirmed to have appendicitis through histopathological examination. Table shows that the 16-25 age groupshowed the highest occurrence, with 66 cases (44.0%) of severe acute appendicitis, representing over half of the total cases (52.0%). The 6-15 years group had 29 cases (19.33%) and the 26-35 years group had the fewest, with 18 cases (12.00%). Overall, 113 patients (75.33%) had acute appendicitis, while 37(24.67%) had non-acute appendicitis. The study found a higher prevalence of appendicitis in males (60%) compared to females (40.0%) (Figure 1). Out of 100 histopathologically proven cases of acute appendicitis, USG findings revealed that the appendix was visible in 95.0% of the cases and the target sign was also present in 95.0%. Additionally, sonographic McBurney's tenderness (probe tenderness) was noted in all cases (100.0%).

Table I: Age distribution among patients (n=150)

Age group (years)	Acute appendicitis		Severe acute appendicitis		Total	
	n	%	n	%	n	%
6-15	11	7.33	29	19.33	40	26.67
16-25	12	8.00	66	44.00	78	52.00
26-35	14	9.33	18	12.00	32	21.33
Total	37	24.67	113	75.33	150	100.0

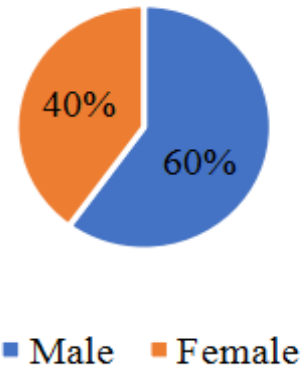


Figure 1: Gender distribution among patients (n=150)

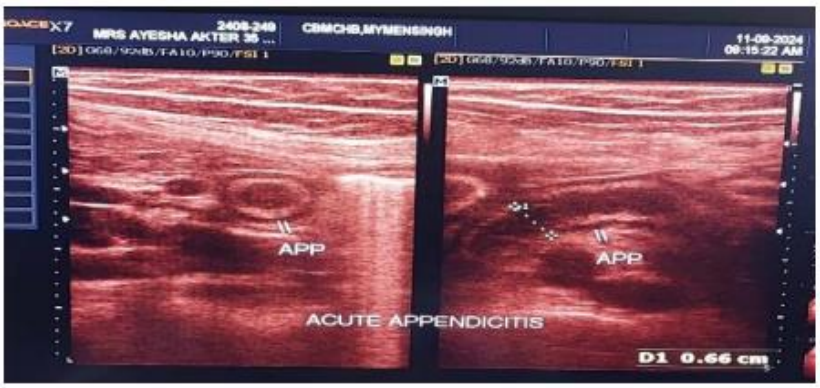
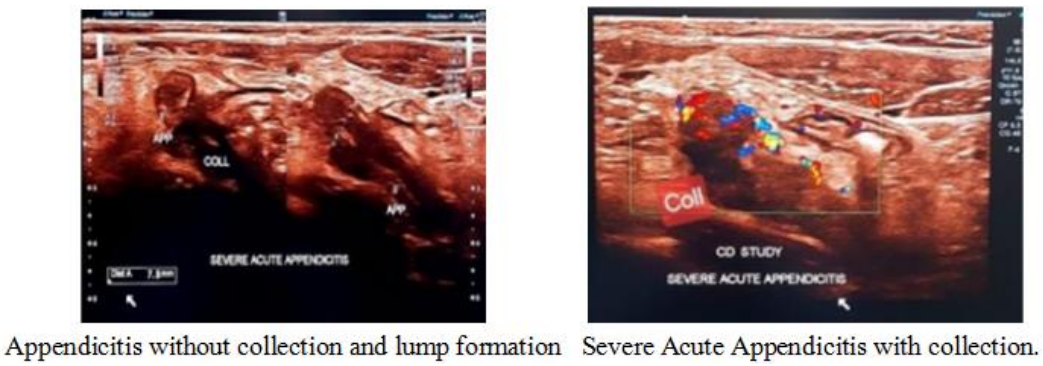


Figure 2: Acute Appendicitis

Additional findings included freefluid in the right iliac fossa in 67.0% of cases, echogenic surrounding mesentery in 81.0%, appendicolith in 7.0% and loss of submucosal integrity in 29.0% of cases (Table II). 79.65% had good results with USG, whereas 20.35% had negative results. In 78.76% patients, HPE confirmed appendicitis, whereas in 1.77% subjects, it was negative. Of the 14 USG-negative patients that underwent surgery, 5(4.42%) were HPE-positive, while 9(7.96%) were HPE-negative. The overall results revealed 88 true positives (77.88%), 18 true negatives (15.93%), 2 false positives (1.77%) and 5 false negatives (4.42%) (Table III). Ultrasound showed a sensitivity of 94.24%, a specificity of 91.7%, a positive predictive value of 97%, a negative predictive value of 82%, and an overall diagnostic accuracy of 92.1% (refer to Table IV).

Table II: USG Findings in Histopathologically Proven Acute Appendicitis (n=100)

USG findings	Number (n)	Percentage (%)
Appendix identification on imaging	95	95.00
Concentric ring appearance in cross-sectional view	95	95.00
Sonographic McBurney's tenderness (probe tenderness)	100	100.00
Appendicolith	07	07.00
Fluid collection in the right lower quadrant	67	67.00
Echogenic surrounding mesentery	81	81.00
Loss of submucosal integrity	29	29.00



Appendicitis without collection and lump formation Severe Acute Appendicitis with collection.

Figure 3-4: Color Doppler shows increased vascularity in the wall of the appendix.

Table III: Corre lation of Ultrasound with Histopathological Examination Report (n=113)

Variables	Frequency (n)	Percentage (%)
USG positive	90	79.65
USG negative	23	20.35
HPE positive	89	78.76
HPE negative	02	01.77
USG negative cases operated	14	12.39
HPE positive	05	4.42
HPE negative	09	7.96
Results		
TRUE positive	88	77.88
TRUE negative	18	15.93
FALSE positive	02	01.77
FALSE negative	05	4.42

Table IV: Diagnostic Role of USG

Evaluation of USG	Value (%)
Sensitivity	94.24
Specificity	91.70
Positive predictive value	97.00
Negative predictive value	82.00
Diagnostic Accuracy	92.10

Discussion

Acute inflammation of the appendix is a leading source of abdominal discomfort necessitating urgent operative care. Identifying its severe form continues to pose a significant diagnostic difficulty, especially when clinical features are atypical and mimicother intra-abdominal conditions[14]. Prompt and precise diagnosis is vital to prevent serious sequelae like perforation, abscess development, or generalized peritoneal inflammation, risks that are heightened in severe cases[15]. Traditionally, diagnosis has relied heavily on clinical evaluation, laboratory findings and imaging techniques. Among these, ultrasonography (USG) has emerged as a non-invasive, readily accessible and cost-effective imaging modality for evaluating patients with suspected appendicitis.This research assesses the utility of ultrasound for diagnosing acute appendicitis and examines how patient age influences its diagnostic accuracy. Evaluating ultrasound's performance across various age brackets aims to guide better imaging approaches for prompt and correct diagnosis, thereby enhancing patient care. Our data indicated the peak incidence of acute appendicitis was within the 16-25-year age range. Age-related prevalence demonstrated that fewer than 19.3% of cases occurred in the 6-15-year group, while over 43.8% affected those above 15 years in Lee et al.'s research[16]. Our results align with the findings of Tariq et al.[6]. Our analysis further showed a higher incidence in males, representing 60.0% of cases versus 40.0% in females. This gender distribution is consistent with the observations of Lamture et al.[17], where males were more frequently affected. Puylaert pioneered the use of graded compression sonography for diagnosing this condition[18]. High-resolution real-time ultrasound is a non-invasive, widely accessible technique that allows direct imaging of an inflamed appendix or adjacent abscess. Comprehensive sonography is also beneficial for patients without clear signs of appendicitis, as it can reveal features suggesting alternative diagnoses- such as mesenteric adenitis, terminal ileitis, or gynecological and urological disorders- as noted by Ooms et al. and Abu- Yousef[13,19]. In this series, ultrasound successfully identified the appendix in 113 out of 150 clinically suspected cases. A separate review documented 70 out of 140 cases of acute appendicitis diagnosed via ultrasound[20]. USG demonstrated its notable effectiveness in diagnosing acute appendicitis. The appendix was visualized in 95.0% of histopathologically confirmed cases and the target sign, a key diagnostic feature, was also present in 95.0%. The presence of sonographic McBurney's tenderness in all cases (100.0%) underscores USG's reliability in detecting acute appendicitis. Additional USG findings, such as free fluid in the right iliac fossa (67.0%), echogenic surrounding mesentery (81.0%), appendicolith (7.0%) and loss of submucosal integrity (29.0%), further support its diagnostic value. Our USG findings from the histological analysis are consistent with the observation of Subash et al.[9]. Among 113 patients, 79.65% of those with positive USG results were confirmed by histopathological examination, while 20.35% of USG-negative cases were also subject to histopathological evaluation. Of the 14 USG-negative patients who underwent surgery, 5(4.42%) had histopathological confirmation of appendicitis and 9 (7.96%) did not. A similar correlation between USG and histopathology was found in another research[9]. This highlights the occasional limitations of USG and the importance of histopathological confirmation in ambiguous cases. The total diagnostic precision of ultrasound for acute appendicitis in our research was 92.1%. Relative to histopathological confirmation, the sensitivity, specificity, positive predictive value and negative predictive value were

94.24%, 91.7%, 97% and 82%, respectively. This indicates that ultra-sonography possesses high diagnostic specificity and sensitivity for this condition. These overall rates align with the findings of Hahn et al. and Tarzan Z et al., where specificity ranged from 90-100% and sensitivity from 70-95%[21,22]. Our sensitivity and specificity figures are also consistent with other investigations[17,23]. These outcomes are comparable to the study by Tauro LF et al., which reported a sensitivity of 91.37%, specificity of 88.09%, positive predictive value of 91.37%, negative predictive value of 88.09% and a diagnostic accuracy of 90.0%[20]. An inflammatory mass following acute appendicitis arises from a contained perforation of the appendix[24].We present a case of acute appendicitis without an associated fluid collection or mass formation (Figure 2). Color Doppler ultrasound findings were deemed positive for appendicitis when enhanced vascularity within the appendiceal wall was observed, as illustrated in Figure 4 of this study.

Limitations of the study

Several limitations exist within this study. The ultrasonography is operator-dependent and variations in skill and experience may affect diagnostic accuracy, which was not standardized across multiple radiologists in this study. Additionally, the exclusion of pregnant patients and moribund individuals reduced the applicability of the results tothese specific groups.

Conclusion

This research underscores the efficacy of ultrasound imaging as a useful diagnostic method for identifying serious acute appendicitis. USG proved to be a reliable, non-invasive and cost-effective method for diagnosing appendicitis, particularly in patients with clear clinical presentations. The study demonstrated a strong correlation between USG findings and histopathological confirmation, supporting its use as an initial imaging modality in cases of suspected acute appendicitis and severe acute appendicitis. Furthermore, USG displayed high sensitivity and specificity, making it useful in reducing unnecessary surgeries and aiding in prompt decision-making for surgical intervention. Age emerged as a significant variable in the incidence of acute appendicitis, with a higher prevalence observed among younger populations, particularly males.

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Original Article

Short-Term Outcomes of Colorectal Cancer Surgeries: Insights from National Institute of Cancer Research and Hospital

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Abstract

Background*: Surgical resection is the principal treatment for colorectal cancer, but it carries significant morbidity and mortality. To see the Short-Term Outcomes of Colorectal cancer surgeries.*
Methods and Materials*: This cross-sectional study was carried out from January 2017 to August 2018.*
Results*: A total fifty-nine (59) patients with colorectal Aden carcinoma with defined inclusion and exclusioncriteria were evaluated. Most of the complications occurred in the male group (33.9%) and in the >50 yearsage group (17%). Forty four percent (44%) of cases were in the pathological stage pT2NOMX (Stage I). Themost common complication (56%) was wound infection. The most frequent complication of surgery was wound infection. Postoperative complications were more in the rectal cancer patients than in coloniccancer cases. Patients operated only laparoscopy had less preoperative blood loss, less postoperative hospital stays and lower complication rates (p<0.05).*
Conclusions*: The laparoscopic approach influences the postoperative outcomes following colorectal cancer surgery. Measures should be taken to reduce the postoperative wound infection.*

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Keywords: *Colorectal cancer, Postoperative complications, Wound infection, Laparoscopic surgery, Short-term outcome*

Introduction

Colorectal cancer (CRC) is the third most common malignancy and the second leading cause of cancer related deaths worldwide[1]. Its prevalence in Bangladesh is 6.5% in males and 2.7% in females[2]. The curative treatment of CRC is surgery. This has been revolutionized by the concept of "Complete mesocolic excision (CME), central vascular ligation (CVL) and D3 lymphadenectomy" i.e., mesocolic excision within an intact mesenteric fascia, ligation of the vascular origin and removal of all lymph nodes along the arterial root, introduced by Hohenberger et al. in 2009[3]. Although the molecular approach to treatment has recently been introduced in the treatment algorithm of colorectal cancer treatment guidelines. National Comprehensive Cancer Network (NCCN) recommends examination of at least 12 lymph nodes to establish the N stage in CRC[4]. Surgical resection of metastatic disease is considered whenever possible. The survival of CRC patients depends on multiple factors, including the stage of disease at diagnosis, patient characteristics, tumor biology and treatment differences. Short term Short term outcome of surgery for CRC is important in the sense that it reflects not only the immediate effect of surgery but also affects the long-term outcomes[5]. Patients who experience complications in the early postoperative period demonstrate long termfunctional results, increased local recurrence rates and reduced 5-year cancer survival 5 and the complication rates are more in the advanced stage of the tumour[6]. Surgical institutions often use operative mortality, complications, length of stay, readmission rate, patients' satisfaction, functional health status and other measures of health-related quality of life.

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Measurement and comparison of postoperative outcomes may derive improvement in perioperative care and may help surgeons to improve their practice. Several studies measured short term outcomes in terms of postoperative complications, duration of operation, intraoperative blood loss, postoperative pain, recovery of intestinal function, length of hospital stay, mortality andqualityoflife. Oncological outcomes described inliteratures as short-term outcomes are extent of of resection, number of lymph nodes (LNs) harvested, local recurrence, survival, etc[7,8].

Methods

This cross-sectional study was carried out from January 2017 to August 2018 in the Department of Surgical Oncology, NICRH, Dhaka, Bangladesh. A total fifty-nine postoperative patients with histologically proven primary colorectal adenocarcinoma comprising new cases ofcolon cancers and rectal cancer (including cases who received total neoadjuvant therapy, TNT) were included. Patients with residual cancer, recurrent CRC, who underwent palliative surgery were excluded from the study. CRCs were grouped into colon and rectal cancers. All patients were followed up in the postoperative period during hospital stay and inquired about any morbidity and mortality also recorded within 30 days following surgery. Patients who had uneventful recovery were compared to those who had eventful recovery. The short-term outcomes of open and laparoscopic colorectal cancer surgeries were measured in terms of complication rate, stage of disease, total time needed for operation, preoperativeblood loss, time to pass first flatus, time to tolerate normal diet, time to become ambulant, mortality, postoperative hospital stay, pathological status, margin status and number of lymph nodes retrieved. Quantitative data were expressed as mean and standard deviation. Qualitative data were expressed as frequency and percentage and comparison was carried out by Chi-square (χ^2) test. A probability value (p) of less than 0.05 was considered to indicate statistical significance.

Results

Among 59 patients, most of the patients (46,78%) were in the 40-60-year age group and complications developed in 52.0% of 46 patients in this age group. Thirty-eight (38, 64.0%) were male, 21(36.0%) were female and complications developed in 21(55.0%) of the male and 11(52.0%) of the female patients. Thirty-six cases (61.0%) were rectal cancer and 23(39.0%) were colonic cancers and most of the complications occurred in rectal cancer group (20, 34.0%). Complications developed in 32(54.0%) out of 59 cases, ranging from Clavein Dindo Grade I surgical complication (e.g., minor wound infection) to Grade V complication (e.g., postoperative death). A total of 17 patients had comorbidities, 6 of them had more than one comorbidity at the same time. In all atients' comorbidities were corrected preoperatively. Fifteen of the patients with comorbidities developed postoperative complications.

Table I: Demographic and clinicopathological characteristics of patients (n=59)

Variables	Frequency (n)	Percentage (%)
Age	05	08.4
<40	46	78.0
40-60	08	13.6
>60	05	08.4
Sex		
Male	38	64.4
Femal	21	35.6
Site of lesion		
Colon	23	39.0
Rectum	36	61.0
Comorbidity		
Absent	42	71.2
Present	17	28.8
Tumour grade		
Well differentiated	09	15.2
Moderately differentiated	33	56.0
Poorly differentiated	17	28.8
Stage of disease		
Stage I	26	44.0
Stage II	09	15.3
Stage III	21	35.6
Stage unknown	03	05.1

In this study, 33(56.0%) patients had moderately differentiated tumour grade. Most of the patients (26 cases, 44.0%) were in the pathological stage I (PT2N0) stage group. Twenty-one patients were in stage III. In three patients, accurate histopathological staging could not be done

(pT2NX, pT3NX) (Table I). Forty-eight (n=48, 80.0%) patients underwent open surgery and 11(20.0%) underwent laparoscopic surgery. Complications developed in 29(49.0%) of open surgeries and 3(5.0%) of laparoscopic surgeries. Two laparoscopic cases were converted to open surgery (conversion rate 3.4%). The most performed operation was open abdominoperineal resection of the rectum (19 cases, 32.0% (Table I). Among 32 patients with complications the most frequent complications was surgical siteinfection, accounting for 59 cases (30.50%). The next common complication was anastomotic leak, 4 cases (6.77%) which were more frequent in open surgeries.

Table II: Frequency of different postoperative complications following Open Surgery (OS) and Laparoscopic Surgery (LS).

Postoperative complications	OS Approach	LS Approach
	n (%)	n (%)
Seroma	02 (03.4)	-
Surgical site infection	14 (24.0)	02 (03.4)
Anstomotic leak	04 (06.8)	-
Intra abdominal abscess	01 (01.7)	-
Ileostomy/colostomy somplicaions	01 (01.7)	-
Burst abdomen	01 (01.7)	-
Perineal wound dehiscence	02 (03.4)	-
Acuterenal failure	01 (01.7)	-
Ureteric injury	-	01 (01.7)
Postoperative bleeding	01 (01.7)	01 (01.7)
Urethral injury	01 (01.7)	-
Total	28 (47.4)	04 (06.7)

Surgical site infection was more common in patients operated by open approach 15(48.0%). Next frequent complication was anastomotic leak (4 cases, 13.0%), which occurred in open surgery group (Table II). The laparoscopy group was found to have less preoperative bleeding, less mortality, less postoperative hospital stays and earlier functional recovery. But time to ambulate following surgery were similar in both groups. More than 12 lymph nodes retrieved in 64.0% of open approaches and 54.0% of laparoscopic approach.

Table III: Short term outcomes in open surgery (OS) and laparoscopic surgery (LS) approaches.

Outcomes	Values	OS	LS	p value
	n (%)	n (%)	n (%)	
Operating time (min)	<180	28 (47.0)	05 (08.0)	>0.05
	>180	20 (34.0)	06 (10.0)	
Peroperative blood loss (ml)	>300	36 (61.0)	10 (17.0)	<0.05
	<300	12 (20.0)	01 (02.0)	
Time of pass flatus	-	4.16±2.07	2.18±0.40	-
Time of tolerate normal diet	-	6.91±3.88	3.36±1.50	-
Time of abulate		4.87±3.58	4.00±1.89	
Postoperative (days)	<10	19 (32.0)	08 (14.0)	<0.05
Hospital stay (days)	>10	29 (49.0)	03 (05.0)	
LN retrieved	<12	17 (29.0)	05 (08.0)	<0.05
	>12	31 (53.0)	06 (10.0)	
Postoperative complications	No	20 (34.0)	07 (12.0)	<0.05
	Yes	28 (47.0)	04 (07.0)	
Mortality	No	46 (78.0)	11 (19.0)	
	Yes	02 (03.0)	-	

Discussion

This study was conducted to evaluate the risk factors for morbidity following surgeries for CRCs. The observed difference in postoperative complication rate in colon cancer group (12 out of 23 cases, 52.0%) and rectal cancer group (20 out of 36 cases, 55.0%) was not statistically significant, (χ^2=0.064, p-value >0.05). Surgical site infection was the most frequent surgical complication, occurred in 30.5% cases. It was present more in rectal cancer surgery than colon cancer operations, (23.7% vs 6.8% cases) (χ^2=3.06, p-value >0.05) (Table II) and in open surgery than laparoscopic surgery (33.3% vs 18.2%) (χ^2=3.63, p-value >0.05). Study by Murray et al. Shows overall surgical site infection rate 12.3%[9]. Rectal resection was associated with overall surgical site infection in comparison with left-or right-sided colonic resections[10]. A study conducted by Rahman et al. found surgical site infection in 52.0% of open surgery cases but no infection in the laparoscopic group. The 30-day mortality rate was 3.4%. In literatures, postoperative mortality of 2.0% to 6.0% following colorectal cancer resection has been described[11]. In a study by Sjo et al. The mortality rate was 3.5% in elective cases and 10.0% in emergency patients and the overall complications rates were 24.0% and 38.0%, respectively[12], whereas Nickelsen et al. recorded 3.9% 30-day mortality[13]. The high incidence of postoperative overall complication rates in this study may be due to the high rate of surgical site infection (56.0% of 32 cases of complication. The higher rate of complications in rectal cancer surgeries may be due to the increased number of patients receiving CCRT before surgery, thereby downstaging the tumourbut increasing the rate of complications. Short-term outcome differences between laparoscopic and open surgery were observed. Regarding operating time, 58.3% (28 cases) of open surgery cases had an operating time less than 180 minutes and six (54.5%) of LS cases took more than 180 minutes. The difference was not statistically significant (χ^2=0.60 p-value >0.05). This may be due to extensive adhesion following CCRT, surgeons' expertness in the early stage of the learning curve of LS, thereby a reason for increased time of operation. These findings are same as the finding of study by Tominaga where the LS group had longer operating time[14]. The observed difference in postoperative complication rate in the OS group and LS group was not statistically significant (χ^2 =1.73, p-value >0.05). A recent study shows that OS group had a higher incidence of overall postoperative complications than the LS group, 25.1% vs. 35.2%[6]; The length of postoperative hospital stays (prolonged when >14 days) significantly differed between 29(76.0%) out of 38 patients of OS and in 3(27.0%) out of 11 cases of LS (χ^2=3.96 p-value <0.05) (Table II), indicating that the LS group had a lower length of postoperative hospital stay. This is in contrast to a study by Ding et al., which shows no differences in terms of length of hospital stay between LS and OS group[15]. Whereas, Chen et al. found the LS group was associated with shorter postoperative hospital stay than the OS group (11.12 days vs. 12.47 days, respectively)[16]. The number of LNs retrieved >12 following OS were observed in 28(62%) cases and 540% of LS group. This observed value was not statistically significant, (χ^2=1.02 p-value >0.05), signifying that LS approach of CRC achieved LNs retrieval similar to that achieved by the OS approach. The mean time to pass flatus after surgery (4.16±2.07 days in the OS group vs. 2.18±0.40 days in the LS group and the mean time to start oral feeding were shorter in the LS group (6.91±3.88 days in open surgery vs. 3.36±1.50 days in LS group). However, the mean time ambulate following surgery was almost same in both groups.

Conclusion

The laparoscopic approach to surgery resulted in less peroperative blood loss and shorter lengths of postoperative hospital stays. The number of lymph nodes retrieved is not affected by the approach of surgery. Preoperative optimization, treatment of comorbidities and adopting a laparoscopic approach may result in improved postoperative outcomes.

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Case Report

Cortical Blindness in Pregnancy: A Case Report

***Siddiqi MH**

Abstract

Cortical blindness originating from the brain's cortex is an uncommon yet severe pregnancy-related complication, often associated with preeclampsia or posterior reversible encephalopathy syndrome (PRES). We present the instance of a first-time pregnant woman, aged 28, who experienced sudden bilateral visual loss postpartum. Neuroimaging revealed findings consistent with PRES. Timely diagnosis and management led to partial visual recovery. This report highlights the critical importance of prompt diagnosis and management for pregnancy-induced cortical blindness.

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Keywords: *Cortical blindness, Pregnancy, Preeclampsia, PRES, Posterior reversible encephalopathy syndrome, Visual loss*

Introduction

Cortical blindness, characterized by bilateral loss of vision with preserved pupillary reflexes and normal ocular examination, results from occipital lobe dysfunction. In pregnancy, it is most commonly linked to hypertensive disorders, including preeclampsia as well as eclampsia. Posterior reversible encephalopathy syndrome (PRES) is a key underlying etiology. We present a case of postpartum cortical blindness due to PRES and discuss its clinical significance.

Case Report

A 28-year-old woman in her first pregnancy (primigravida) and with no prior health issues arrived at the hospital during her 38th week of gestation with headache, nausea, and visual disturbances. She was diagnosed with severe preeclampsia based on elevatedblood pressure (160/110 mmHg), proteinuria, and neurological symptoms. An emergency cesarean delivery was performed using a spinal anesthetic because of concerning fetal heart rate patterns. On the first postpartum day, she reported complete bilateral vision loss. Ophthalmologic evaluation showed intact pupillary reflexes, normal fundus examination, and no signs of retinal or optic nerve pathology. The neurological assessment showed no abnormalities aside from the vision impairment. Brain MRI displayed areas of increased signal in both occipital regions, aligning with a diagnosis of PRES. The patient was managed with antihypertensive therapy, magnesium sulfate, and supportive care. Her vision began improving within48 hours, and partial recovery (6/24) was noted by three weeks postpartum.

Discussion

Cortical blindness in pregnancy is a rare but reversible condition, often associated with hypertensive emergencies. PRES, first described by Hinchey et al., is a neurotoxic syndrome characterized by vasogenic edema predominantly affecting the posterior circulation territories of the brain[1]. The underlying mechanism of PRES is related to impaired endothelial function, which compromises the integrity of the blood-brain barrier and results in brain swelling[2]. Hypertension, renal dysfunction, and endothelial damage are recognized as major contributing factors[3]. In the context of pregnancy, hypertensive disorders such as preeclampsia and eclampsia remain the leading causes of PRES, occurring in up to 90.0% of affected individuals[4]. Neuroimaging, particularly MRI with fluid-attenuated inversion recovery (FLAIR) sequences, is essential for diagnosis. Typical findings include symmetrical hyperintensities in the occipital, parietal, and sometimes frontal lobes[5]. In our case, bilateral occipital lobe involvement confirmed the diagnosis. Although computed tomography (CT) scans can detect some changes, MRI remains the gold standard for early and accurate diagnosis[6].

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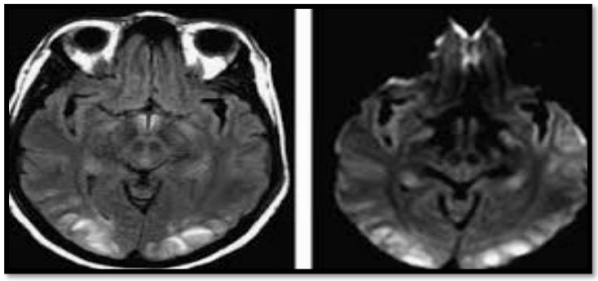
This case underscores the importance of recognizing PRES as a reversible cause of cortical blindness in pregnancy. Obstetricians, neurologists, and ophthalmologists should collaborate to ensure timely diagnosis and appropriate management. Further investigation is necessary to enhance comprehension of therisk factors and extended prognosis for PRES during gestation[6].



Photograph 1: MRI (brain) revealed bilateral hyperintensities (occipital lobes).

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Review Article

About the Pathogenesis of Inflammatory Bowel Disease - A Review

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Abstract

Inflammatory bowel disease (IBD) refers to a persistent inflammatory condition of the gut arising from interactions between the host's physiology and intestinal microbes in those with a genetic predisposition. This category of autoimmune disorders involvesinflammation of the intestinal tract, where the body's immune system targets components of its own digestive system. The development of IBD is multifactorial. Its worldwide prevalence is rising, prompting more individuals to consider dietary choices as both an explanatory factor and a therapeutic approach for their condition. Indeed, many patients are convinced that diet is crucial in triggering and controlling their IBD symptoms. Recent research has significantly advanced our understanding of IBD's underlying mechanisms, resulting in major progress in both its treatment and diagnosis. This review systematically examines the disease's pathogenesis and emphasizes current discoveries concerning host genetics, intestinal flora, the influence of diet and environmental triggers. These areas may provide vital insights for discovering new predictive or prognostic markers and creating innovative treatments.

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Keywords: *Inflammatory bowel disease, Crohn's diesease, Ulcerative colitis, Diet*

Introduction

Inflammatory bowel disease (IBD) is a long-term inflammatory disorder of the digestive system, clinically encompassing Crohn's disease, ulcerative colitis and related diagnoses[1]. Crohn's disease can involve any part of the gastrointestinal tract from the mouth to the anus, while ulcerative colitis is typically confined to the colon and rectum[2,3,4]. Histologically, Crohn's disease exhibits transmural inflammation, whereas ulcerative colitis is limited to the mucosal layer of the gut lining[5]. IBD is a chronic condition often diagnosed in early adulthood, affecting both sexes[6]. Crohn's disease has a slight female predominance, but ulcerative colitis occurs equally in men and women. Geographically, IBD is more common in industrialized nations and regions with colder climates[7]. The occurrence and prevalence of IBD rose significantly during the latter half of the 20th century. In the 21st century, it has become one of the most common gastrointestinal disorders, with a rising incidence in newly industrialized nations[8,9,10]. Crohn's disease prevalence is notably elevated in highly developed countries, with an incidence of approximately 5 per 100,000 individuals and an estimated prevalence of, 30-50 per 100,000 in Western populations[11]. In Canada, for instance, about 129,000 individuals are affected[12]. While diagnosis typically happens in adulthood, pediatric cases are becoming more frequent. Since 1990, incidence rates in Western nations have stabilized or begun to decline, whereas rates in newly industrializing countries across Asia, Africa and South America continue to climb[10]. Although Crohn's disease and ulcerative colitis have overlapping features, differentiating between them can be challenging; they are distinguished by the specific sites and patterns of inflammation within the gut[11]. The exact etiology of IBD is still unclear, but substantial advances have recently been made in understanding its development. Research indicates that its pathogenesis is linked to genetic predisposition,

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gut microbiome composition, additional environmental influences and immune system dysregulation[13,14].

Signs and symptoms of IBD

The intestinal lining inflammation in IBD presents with recurrent symptoms such as abdominal cramping, diarrhoea, bloody feces and loss of weight. This is driven by an infiltration of immune cells like neutrophils and macrophages, which release inflammatory signals, enzymes and reactive molecules that cause tissue damage and ulceration[1,15]. Clinical manifestations often differ based on the specific IBD form[16]. In ulcerative colitis, continuous mucosal inflammation leads to swelling, ulceration, hemorrhage and fluid-electrolyte imbalance. The inflammation typically begins in the rectum and spreads contiguously upward through the colon[7]. Affected individuals commonly report pain in the lower left abdomen alongside diarrhoea, which can lead to weight reduction and rectal bleeding[17,18].

Conversely, Crohn's disease more frequently causes discomfort in the lower right abdomen and visible rectal bleeding is less common than in ulcerative colitis. Afrequent complication is intestinal obstruction from pronounced swelling and bowel wall thickening. Additionally, malabsorption issues in Crohn's disease often result in nutritional deficits or malnutrition[17,18]. Crohn's disease can also lead to strictures, persistent inflammation, or fistula formation. Its hallmark pathological characteristic is

transmural involvement, meaning it affects the entire depth of the intestinal wall[7].

World Gastroenterology Organization (WGO) Diagnostic Criteria for IBD

i. Diarrhea, which can contain blood or mucus, often occurs nocturnally and involuntary stool leakage is frequently reported.

ii. Constipation may be a symptom in ulcerative colitis cases where inflammation is restricted to the rectal area.

iii. Other frequent symptoms include abdominal cramping, a constant feeling of needing to pass stool and intense, sudden urgency.

iv. Pain in the right lower abdomen is typical for Crohn's disease, while left lower quadrant pain is more common in ulcerative colitis.

v. Feelings of nausea and episodes of vomiting are observed more often in Crohn's disease than in ulcerative colitis.

Physical Exam

•Elevated heart rate, restlessness, pyrexia and fluid loss are frequently observed.

•Skin pallor may be apparent, correlating with the presence and degree of anemia.

•Toxic megacolon can manifest with intense pain, fever, abdominal bloating, rigors and profound fatigue. This critical surgical condition must always be considered, as it is life-threatening if not recognized.

•Individuals with Crohn's disease may present with perianal fistulae, abscesses, or rectal prolapse.

•A digital rectal examination commonly reveals hidden blood.

•In pediatric cases, the sole presenting sign may be a failure to achieve expected growth milestones.

Etiology

The etiology of IBD is still not fully understood. Multiple factors have been proposed, but no single cause is consistently identified in every case[7]. IBD is a multifactorial disorder triggered by a combination of genetic predisposition and environmental exposures, which then initiates an abnormal immune reaction and intestinal inflammation[2]. A well-established characteristic of Crohn's disease is its strong association with tobacco use. Conversely, smoking appears to reduce the risk of developing ulcerative colitis[7]. This is an example of a factor that differentially influences disease subtypes, exacerbating Crohn's disease while offering protection against ulcerative colitis[19,20]. Smoking has been demonstrated to alter both cellular and antibody-mediated immune functions and to stimulate mucus production in the colon[19,21]. Research also indicates that smoking disrupts autophagy, a cellular process believed to be particularly relevant in Crohn's disease pathogenesis[22]. The influence of diet continues to be asubject of debate[7]. A 2022 investigation concluded that dietary patterns emphasizing higher consumption of fruits and vegetables, lower intake of processed meats and refined carbohydrates and adequate hydration with water correlated with a decreased likelihood of active IBD symptoms. However, increasing fruit and vegetable intake alone did not lower symptom risk specifically for Crohn's disease[23].

Pathogenesis of IBD

1. Genetic Factors

Research utilizing genome-wide association studies (GWAS), next-generation sequencing and related methods has uncovered more than 240 distinct genetic regions associated with disease risk. Approximately 30 of these loci are common to both Crohn's diseaseand ulcerative colitis[24,25,26].

Examination of these genes and loci reveals that multiple biological pathwayscrucial for intestinal balance are involved, including the integrity of the epithelial barrier, innate immune defense at the mucosa, regulation of immunity, cellular movement, autophagy processes, adaptive immune responses and metabolic pathways linked to maintaining cellular stability[27,28,29,30]. Variations in the CARD15 gene are linked to IBD, but due to its polymorphic nature, predicting the specific segment of the gastrointestinal tract that will be affected is not possible. Genetic factors appear to play a less dominant role in ulcerative colitis compared to their influence in Crohn's disease[7].

2. Gut Microbial Factors

IBD is thought to develop from an aberrant immune reaction by the host to the microbes residing in the gut[31,32,33]. The intestinal microbiome acts as the primary environmental factor influencing IBD. From birth, the human digestive system is inhabited by an immense diversity of microorganisms, which outnumber the body's own cells by roughly tenfold[33,34]. Collectively, these microbes possess a gene repertoire approximately one hundred times larger than the human genome[31,33,34]. Factors such as diet, probiotic and prebiotic intake, antibiotic use, supplemental enzymes, fecal microbiota transplants and other external elements can alter the composition of this gut flora[32] .A balanced gut microbiota is essential for maintaining normal intestinal function, stability and overall health and is implicated in various disease states[33,34,35].

3. Environmental Factors

The significant influence of environmental factors on IBD development is reinforced by recent epidemiological research. The incidence of Crohn's disease has risen notably in industrialized nations over the last half-century and its identification has similarly grown in developing regions as they industrialize[36,37]. Diet is a key environmental element impacting IBD onset[38]. Evidence suggests that consuming fruits and vegetables correlates with a lower risk of Crohn's disease[39]. while diets high in fast food, fats and sugars may worsen its development[38]. High intake of protein, especially from animal sources and sugars may also be linked to an elevated risk of IBD and symptom recurrence[40,41]. Meat contains sulfur amino acids, which gut bacteria ferment to produce hydrogen sulfide. This compound may contribute to ulcerative colitis by interfering with butyrate metabolism in colon cells and weakening the intestinal mucus barrier, thereby increasing permeability to pathogens[42,43]. Research indicates that dietsrich in animal protein can promote pro-inflammatory immune cell responses and worsen colitis in animal models[44]. Dietary fats, particularly polyunsaturated fatty acids (PUFAs), have also been implicated in IBD pathogenesis[45]. Recently, emulsifiers- food additives used to improve texture and shelf life- have gained attention as potential inflammatory agents that may contribute to IBD[46]. They can alter the gut microbiome by reducing its diversity and encouraging pro-inflammatory bacteria. Common sources of emulsifiers include ice cream, plant-based milks, dressings and pasta[47,48,49]. A systematic review by Hou et al. generally found that higher dietary fiber intake was associated with reduced risk of both UC and CD, though only one study reported a statistically significant reduction for CD[50]. Fiber helps maintain gut barrier integrity by supporting the inner mucus layer, which contains antimicrobial compounds and acts as a defense against pathogens[51]. Fiber deprivation shifts the microbiome toward mucus-degrading bacteria and thins the protective mucus layer, bringing bacteria closer to the intestinal epithelium[52,53]. Overall, components like meat, fats, fiber and additives interact with the microbiome to either strengthen or compromise intestinal barrier function, influencing pathogen exposure. Many IBD patients modify their diets to manage symptoms, commonly avoiding spicy foods, dairy, fatty foods and high-fiber vegetables- often cited as "trigger" foods[54,55,56,57,58]. Alcohol is also reported to worsen symptoms but has been less consistently studied[54,56]. A survey of 2329 IBD patients found that those with active disease were more likely to avoid fruits, vegetables, tomatoes, beans and ice cream compared to those in remission[56]. Conversely, yogurt and rice were frequently reported to alleviate symptoms. Importantly, while certain foods may worsen gastrointestinal

symptoms, studies have not proven they increase underlying inflammation. Thus, individualized dietary adjustments can improve comfort even if they don't directly reduce inflammation. Other environmental factors linked to IBD include psychological stress, prior appendectomy and certain medications[59]. For instance, appendectomy is a risk factor for Crohn's disease but appears protective against ulcerative colitis[60]. Although epidemiological studies have identified these associations, understanding the precise mechanisms by which environmental factors influence IBD progression remains a challenge[59].

4.Immunological Abnormalities

The immune system's reaction to gut bacteria is precisely controlled, a process that dictates whether immune tolerance or a protective inflammatory reaction occurs. Disruption of this equilibrium can lead to IBD[61]. The gut-associated immune system plays acentral role in IBD's development. The intestinal lining normally blocks microbes and antigens from entering the bloodstream via tight cellular connections[7]. In IBD, these connections are impaired, either due to an intrinsic barrier defect or as a consequence of intense inflammation. Other defenses include mucus secreted by goblet cells and antimicrobial peptides like α-defensins released by Paneth cells. Excessive inflammation causes ongoing damage to the epithelial layer, increasing exposure to intestinalbacteria and creating a cycle of worsening inflammation[7]. The immune dysfunction in IBD involves multiple components: epithelial injury (including faulty mucus production and impaired healing); inflammation amplified by the gut flora and a heavy infiltration of immune cells- such as T cells, B cells, macrophages, dendritic cells and neutrophils- into the mucosal tissue; and a breakdown in the regulatory mechanisms needed to resolve the inflammatory response[8,62,63].

Diagnosis

Diagnosis typically involves evaluating fecal markers of inflammation, then proceeding to colonoscopy with tissue sampling from affected areas[64]. Confirmation is usually achieved through biopsy during colonoscopic examination. Measuring fecal calprotectinserves as a helpful initial test, as it can indicate the likelihood of IBD due to its high sensitivity, although it is not specific to the disease[65,66].

Histopathology

Histological examination in ulcerative colitis reveals inflammation confined to the mucosal and submucosal layers, featuring crypt abscesses and superficial ulcers. Tissue samples demonstrate infiltration by neutrophils, architectural distortion of the crypts and these characteristic abscesses. Granulomas are not present. The inflammation is continuous and almost always includes the rectum.

The formation of pseudo polyps is another typical finding. In Crohn's disease, inflammation affects the full thickness of the intestinal wall and may include granulomas. The inflammatory pattern is transmural and marked by a lymphocytic infiltrate[7]. Histologically, Crohn's disease shows a widened submucosa, transmural inflammation, fissuring ulcers and granulomas. By contrast, ulcerative colitis involves only the mucosa and submucosa, with cryptitis and crypt abscesses as hallmarks[67,68,69].

Complication

Ulcerative colitis increases the risk for complications outside the intestines, commonly affecting the skin, eyes and joints. These most frequently include inflammatory joint conditions and primary sclerosing cholangitis. Crohn's disease primarily targetsthe ileum and colon but can also involve the esophagus, stomach, or duodenum. Similar to UC, Crohn's is associated with systemic manifestations such as arthritis, mouth ulcers, uveitis, erythema nodosum and ankylosing spondylitis[70,71]. Research indicatesa higher mortality risk for individuals with Crohn's disease. Analyses focusing on intestinal cancers reveal that these patients also have increased comorbidities, including colorectal cancer, cardiovascular disorders and respiratory illnesses[17,18]. In Crohn's disease, kidney disorders and gallstones occur more frequently due to impaired absorption of bile acids and fatty acids. Patients who have had ileal resection while retaining their colon are also at greater risk for developing calcium oxalate kidneystones[7].

Conclusion

IBD is a persistent and potentially severe disorder marked by recurrent gut inflammation. Living with this condition can be difficult, though many affected individuals maintain a largely regular lifestyle. The diagnosis often carries a psychological weightdue to associated stigma, frequently resulting in elevated anxiety, depressive

symptoms and an overall decline in life quality. Significant advancements over recent decades have deepened our insight into IBD's mechanisms, particularly in immunology and have opened new avenues for therapeutic discovery. Nonetheless, unanswered questions remain regarding its origin, disease progression patterns and the specific triggers of inflammation across different patient subsets, necessitating further research. Theinteractions between pro-inflammatory and anti-inflammatory cells and signaling molecules, combined with various genetic predispositions, gut flora compositions and external factors (such as diet, tobacco use and stress), are under ongoing investigation. This work aims to build a complete picture of the disease process. With this growingunderstanding, we may be able to create innovative, tailored therapies for those with IBD.

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