

Clinical implications of the presence of anti-parietal cell antibody in patients with stomach neoplasms

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INTRODUCTION

Autoimmune gastritis is an overlooked disease entity in the diagnosis and treatment of stomach neoplasm. We aimed to evaluate the prevalence of anti-parietal cell antibody (APCA) positivity and its clinical implication among patients with stomach neoplasm.

METHODS

Between April 2021 and June 2023, 767 patients visiting Samsung Medical Center for stomach neoplasm underwent blood tests for APCA. Clinicopathological features of patients were reviewed and compared between those with and without APCA. Endoscopic images were reviewed for 53 patients with positive APCA.

RESULTS

Among 767 patients with APCA results, there were 734 gastric cancer (GC)/adenoma and 12 neuroendocrine tumor (NET) patients. The rate of APCA positivity was 6.9% (53/767), 6.5% (48/734), and 25% (3/12) among total study population, GC/adenoma, and NET patients, respectively. Compared to APCA negative patients, APCA positive patients had lower pepsinogen I level (median 43.3 ng/mL versus 60.2 ng/mL, P = 0.005) and lower pepsinogen I/pepsinogen II ratio (median 2.1 versus 2.5, P = 0.017). There was no difference in the rate of anti-Helicobacter pylori (HP) antibody positivity between those with and without APCA (75.5% versus 77.8%, P = 0.819). Upon review of endoscopic images, reverse atrophy consistent with autoimmune gastritis was found in 13.2% (7/53) in all patients with positive APCA and 8.3% (4/48) and 100% (3/3) in GC and NET patients with positive APCA, respectively. Among patients with reverse atrophy, median pepsinogen I level and pepsinogen I/pepsinogen II ratio were 23.8 ng/mL (interquartile range: 13.6-59.4 ng/mL) and 1.8 (interquartile range: 1.2-2.6), respectively.

Table 1. Clinical characteristics of patients with and without anti-parietal cell antibody

	APCA (+) (n = 53)	APCA (-) (n = 714)	Total (n = 767)	P-value
Sex				0.243
F	23 (43.4%)	246 (34.5%)	269 (35.1%)	
М	30 (56.6%)	468 (65.5%)	498 (64.9%)	
Age	61.0 [54.0;67.0]	63.0 [54.0;71.0]	63.0 [54.0;71.0]	0.238
H. Pylori Ab				0.819
Negative	13 (24.5%)	158 (22.2%)	171 (22.3%)	
Positive	40 (75.5%)	555 (77.8%)	595 (77.7%)	
ВМІ	24.1 [21.9;26.4]	23.8 [22.0;26.4]	23.9 [21.9;26.4]	0.682
Pepsinogen I (ng/mL)	43.3 [23.8;75.9]	60.2 [37.5;94.1]	59.0 [36.5;93.0]	0.005
Pepsinogen II (ng/mL)	24.2 [13.8;33.3]	23.9 [14.7;38.7]	23.9 [14.6;38.5]	0.291
P1/P2 ratio	2.1 [1.2; 2.9]	2.5 [1.7; 3.5]	2.5 [1.7; 3.5]	0.017
Hemoglobin (g/dL)	13.6 [12.8;14.7]	13.7 [12.6;14.8]	13.7 [12.6;14.8]	0.902

Table 2. Comparison of clinical characteristics in anti-parietal cell antibody positive patients with and without reverse atrophy

cell antibody positive patients with and without reverse altophy								
Reverse atrophy	Yes (N=7)	No (N=46)	Total ACPA (+) (N=53)	P-value				
Age	56.3 ± 12.2	61.3 ± 9.5	60.7 ± 9.9	0.214				
Sex				0.034				
F	6 (85.7%)	17 (37.0%)	23 (43.4%)					
M	1 (14.3%)	29 (63.0%)	30 (56.6%)					
Helicobacter Ab				0.053				
Negative	4 (57.1%)	9 (19.6%)	13 (24.5%)					
Positive	3 (42.9%)	37 (80.4%)	40 (75.5%)					
Pepsinogen 1 (ng/mL)	23.8 [13.9;54.6]	43.8 [27.1;88.6]	43.3 [23.8;75.9]	0.172				
Pepsinogen 2 (ng/mL)	13.5 [11.1;20.2]	25.2 [14.0;35.5]	24.2 [13.8;33.3]	0.059				
P1/P2 ratio	1.8 [1.2; 2.5]	2.2 [1.2; 2.9]	2.1 [1.2; 2.9]	0.617				
Hemoglobin								
Anemia	1 (14.3%)	11 (23.9%)	12 (22.6%)	1.000				
Normal	6 (85.7%)	35 (76.1%)	41 (77.4%)					

Table 3. Clinical characteristics of patients with reverse atrophy in endoscopic examination

Histology	Sex	Age	H. Pylori Ab	P1 level (ng/mL)	P1 (≥70.1 ng/mL)	P1/P2 ratio	P1/P2 ratio (≥3.1)	Gastrin (pg/mL)	Hb (g/dL)	Tx
NET	F	52	Negative	14.2	Low	1.2	L	1000	12.3	ESD
NET	F	42	Negative	23.8	Low	1.8	L	735.1	13.8	ESD
NET	F	52	Negative	49.8	Low	6.6	Н	NA	11.4	F/U loss
GC	F	73	Negative	5.3	Low	0.3	L	NA	12.1	OP
GC	M	72	Positive	13.6	Low	1.3	L	NA	13.2	ESD
GC	F	58	Positive	59.4	Low	2.6	L	NA	13.0	ESD
GC	F	45	Positive	78.1	High	2.4	L	NA	13.4	OP

CONCLUSIONS

In patient with stomach neoplasm, APCA was present more frequently than previously acknowledged and autoimmune gastritis may have been an underestimated etiology. Further larger scale studies are warranted to confirm the clinical implications of the presence of APCA and autoimmune gastritis in patients with stomach neoplasm.