DOI: 10.7860/JCDR/2025/75349.20968

Original Article

Pathology Section

Precision versus Protocol: A Cross-sectional Analysis of Gastric Biopsy Techniques in Detecting Premalignant Lesions

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ABSTRACT

Introduction: Patients with Chronic Atrophic Gastritis (CAG) and Gastric Intestinal Metaplasia (GIM) are at risk of developing gastric adenocarcinoma. The early and accurate detection of these lesions is critical for effective intervention and improved patient outcomes.

Aim: To study the Sydney protocol and targeted single-site biopsy in detecting premalignant gastric lesions, identifying the most reliable method for early diagnosis.

Materials and Methods: This was a cross-sectional study conducted in the Department of Pathology and Laboratory Medicine at Jagjivan Ram Hospital, Western Railway, Mumbai, Maharashtra, India from June 2023 to February 2024. A total of 100 gastric biopsies were included, comprising 50 cases of targeted single-site biopsy and 50 cases following the Sydney protocol. The Sydney protocol recommends obtaining biopsies from five specific sites in the stomach: two from the antrum, two from the body and one from the incisura angularis. The parameters studied were atrophy and intestinal metaplasia, which were assessed using histopathological evaluation of gastric biopsy specimens. The results were analysed by calculating interobserver agreement using kappa statistics to evaluate the reproducibility and reliability of the diagnostic criteria.

Results: Out of 50 targeted single-site biopsies, urease was positive in 14 (28%) cases. A total of 14 (28%) patients had CAG, of which 12 were stage I and 2 were stage II. A total of 4 (8%) patients had intestinal metaplasia, out of which three were stage I and one was stage II. Out of 50 Sydney protocol-compliant biopsies, 20 (40%) patients' urease tests were positive in at least one biopsy site. A total of 30 (60%) patients had CAG, of which 15 (30%) had CAG in all three sites (corpus, antrum and incisura), 4 (8%) had CAG only in the corpus, 4 (8%) had CAG only in the antrum, 3 (6%) had CAG in both the antrum and incisura, 2 (4%) had CAG in both the corpus and incisura, and 2 (4%) had CAG only in the corpus. Out of these 30 patients with CAG, 18 (36%) were classified as stage I, 9 (18%) as stage II, and 3 (6%) as stage III. A total of 10 (20%) patients had GIM, of which 6 (12%) had GIM only in the antrum, 2 (4%) had intestinal metaplasia in both the corpus and antrum, 1 (2%) had intestinal metaplasia in both the antrum and incisura and 1 (2%) had intestinal metaplasia only in the incisura. Out of these 10 patients with intestinal metaplasia, 5 (10%) were classified as stage I, 3 (6%) as stage II, 1 (2%) as stage III, and 1 (2%) as stage IV. Additionally, 3 (6%) patients had low-grade dysplasia.

Conclusion: The Sydney protocol outperforms targeted single-site biopsy in detecting premalignant gastric lesions due to its comprehensive sampling from multiple sites, thereby reducing the risk of missed lesions. This thorough approach ensures accurate diagnosis and highlights the importance of endoscopic follow-up for ongoing patient management.

Keywords: Operative link on gastric intestinal metaplasia, Operative link on gastritis assessment, Sydney protocol, Targeted biopsy

INTRODUCTION

Gastric cancer remains a significant global health concern and is the fourth leading cause of cancer-related deaths worldwide. By 2040, the global incidence of gastric cancer is expected to rise to approximately 1.8 million new cases, with an estimated 1.3 million deaths annually [1]. Patients with CAG and GIM are at risk of developing gastric adenocarcinoma and Neuroendocrine Tumors (NET), as these conditions are considered precancerous lesions. The early and accurate detection of these lesions is critical for effective intervention and improved patient outcomes [2].

The adoption of clinical guidelines has strengthened evidence-based medicine in the management of chronic gastritis, highlighting that endoscopic evaluation of gastric mucosal pathology alone is insufficient and not considered best practice. Therefore, pathologists are integral in accurately diagnosing these preneoplastic lesions. *Helicobacter pylori* (*H. pylori*) is a known risk factor for gastric adenocarcinoma, causing chronic inflammation that leads to DNA damage and genetic changes, thereby increasing cancer risk. Its virulence factors disrupt cellular signaling and promote Epithelial-Mesenchymal Transition (EMT), a process where cells become more invasive and prone to metastasis. Early eradication of *H. pylori* can significantly reduce the likelihood of gastric adenocarcinoma [2].

H. pylori infection continues to be highly prevalent, with a systematic review of 184 studies (1970-2015) reporting a global prevalence of 48.5% across 62 countries. Its distribution varies significantly based on age, underlying conditions, geographic region, socio-economic status, hygiene and ethnic background [3].

The Sydney Protocol recommends taking biopsies from five different sites in the stomach to ensure a thorough evaluation of the gastric mucosa: two from the antrum (2-3 cm from the pylorus, one each from the lesser and greater curvature), two from the body (8 cm from the cardia, one each from the lesser and greater curvature), and one from the incisura angularis [4]. Virtual chromoendoscopy has shown greater accuracy than traditional white light endoscopy in detecting GIM and CAG; however, its adoption in clinical practice is still not widespread [5].

The primary objective of this study was to evaluate the diagnostic yield of both the targeted biopsy approach and the Sydney Protocol in detecting premalignant gastric lesions, categorising these lesions using the Operative Link on Gastritis Assessment (OLGA) and Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM) systems. The novelty of this study lies in comparing targeted single-site biopsy with the Sydney Protocol

for detecting premalignant gastric lesions, focusing on atrophy and intestinal metaplasia. By analysing interobserver agreement with high kappa values, this study highlights the reliability of these diagnostic methods, providing valuable insights not widely explored in previous research.

MATERIALS AND METHODS

This was a 9-month cross-sectional study conducted in the Department of Pathology and Laboratory Medicine at Jagjivan Ram Hospital, Western Railway, Mumbai, Maharashtra, India from June 2023 to February 2024. Institutional Ethical Committee (IEC) approval was obtained for this study (IEC No. EC/19/00153). A total of 100 gastric biopsies were included, comprising 50 cases of targeted single-site biopsy and 50 cases following the Sydney protocol.

Inclusion criteria: Patients who had complaints (dysphagia, dyspepsia, unexplained weight loss, persistent nausea or vomiting, malena) for at least a month were included in the study.

Exclusion criteria: Patients who had used Proton Pump Inhibitors (PPIs) in the past two weeks, had a history of gastric malignancy or surgery, or had previously undergone *H. pylori* eradication therapy were excluded from the study.

Study Procedure

Biopsy specimens from each site were fixed separately in 10% neutral formaldehyde for eight hours. During macroscopic examination, the samples were placed in coded cassettes based on their location, with size and quantity noted and wrapped in blotting paper to prevent loss. After routine tissue processing (Infinity MPTP-17A, India), the samples were embedded in paraffin blocks. Sections four microns thick were cut from the blocks using a microtome and stained with Haematoxylin and Eosin (H&E), Periodic Acid Schiff-Alcian Blue (PASAB) and Giemsa stains.

The slides were then examined independently under a light microscope by two pathologists specialising in gastroenterology. In cases of uncertainty, the pathologists consulted each other and prepared a consensus report. The interobserver agreement for intestinal metaplasia and atrophy was excellent, with kappa values of 1.0 and 0.918, respectively, highlighting the reliability and reproducibility of the diagnostic criteria used in the study [Table/Fig-1,2].

Intestinal metaplasia (Kappa=1.0)		Pathologist B		
		Present	Absent	Total
Pathologist A	Present	14	0	14
	Absent	0	86	86
	Total	14	86	100

[Table/Fig-1]: Kappa statistics for intestinal metaplasia.

Atrophy (Kappa=0.918)		Pathologist B		
		Present	Absent	Total
Pathologist A	Present	40	2	42
	Absent	2	56	58
	Total	42	58	100

[Table/Fig-2]: Kappa statistics for gastric atrophy.

Atrophy and intestinal metaplasia were graded as mild (<30%), moderate (31-60%), and severe (>60%), and staged from I to IV using the OLGA/OLGIM system. The OLGA/OLGIM stage (I to IV) is determined by combining the overall antrum score with the overall corpus score for atrophy and intestinal metaplasia [6].

STATISTICAL ANALYSIS

Data collected were entered into a Microsoft Excel spreadsheet for analysis and results were expressed in terms of frequency and percentage.

RESULTS

A total of 100 cases were included in the study. Of these, 55% of the patients were male and 45% female, with an age range of 14 to 88 years [Table/Fig-3]. The distribution of cases is shown in [Table/Fig-4]. Urease positivity was observed in 14 cases (28%) of targeted biopsies, while 20 cases (40%) of Sydney-compliant biopsies showed urease positivity in at least one biopsy site (antrum, corpus, or incisura angularis). The presence of *H. pylori* in these positive cases was further confirmed using Giemsa special stain.

Age groups (years)	Male (No. of cases)	Female (No. of cases)	Total (No. of cases)
0-10	0	0	0
11-20	2	1	3
21-30	4	4	8
31-40	9	4	13
41-50	8	14	22
51-60	8	13	21
61-70	16	6	22
71-80	6	2	8
81-90	2	1	3
Total	55	45	100

[Table/Fig-3]: Age distribution of cases.

		Targeted single site biopsies		Sydney compliant biopsies	
Total number of cases		50		50	
Urease	Positive	14 (28%)		20 (40%)	
Chronic Atrophic Gastritis (CAG)		14 (28%)	Stage-I - 12 (24%)	30 (60%)	Stage-I - 18 (36%)
			Stage-II - 2 (4%)		Stage-II - 9 (18%)
					Stage-III - 3 (6%)
Gastric Intestinal Metaplasia (GIM)		4 (8%)	Stage-I - 3 (6%)	10 (20%)	Stage-I - 5 (10%)
			Stage-II - 1 (2%)		Stage-II - 3 (6%)
					Stage-III - 1 (2%)
					Stage-IV - 1 (2%)
Low-grade dysplasia		0		3 (6%)	

[Table/Fig-4]: Targeted single site and Sydney protocol compliant biopsies.

Targeted single site biopsies: Out of 50 cases, 36 biopsies were taken from the antrum, seven from the corpus, five from the fundus, and two from the pylorus. The distribution of cases is shown in [Table/Fig-5]. Targeted single-site biopsy data revealed Chronic Atrophic Gastritis (CAG) in 14 cases, with the most common site being the antrum, followed by the corpus. Out of these 14 cases, 12 were classified as stage I, and 2 were classified as stage II. Urease positivity was observed in four cases, all from antrum biopsies. Gastric Intestinal Metaplasia (GIM) was seen in four cases, with two cases each from the antrum and the corpus. Three cases were classified as stage I, and one case was classified as stage II. The limited sampling scope of targeted biopsies prevents accurate classification of CAG and IM.

	Total cases	Antrum	Corpus	Fundus	Urease positive cases
CAG	14	11	2	1	4 (All Antrum)
GIM	4	2	2	-	-

[Table/Fig-5]: CAG and GIM- targeted single site biopsies.

Sydney Protocol Compliant Biopsies

A) **Chronic Atrophic Gastritis (CAG):** Overall, there were 30 cases of CAG, 12 of which were urease positive, indicating potential *H. pylori* infection. The majority (15 cases) had CAG at all three sites, while others had localised involvement, mainly in the antrum (4 cases) and corpus (4 cases). Less

common patterns included CAG in both the antrum and incisura (3 cases), corpus and incisura (2 cases), and isolated incisura involvement (2 cases). CAG was observed in 21 cases involving the corpus, of which seven exhibited ECL cell hyperplasia. The distribution of cases is shown in [Table/Fig-6].

Antrum	Corpus	Incisura angularis	Urease positive cases	Enterochromaffin-like cell hyperplasia cases	Chronic Atrophic Gastritis (CAG) positive cases
+	+	+	8	4	15
+	-	+	2	Nil	3
-	+	+	1	1	2
-	-	+	Nil	Nil	2
-	+	-	1	2	4
+	-	-	Nil	Nil	4
Total cases			12	7	30

[Table/Fig-6]: CAG - Sydney protocol compliant biopsies: (+: Atrophy present; -: Atrophy absent)

B) Gastric Intestinal Metaplasia (GIM): Among 10 GIM cases, one was urease positive. Most (6 cases) had GIM in the antrum, with fewer cases involving multiple sites. Staging showed 5 cases in stage I, 3 in stage II, and 1 each in stage III and stage IV. The distribution of cases is shown in [Table/Fig-7].

Antrum	Corpus	Incisura angularis	Urease positive cases	Gastric Intestinal Metaplasia (GIM) positive cases
+	+	-	Nil	2
+	-	+	Nil	1
+	-	-	1	6
-	-	+	Nil	1
Total cases			1	10

[Table/Fig-7]: GIM - Sydney protocol compliant biopsies: (+: Present; -: Absent)

With biopsies taken according to the Sydney protocol, a significant number of cases would have been missed if samples were obtained from a single site. Atrophic gastritis would have been underdiagnosed in 26-30% of cases, while intestinal metaplasia, particularly from the corpus and incisura angularis, would have been missed in 80% of cases [Table/Fig-8].

Biopsy site	No. of atrophic gastritis cases that would have been missed if biopsies were taken exclusively from the respective site	No. of Intestinal metaplasia cases that would have been missed if biopsies were taken exclusively from the respective site
Antrum	8 (26%)	1 (10%)
Corpus	9 (30%)	8 (80%)
Incisura angularis	8 (26%)	8 (80%)

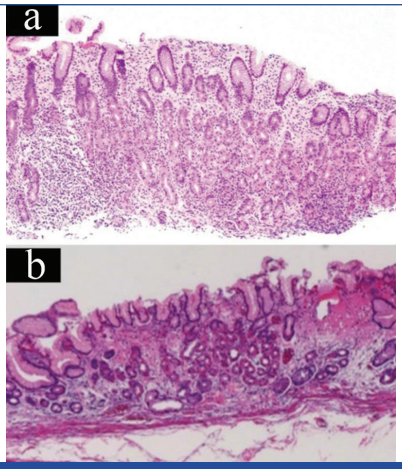
[Table/Fig-8]: No. of CAG and GIM cases that would have been missed if biopsies were taken exclusively from the respective site.

C) Chronic Atrophic Gastritis (CAG) and Gastric Intestinal Metaplasia (GIM): Two cases had both CAG and GIM.

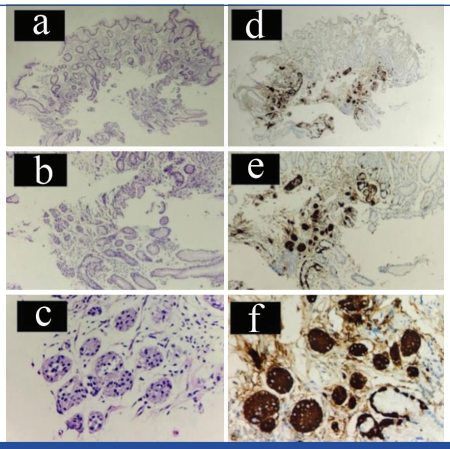
D) Dysplasia: Three cases had low-grade dysplasia and were graded according to the modified Vienna classification of gastrointestinal epithelial neoplasia. Histopathological features of various lesions are depicted in [Table/Fig-9-12], showing various levels of atrophy and metaplasia.

DISCUSSION

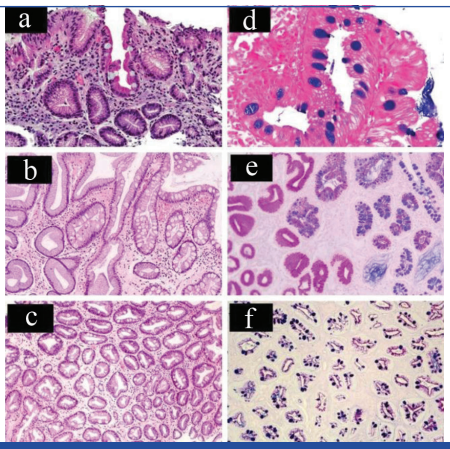
The updated Sydney system emphasises the importance of obtaining biopsies from the antrum, corpus and incisura angularis for an accurate diagnosis of gastric diseases. However, in everyday clinical practice, this approach is often modified, with only one or two antral biopsies being taken. The biopsy approach is often adjusted due to practical challenges, including time limitations during endoscopic



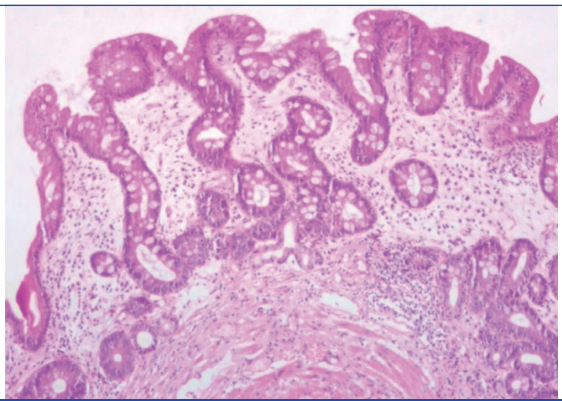
[Table/Fig-9]: Gastric body with mild patchy atrophy (A- Grade I) and moderate atrophy (B- Grade II) (H&E, 100x).



[Table/Fig-10]: (a-c) Gastric body with severe atrophy (Grade III) exhibiting loss of oxyntic mucosa (antralisation) with ECL Cell hyperplasia – both linear and nodular (<0.5 mm), highlighted by chromogranin A immunostain (d-f) (H&E, 100x).



[Table/Fig-11]: (a-c) Mild, moderate and severe Gastric Intestinal Metaplasia (GIM) respectively, as highlighted by AB-PAS special stain (d-f) (H&E, 100x).



[Table/Fig-12]: Gastric body exhibits severe intestinal metaplasia with complete loss of oxyntic mucosal glands (H&E, 100x).

procedures and the costs associated with taking multiple biopsies. In high-volume settings, endoscopists tend to focus on biopsies from the antrum to save time, particularly in resource-limited environments where financial constraints play a significant role.

Chronic *H. pylori* gastritis affects approximately two-thirds of the global population and is a significant cause of most Non Steroidal Anti-Inflammatory Drugs (NSAIDs)-induced duodenal and gastric ulcers, as well as the majority of gastric Mucosa-Associated Lymphoid Tissue (MALT) lymphomas. *H. pylori* infection increases the lifetime risk of peptic ulcer disease to 15 to 20% and is directly associated with 70% of gastric cancers and 85 to 90% of primary gastric MALT-type extranodal marginal zone lymphomas [7]. *H. pylori* contributes to the development of gastric cancer through its virulence factors, such as the VacA and CagA proteins. VacA (vacuolating cytotoxin A) induces cell damage and immune evasion, while CagA (cytotoxin-associated gene A) disrupts cellular signaling pathways, leading to chronic inflammation and an increased risk of malignant transformation in the gastric epithelium. Current guidelines recommend eradication therapy only for individuals in whom the infection is identified, due to the increasing resistance of *H. pylori* to antibiotics in the current treatment protocols [8].

Urease positivity was detected in 14 cases (28%) of targeted biopsies. Sydney-compliant biopsies showed urease positivity in 20 cases (40%) at one or more biopsy sites, aligning with findings from a prospective study by Torun C et al., where 37% of patients tested positive for *H. pylori* in at least one biopsy site [2]. Using the Sydney protocol biopsy, atrophic gastritis without intestinal metaplasia was identified in 30 patients (60%). This approach enhances the sensitivity of detecting atrophic gastritis, as relying on biopsies from only a single site—whether the antrum, corpus, or incisura angularis—would have resulted in 26%, 30%, and 26% of atrophic gastritis cases being missed, respectively [Table/Fig-8]. Notably, three patients (6%) were diagnosed with Stage III CAG, characterised by moderate atrophy in both the antrum and corpus. Had only targeted biopsies been performed, these cases would likely have been understated as Stage II, potentially leading to the omission of crucial endoscopic follow-ups. Supporting this, Torun C et al., reported that if a single biopsy were taken from the antrum or incisura, extensive atrophy would have been missed in 33% and 22% of cases, respectively [2]. The higher discrepancy between these findings may be attributed to differences in sample size, as their study included a larger cohort. CAG was observed in 21 cases involving the corpus, with seven of these cases showing ECL cell hyperplasia. Urease positivity was detected in eight of the 21 cases, but it was negative in those cases with ECL cell hyperplasia. Patients with severe degrees of ECL-cell hyperplasia over a period of several years can progress to ECL-cell carcinoids/Type I NET. Follow-up endoscopic surveillance with biopsies and Sr. Chromogranin monitoring is recommended every 1-2 years for these patients [9,10].

CAG was observed in 21 cases involving the corpus, with seven of these cases showing ECL cell hyperplasia. Urease positivity was detected in eight of the 21 cases, but it was negative in those cases with ECL cell hyperplasia. Patients with severe degrees of ECL cell hyperplasia over a period of several years can progress to ECL cell carcinoids/Type I NET. Follow-up endoscopic surveillance with biopsies and serum Chromogranin monitoring is recommended every 1 to 2 years for these patients [9,10].

Endocrine cell hyperplasia frequently arises from functional changes in the stomach, particularly in autoimmune atrophic gastritis. In this condition, decreased stomach acid (hypochlorhydria or achlorhydria) leads to increased G-cell activity in the antrum and elevated serum gastrin levels. This results in the proliferation of histamine-secreting Enterochromaffin-Like (ECL) cells in the oxyntic mucosa. Neuroendocrine cell proliferation, which is common in advanced atrophic gastritis, is most accurately assessed using specific immunostains. Notably, micronodular ECL hyperplasia is associated with an increased risk of developing type I (well-differentiated) Neuroendocrine Tumours (NETs) [11].

The prevalence of GIM varies across different geographic regions. In present study, using the Sydney protocol, GIM was identified in 10 patients (20%). Similarly, Eriksson NK et al., reported a prevalence of approximately 19%, while Olmez S et al., found it to be around 13.8%, and Almouradi T et al., reported a prevalence of about 15% [12-14]. Using the Sydney protocol biopsy, GIM was identified in 10 patients (20%), with single-site biopsies from the antrum, corpus, or incisura angularis missing 10%, 80%, and 80% of cases, respectively [Table/Fig-8]. One case of Stage III intestinal metaplasia would have been downgraded to Stage II if only targeted biopsies had been performed, potentially leading to inadequate follow-up. These findings reinforce that intestinal metaplasia is more commonly seen in the antrum, a pattern also observed by Eriksson NK et al., who reported its highest prevalence in the antrum, followed by the incisura angularis [12].

Among the 30 cases of CAG, 18 were classified as Stage I, nine as Stage II, and three as Stage III. In the 10 cases of GIM, five were Stage I, three were Stage II, one was Stage III, and one was Stage IV. OLGIM/OLGA Stage I and Stage II were classified as low-risk, and these patients can be discharged from gastric surveillance programs. Lesions classified as OLGIM/OLGA Stage III and IV are associated with an increased risk of gastric cancer. The European Society of Gastrointestinal Endoscopy (ESGE) recommends endoscopic surveillance every three years for patients with OLGIM Stage III and IV lesions [15].

Virtual chromoendoscopy has demonstrated higher accuracy compared to traditional white light endoscopy in identifying GIM and CAG. Histopathology is crucial for obtaining a definitive diagnosis and understanding the pathology of the disease, while chromoendoscopy is valuable for improving the detection and targeting of lesions during endoscopy. Both methods complement each other and are often used together to provide a comprehensive evaluation of gastrointestinal conditions. However, their use in clinical practice remains limited, making it advisable to take multiple biopsies when these advanced modalities are not available [16].

In conclusion, a single-site biopsy may be sufficient for diagnosing *H. pylori* infection, but it falls short for identifying atrophy, intestinal metaplasia, and assessing their extent. Following the Sydney protocol improves the detection of CAG and GIM.

Limitation(s)

1. With a relatively small sample size and the study being a single-centre study, these findings may not be generalisable to a broader population.
2. Lack of longitudinal data: The study does not include follow-up data, limiting the ability to assess the progression of detected lesions over time.

CONCLUSION(S)

The Sydney protocol outperforms targeted single-site biopsy in detecting premalignant gastric lesions due to its comprehensive sampling from multiple sites, thereby reducing the risk of missed lesions and minimising the chance of false-negative cases. Relying solely on targeted biopsies can overlook the patchy, subtle and region-specific nature of these lesions. This method enhances diagnostic accuracy and reinforces the significance of endoscopic follow-up in patient care, ultimately lowering morbidity and mortality rates.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 13, 2024
- Manual Googling: Jan 31, 2025
- iThenticate Software: Mar 08, 2025 (14%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Sep 10, 2024**
Date of Peer Review: **Dec 03, 2024**
Date of Acceptance: **Mar 11, 2025**
Date of Publishing: **May 01, 2025**