Al-Azhar Med. J . (Medicine). *Vol. 51(3), July, 2022, 1579 – 1590*
DOI: 10.21608/amj.2022.240690
https://amj.journals.ekb.eg/article_240690.html

CAN CHRONIC GASTRITIS CAUSE AN INCREASE IN FECAL CALPROTECTIN CONCENTRATIONS?

By

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ABSTRACT

Background: Chronic gastritis represents a common and heterogeneous inflammatory process. It can be morphologically characterized by a variable inflammatory infiltrate in the lamina propria, within the epithelium, and within the foveolar lumen. Calprotectin is a calcium and zinc binding protein, mainly contained in neutrophils where it accounts for more than 60% of cytosolic proteins. It has well-known antimicrobial activity, both bacterial and fungicidal.

Objective: To evaluate fecal calprotectin concentrations in subjects with chronic gastritis that proved by endoscopic features and comparing them with fecal calprotectin concentrations in normal subjects.

Patients and methods: This study was carried out on 100 subjects divided into two equal groups: Cases with chronic gastritis, and control group of normal subjects. Their ages ranged between 1 day to 18 years who were referred to the Endoscopy Center of Al-Hussien Hospital for upper gastrointestinal endoscopy, between August 2018 and May 2020.

Results: The results showed that the demographic data which included age and sex in cases and control were matched without significant difference. The results of calprotectin showed a significant increase in the level of calprotectin in cases more than control. On the other hand, it was found that 5 cases were positive in patients group, while no one was positive in control group. At cut off value 50.0, it was found that the sensitivity of diagnosis chronic gastritis was 64.5%, specificity was 61.0 and accuracy was 63.0%.

Conclusion: Fecal calprotectin has the potential to facilitate the diagnostic workup in children with chronic gastritis, and has also the potential for monitoring disease activity in pediatric.

Keywords: Chronic Gastritis, Fecal Calprotectin Concentrations.

INTRODUCTION

Calprotectin is a calcium-binding protein that makes up around 60% of the total cytosolic protein content of neutrophils and mononuclear cells. It is a heterocomplex protein comprising two heavy (L1H) chains and one light (L1L) chain which are noncovalently linked, and is an important regulatory protein in inflammatory reactions *(Kallberg et al., 2012)*.

Studies in pediatric and adult cohorts have demonstrated a correlation between fecal calprotectin concentrations and the severity of mucosal inflammation *(Pathirana et al., 2018)*.

Fecal calprotectin has also been shown to correlate with changes in C-reactive protein and erythrocyte sedimentation rate

in active inflammation. The stability of calprotectin at room temperature adds to its value as a practical and convenient marker in intestinal inflammation, and for the differential diagnosis of inflammatory bowel diseases (IBD) and irritable bowel syndrome (IBS) *(Wang et al., 2013)*.

Calprotectin extracted from stool can be detected easily using standard enzyme linked immunosorbent assays (ELISA). Numerous studies have shown that fecal calprotectin concentrations demonstrate a good correlation with intestinal inflammation *(Bello et al., 2017* and *Xie et al., 2017)*.

Chronic gastritis represents a common and heterogeneous inflammatory process. It can be morphologically characterized by a variable inflammatory infiltrate in the lamina propria, within the epithelium and within the foveolar lumen. According to the updated Sydney System, the presence of a neutrophil infiltrate characterizes the "activity" of gastritis *(Hamid et al., 2017)*.

Calprotectin has been considered as a marker for diagnosis, treatment and monitoring of inflammatory bowel disease (IBD) and gastrointestinal (GI) malignancies as well. The level of fecal calprotectin increases in these conditions and its amount varies depending on the type and severity of the disease. Various studies in IBD have shown an increase in stool calprotectin. The measurement of fecal calprotectin is a completely noninvasive method. It could be used as an alternative diagnostic method *(Grossman and Baldassano, 2016)*.

The aim of our study was to evaluate fecal calprotectin concentrations in subjects with chronic gastritis that proved by endoscopic features, and comparing them with fecal calprotectin concentrations in normal subjects.

PATIENTS AND METHODS

This study was carried out on 100 subjects divided into two equal groups, Cases with chronic gastritis which included 50 cases, and control group included 50 normal subjects. Their ages ranged between 1 day to 18 years who were referred to the Endoscopy Center of Al-Hussien Hospital for upper gastrointestinal endoscopy, during the period from August 2018 and May 2020.

The extraction of at least 6 biopsy samples (2 from the antrum, 2 from the corpus and 2 from the incisura angularis) had been undertaken to correctly characterize an eventual gastritis process. However, when esophageal lesions, gastric ulcers, gastric polyps or duodenal lesions were found during the endoscopy. The necessary biopsy specimens were taken, and these subjects were not included in the study. In addition, subjects with IBDs or family history of IBDs, colorectal cancer, chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs), history of gastric resection, coexisting and severe cardiopulmonary, hepatic, renal, neurologic, psychiatric, endocrine and rheumatologic diseases, malignancy, other intestinal disorders characterized by increased mucosal permeability and inflammatory changes, were not considered for the study.

All the eligible subjects were asked to provide a stool sample for measurement of calprotectin levels, within 2 days of endoscopic examination, before starting specific therapy. Stools were also examined to exclude infectious intestinal

diseases. All subjects were asked if they were taking proton pump inhibitor (PPI) therapy for at least 1 month before the endoscopy.

Histological evaluation:

The biopsy samples were fixed in 4% buffered formalin, processed in the usual manner, and paraffin embedded. The sections were stained with hematoxylin and eosin for histological evaluation; Giemsa stain was also used to evaluate the presence of Helicobacter pylori (H. pylori). The sections were evaluated by an expert gastrointestinal pathologist in Dr. Khaled Refaat Pathology Lab., Al-Hussein University Hospital.

H. pylori status was evaluated as present/absent in all the examined biopsy samples.

Fecal calprotectin measurement:

Each subject was instructed to collect and return a single stool sample within 48 h of defecation. Upon receipt, the stools were frozen and stored at -20°C for subsequent biomarker determination.

The stool samples were prepared and analyzed according to the manufacturer's instructions (Calprest; EurospitalSpA, Trieste, Italy). A portion of each sample (40-120 mg) was measured and an extraction buffer containing citrate and urea was added in a weight per volume ratio of 1:50. The samples were mixed for 30 second by a vortex method and homogenized for 25 min. One milliliter of the homogenate was transferred to a tube and centrifuged for 20 min. Finally, the supernatant was collected and frozen at -20°C. In most cases, time from sampling to preparation and freezing was estimated to be 1-3 day, except for a few samples that took 4-6 day before handling. The supernatants were thawed and analyzed later with Calprest, a quantitative calprotectin ELISA, for determination of calprotectin in stools. The within-assay coefficient of variation was 1.5%. Calprotectin was expressed as μg/g of feces.

Statistical analysis:

Data were fed to the computer using IBM SPSS software package version 24.0. Qualitative data were described using number and percent. Comparison between different groups regarding categorical variables was tested using Chi-square test or Fisher's exact test. Quantitative data were described using range mean and standard deviation independent t-test or Mann-Whitney U test. Significance test results were quoted as two-tailed probabilities. Significance of the obtained results was judged at the 5% level. The ROC curve was used to measure the diagnostic ability of a biomarker.

RESULTS

This study was carried out on 100 subjects divided into two groups, cases with chronic gastritis which include 50 cases, and control group include 50 normal subjects. There was no statistical significant difference between the two studied groups regarding age and sex (P > 0.05) **(Table 1)**.

Table (1): Comparison between the two studied groups regarding age and sex

Groups / Parameters	Cases "n=50"		Control "n=50"		P value
	No.	%	No.	%	
Age:					
<1 year	2	4.0	1	2.0	0.736
1-4 years	14	28.0	12	24.0	
>4 years	34	68.0	37	74.0	
Range	0.83-18.0		0.42-18.0		0.183
Mean+ SD	7.34+4.71		6.16+4.13		
Sex:					
Male	16	32.0	28	56.0	0.016
Female	34	68.0	22	44.0	

There was a statistical significant difference between the two studied groups regarding calprotectin category (P < 0.05). There was no statistical significant difference between the two studied groups regarding age group less than 1 year (P > 0.05), while there was a statistical significant difference regarding age from 1-4 years and more than 4 years (P < 0.05). There was a statistical significant difference between the two studied groups regarding males (P < 0.05), while there was no statistical significant difference regarding females (P > 0.05) **(Table 2)**.

Table (2): Comparison between the two studied groups regarding calprotectin category, Age group and Sex group

Parameters / Groups			Cases "n=50"		Control "n=50"		P value
			No.	%	No.	%	
Calprotectin category	**Negative**		45	90.0	50	100.0	0.056
	Positive		5	10.0	0	0	
	Range		7.0-344.0		7.00-76.0		0.005
	Mean+SD		55.90+56.78		31.90+16.02		
Age group	< 1 year	Negative	2	4.0	1	2.0	0.001
		Positive	0	0.0	0	0.0	
	1-4 years	Negative	13	26.0	12	24.0	0.001
		Positive	1	2.0	0	0.0	
	> 4 years	Negative	30	60.0	37	74.0	0.048
		Positive	4	8.0	0	0.0	
Sex group	Male	Negative	12	24.0	28	56.0	0.013
		Positive	4	8.0	0	0.0	
	Female	Negative	33	66.0	22	44.0	0.001
		Positive	1	2.0	0	0.0	

At cut off value 50.0 it was found that the sensitivity of diagnosis chronic gastritis was 64.5%, specificity was 61.0 and accuracy was 63.0% **(Table 3** and **Figure 1)**.

Table (3): Sensitivity, specificity and accuracy of calprotectin in prediction chronic gastritis

Calprotectin	Area under the curve	Cut off value	P value	Asymptotic 95% C.I.	
				Lower Bound	Upper Bound
	.625	50.0	0.031	.515	.735
Sensitivity	64.5				
Specificity	61.0				
Accuracy	63.0				

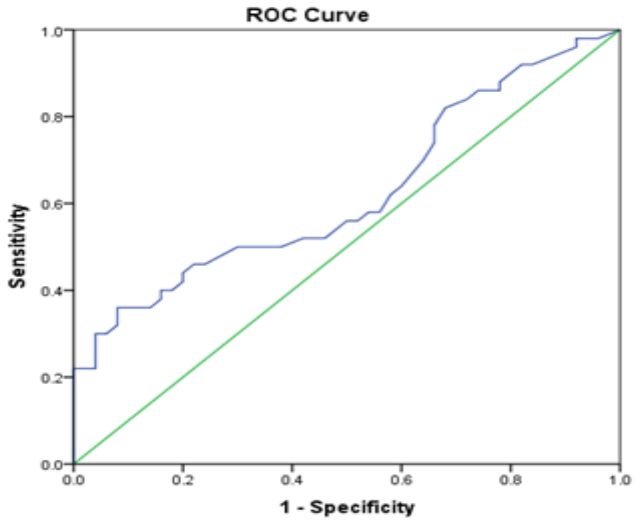

Figure (1): ROC curve to predict the sensitivity specificity and accuracy of calprotectin in prediction chronic gastritis

There was no statistical significant relation between biopsy findings and calprotectin (P > 0.05). There was no statistical relation between endoscope and calprotectin (P > 0.05) **(Table 4)**.

Table (4): Relation between biopsy findings and endoscope and calprotectin

Calprotectin / Parameters	Negative		Positive		Total	
	No	%	No	%	No	%
Biopsy:						
H. pylori gastritis	27	60.0%	5	100.0%	32	64.0%
Mild superficial gastritis+non-specific duodenitis	18	40.0%	0	0.0%	18	36.0%
P	0.095					
Endoscope:						
Pan gastritis	22	48.9	4	80.0	26	52.0
Low esophagitis	12	26.7	1	20.0	13	26.0
Pan gastritis + lower esophagitis	11	24.4	0	0.0	11	22.0
P	0.344					

DISCUSSION

In our study, the results showed that the demographic data which included age and sex in cases and control was matched without significant difference. These results were important to eliminate the effect of demographic data on the net results. There was a significant increase in the level of calprotectin in cases more than control. On the other hand, it was found that 5 cases were positive in patients group while no one was positive in control group. At cut off value 50.0, it was found that the sensitivity of diagnosis chronic gastritis was 64.5%, specificity was 61.0 and accuracy was 63.0%.

In agreement with our study, *Paek et al. (2020)* stated that the fecal calprotectin as a marker of gastrointestinal involvement in pediatric. The results showed that the clinical manifestations of patients with fecal calprotectin levels over and under 50 mg/kg were compared. Patients with fecal calprotectin levels of > 50 mg/kg showed more frequent gastrointestinal involvement, longer hospitalization duration, and longer gastrointestinal symptom duration.

In partial agreement with our results, *Wang et al. (2013)* found that calprotectin significantly elevated in patients with ulcerative colitis or Crohn's disease, compared with controls or patients with colorectal polyps or irritable bowel syndrome. In addition, calprotectin significantly elevated in patients with esophageal polyps/ gastric neoplasm compared with controls or patients with chronic gastritis/stomach ulcer/duodenal ulcer/acute pancreatitis. There were no other statistically significant between-group differences.

The fecal calprotectin concentration significantly differentiated between IBD and non-IBD. A cut-off value of 45.40 mg/g resulted in a sensitivity of 0.944 and specificity of 0.643; a cut-off of 110.65 mg/g resulted in a sensitivity of 0.944 and specificity of 0.429.

Studies have found significantly higher fecal calprotectin concentrations in some gastrointestinal disorders (including oesophageal/ gastric carcinoma, Crohn's disease, ulcerative colitis and colorectal carcinoma) than in others (Barrett's esophagus, gastric ulcer, gastritis/duodenitis, colorectal polyps and adenoma) *(Sidler et al., 2011)*.

In addition, ROC curve analysis confirmed that the fecal calprotectin concentration effectively differentiated between IBD and IBS, with increased calprotectin being associated with a heightened risk of IBD. The elevated levels of calprotectin seen in patients with esophageal polyps and gastric neoplasms in the present study (in contrast to those with chronic gastritis, stomach ulcer, duodenal ulcer or acute pancreatitis) may be explained by the synthesis of calprotectin by squamous epithelial cells, granulocytes and macrophages, or by the large wound surface present on these tissues *(Chang et al., 2014)*.

There are few studies evaluating the relationship between upper GI disorders and fecal calprotectin. Many researchers have investigated the association between inflammatory bowel disease (IBD) with the marker, the severity of intestinal involvement and response to treatment. The results of the present study showed that there was no correlation between the calprotectin level and abnormalities of the

esophagus *(Walsham and Sherwood, 2016)*.

The calprotectin level did not increase by inflammation, erosions of the esophagus, displacement of Z line, hiatus hernia and relaxation of the lower sphincter of the esophagus. There were no similar studies that can be compared with our current findings. With confirmation of these findings by further research, it can be concluded that calprotectin was not a suitable marker for the evaluation of esophageal pathology. It was also found that inflammation of the stomach or gastritis (based on pathologic findings) and nodularity of antrum can increase the level of fecal calprotectin *(Ataee et al., 2017)*.

However, erythema of the stomach in the macroscopic view was not associated with a marked increase in fecal calprotectin. There was a significant relationship between the fecal calprotectin levels and grade and severity of gastritis *(Manz et al., 2012)*.

Pathological changes in the duodenal bulb, such as erythema, ulceration and nodularity do not increase the level of fecal calprotectin. In our study, there was a significant difference in the concentration of fecal calprotectin between chronic active gastritis and chronic nonactive gastritis. It can also be seen a significant difference in chronic gastritis with different severity i.e. mild, moderate, severe *(Montalto et al., 2010)*.

Manz et al. (2012) showed that the level of stool calprotectin was lower in normal patients than in the ones with erosive gastritis.

Another study conducted by *Montalto et al. (2010)* measured the calprotectin level in patients with gastritis (based on histopathological findings). Finally, non-significant difference was found between the groups. The number of patients in our study and the two other studies of *Manz et al. (2012)* and *Montalto et al. (2010)* were not equal, and this issue influenced the results of the investigation. H. pylori infection led to significantly increased levels of calprotectin in stool.

However, *Montalto et al. (2010)* did not find a significant correlation between them. This could be due to the larger number of patients with H. pylori infection in our study. The results of our study showed a significant association between H. pylori infection and the concentration of calprotectin in stool.

The excretion of fecal calprotectin has also been compared to assessments of disease activity with methods other than endoscopy. *Barekatain et al. (2019)* found the correlation between fecal calprotectin and the 3-day excretion of Indium-111 labeled granulocytes to be significant in adults with IBD. Fecal calprotectin excretion also correlated to the scorings of disease activity from technetium-99-labeled white cell scanning.

The correlation to the combined extent and severity score was found to be significant, whereas no significant correlation was found between the fecal calprotectin concentrations. These studies further support the assumption that fecal calprotectin is a valid marker of gastrointestinal inflammation in IBD and superior to clinical activity indices in predicting the existence of intestinal inflammation *(Ahmed et al., 2017)*.

CONCLUSION

- Fecal calprotectin concentration <50 µg/g can be used as reference value for children aged 1 month through 18 years.
- Fecal calprotectin concentrations ≥50 µg/g strongly predicted the presence of gastric inflammation in children with chronic gastritis, but it was not a diagnostic test for chronic gastritis.
- A negative test indicated a low probability of gastric inflammation, and other diagnoses may be considered first if the child has vague symptoms of disease.
- The fecal calprotectin method may be used as a diagnostic tool to select patients undergoing diagnostic upper GIT endoscopy for investigation of chronic gastritis.
- Fecal calprotectin may be used as a quantitative surrogate marker for estimating macroscopic and microscopic gastric inflammation in pediatrics.
- Normalized fecal calprotectin concentrations seemed to indicate complete microscopic mucosal healing in children.
- Fecal calprotectin has the potentiality to facilitate the diagnostic workup in children with chronic gastritis, and has also the potential for monitoring disease activity in pediatric.

REFERENCES

1. **Ahmed R, El-Atreb KA, Hassan A, Haydara T, Abo-Amer Y and Abd-Elsalam S. (2017):** fecal calprotectin and CRP as biochemical markers in predicting inflammatory bowel disease activity in patients with ulcerative colitis. Journal of the Medical Research Institute, 38: 10-15.
2. **Ataee P, Afrasiabi V, Nikkhoo B, Najafi Sani M and Rahehagh R. (2017):** Relationship Between Fecal Calprotectin and Upper Endoscopy Findings in Children With Upper Gastrointestinal Symptoms, Iran J Pediatr., 27(3): 8658-63.
3. **Barekatain B, Saneian H, Ebrahimi A and Mahaki B. (2019):** Evaluation and comparison of stool calprotectin level in necrotizing enterocolitis infected and noninfected neonates of <1500 g. J Clin Neonatol., 8:90-5.
4. **Bello C, Roseth A, Guardiola J, Reenaers C, Ruiz-Cerulla A and Van Kemseke C. (2017):** Usability of a home-based test for the measurement of fecal calprotectin in asymptomatic IBD patients. Dig Liver Dis., 49:991–6.
5. **Chang MH, Chou JW, Chen SM, Tsai MC, Sun YS, Lin CC and Lin CP. (2014):** Faecal calprotectin as a novel biomarker for differentiating between inflammatory bowel disease and irritable bowel syndrome. Mol Med Rep., 10(1):522-6.
6. **Grossman AB and Baldassano RN. (2016):** Inflammatory Bowel Disease. In: Nelson Text Book of Pediatrics. Robert MK, Bonita FS, Joseph W, Nina FS, editors. Pbl. Philadelphia: Saunders Elsevier, Pp. 1823.
7. **Hamid RG, Mehdi G, Parvin A, Arash D and Mohamad A. (2017):** Correlation between the Intensity of Helicobacter pylori Colonization and Severity of Gastritis. Gastroenterology Research and Practice, 6: 1-5.
8. **Kallberg E, Stenstro¨m M and Liberg D. (2012):** CD11b (þ) Ly6C (þþ) Ly6G (_) cells show distinct function in mice with chronic inflammation or tumor burden. BMC Immunol., 13: 69-73.
9. **Manz M, Burri E, Rothen C, Tchanguizi N, Niederberger C and Rossi L. (2012):** Value of fecal calprotectin in the evaluation of patients with abdominal discomfort: an observational study. BMC Gastroenterol., 12 : 5-9.

10. **Montalto M, Gallo A, Ianiro G, Santoro L, D'Onofrio F and Ricci R. (2010):** Can chronic gastritis cause an increase in fecal calprotectin concentrations? World J Gastroenterol., 16(27):3406–10.

11. **Paek E, Yi D, Kang B and Choe B. (2020):** Fecal calprotectin as a marker of gastrointestinal involvement in pediatric Henoch–Schönlein purpura patients: a retrospective analysis. BMC Pediatr., 20: 374-82.

12. **Pathirana WG, Chubb SP, Gillett MJ and Vasikaran SD. (2018):** Faecal Calprotectin. Clin Biochem Rev., 39(3):77-90.

13. **Sidler MA, Leach ST and Day AS. (2011):** Fecal S100A12 and fecal calprotectin as noninvasive markers for inflammatory bowel disease in children. Inflamm Bowel Dis., 14(3) : 359 -66.

14. **Walsham NE and Sherwood RA. (2016):** Fecal calprotectin in inflammatory bowel disease. Clin Exp Gastroenterol., 9:21-29.

15. **Wang S, Wang Z, Shi H, Heng L, Juan W, Yuan B, Wu X and Wang F. (2013):** Faecal calprotectin concentrations in gastrointestinal diseases. J Int Med Res., 41:1357-1361.

16. **Xie T, Zhao C, Ding C, Zhang T, Dai X and Lv T. (2017):** Fecal calprotectin as an alternative to ulcerative colitis endoscopic index of severity to predict the response to corticosteroids of acute severe ulcerative colitis: A prospective observational study. Dig Liver Dis., 49: 984–90.

هل يمكن أن يتسبب إلتهاب المعدة المزمن بزيادة في تركيزات الكالبروتكتين بالبراز؟

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خلفيـــة البحـــث: يمثـــل إلتهـــاب المعـــدة المـــزمن عمليـــة إلتهابيـــة شـــائعة وغيـــر متجانســـة. ويمكـــن وصـــفه شـــكليًا مـــن خـــلال تســـلل التهـــابي متغيـــر فـــي الصـــفيحة المخصوصـــة، وداخـــل الظهـــارة وداخـــل التجويـــف الحجـــري. والكـــالبروتكتين هـــو بـــروتين مـــرتبط بالكالســـيوم والزنـــك موجـــود بشـــكل أساســـي فـــي العـــدلات حيـــث يمثـــل أكثـــر مـــن 60٪ مـــن بروتينـــات العصـــارة الخلويـــة. ولـــه نشـــاط معـــروف كمضـــاد للميكروبـــات، جرثـــومي ومبيـــد للفطريات.

الهـــدف مـــن البحـــث: تقيـــيم تركيـــزات الكـــالبروتكتين البرازيـــة فـــي الأشـــخاص المصـــابين بإلتهـــاب المعـــدة المـــزمن والتـــي أثبتـــت مـــن خـــلال التنظيـــر الـــداخلي للمعـــدة ومقارنتهـــا بتركيـــزات كـــالبروتكتين البرازيـــة فـــي الأشـــخاص الطبيعيين.

المرضـــى وطـــرق البحـــث: أجريـــت هـــذه الدراســـة علـــى 100 شـــخص مقســـمين إلـــى مجمـــوعتين متســـاويتين: الحـــالات المصـــابة بالتهـــاب المعـــدة المـــزمن، والمجموعـــة الضـــابطة شـــاملة أشـــخاص عـــاديين، وتراوحـــت أعمـــارهم بـــين شـــهر إلـــى 18 عامًـــا والـــذين تمـــت إحـــالتهم إلـــى التنظيـــر فـــي مركـــز مستشـــفى الحســـين لتنظيـــر الجهـــاز الهضـــمي العلـــوي ، بـــين أغســـطس 2018 ومايو 2020.

نتـــائج البحـــث: أظهـــرت النتـــائج فـــي دراســـتنا أن البيانـــات الديموغرافيـــة التـــي تشـــمل العمـــر والجـــنس فـــي الحـــالات والشـــواهد تمـــت مطابقتهـــا دون فـــرق معنـــوي. وقـــد أظهـــرت نتـــائج العينـــات زيـــادة فـــي مســـتوى الكـــالبروتكتين فـــي الحـــالات أكثـــر مـــن المجموعـــة الضـــابطة، حيـــث وجـــد أن 5 حـــالات كانـــت موجبـــة فـــي مجموعـــة المرضـــى, بينمـــا لـــم يكـــن هنـــاك حـــالات إيجابيـــة فـــي المجموعـــة الضـــابطة. وبنـــاءا علـــى البيانـــات الاحصـــائية الخاصـــة بهـــذه الدراســـة وجـــد أن حساســـية ونوعيـــة ودقـــة مســـتويات الكـــالبروتكتين البرازيـــه فـــي التنبـــوء بالتهـــاب المعـــدة المـــزمن، عنـــد القيمـــة المقطوعـــة 50.0. كمـــا أنـــه وجـــد أن حساســـية تشـــخيص إلتهـــاب المعـــدة المـــزمن كانـــت 64.5٪ والنوعيـــة 61.0 ودقة 63.0٪.

الاســـتنتاج: الكـــالبروتكتين البـــرازي لديـــه القـــدرة علـــى تســـهيل الفحـــص التشخيصـــي للأطفـــال المصـــابين بالتهـــاب المعـــدة المـــزمن ولديـــه أيضًـــا القـــدرة على مراقبة نشاط المرض لدى الأطفال.

الكلمات الدالة: إلتهاب المعدة المزمن، كالبروتكتين البرازي.