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Doppler sonography of hemodynamic changes of the celiac artery in chronic active gastritis

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Aim: In this study, we determined how Doppler parameters of the celiac artery (CA) are affected in patients with chronic gastritis.

Materials and methods: We examined 23 patients (group 1) with chronic active gastritis (diagnosed by endoscopy and histology) and 19 asymptomatic control subjects (group 2) with duplex Doppler ultrasonography. The patient group (group 1) was classified in 2 subgroups according to the severity of the inflammation. The second classification was done according to the presence of the *Helicobacter pylori* infection within group 1. Mean peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI) of the CA were compared.

Results: The mean PSV and the mean EDV were significantly lower in patients than in controls ($P < 0.05$). The mean PI and the mean RI were higher in the patient group than in the controls. However, these differences were not statistically significant ($P > 0.05$).

Conclusion: In this preliminary study, patients with chronic active gastritis, regardless of the degree of the inflammation and the presence of *H. pylori*, were associated with a decrease in CA flow velocities that could be seen on Doppler sonography. These data might be useful in understanding the pathophysiology of gastritis and lead the way for future studies.

Key words: Doppler ultrasonography, celiac artery, chronic gastritis

Introduction

Gastric mucosal blood flow supplies oxygen and nutrients, removes hydrogen ions derived from hydrochloric acid, and protects mucosa (1). When the blood supply decreases, mucosal ischemia and damage develop (1,2).

Previous reports evaluating the gastric flow studied gastric mucosal flow by using laser Doppler flowmetry during endoscopy. Laser Doppler flowmetry is a technique used to assess perfusion in the skin and other tissues. The Doppler shift can be analyzed to determine the blood velocity in the microvessels as used in the large vessels. The frequencies in the Doppler-shifted spectra in proportion to the velocity of the blood cells, and the intensity of signal at each frequency in the spectra, are proportionate to the number of cells travelling at that velocity. Both velocity of the blood and volumetric blood flow can be determined (3). Guslandi et al. found that mucosal blood flow in chronic gastritis was reduced regardless of concomitant *Helicobacter pylori* infection (3). Wang et al. showed that mucosal blood flow determined with laser Doppler flowmetry was significantly slower in a group with active ulcer than in the normal group (4).

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The aim of our study was to evaluate hemodynamic changes of the celiac artery (CA), feeding the stomach, in patients with chronic gastritis as assessed by transabdominal duplex Doppler ultrasonography.

Materials and methods

We examined 23 patients, aged 18 to 66 years (group 1 = 18 women, 5 men; mean age 45.1 ± 11.6 years), who underwent upper gastrointestinal endoscopy for dyspeptic symptoms, and 19 asymptomatic control subjects, aged 20 to 67 years (group 2 = 14 women, 5 men; mean age 42.3 ± 12.7 years), with transabdominal Doppler sonography. The patients were divided into 2 different subgroups. Group 1a consisted of 17 patients with mild gastric inflammation and group 1b consisted of 6 patients with moderate–severe inflammation determined by endoscopy and histology. The other classification was done according to *Helicobacter pylori* infection within Group 1. Group 1c consisted of 11 patients with *H. pylori* infection and group 1d consisted of 12 patients without *H. pylori* infection. None of these subjects had cardiac disease, diabetes mellitus, or hypertension. All diagnosis of chronic active gastritis and *H. pylori* was based on endoscopic findings and histologic examination. Sonographic examinations were performed before the medical treatment.

Sonography was performed in the morning, after patients had fasted overnight. Since mesenteric blood flow reduces with exercise, examinations were performed under resting conditions, in the supine position. The entire abdomen was also examined with a focus on any possible abnormal finding by B-mode ultrasonography. Neither patient group nor control group had any liver or splenic diseases. Both B-mode and Doppler examinations were performed with a Siemens Sonoline Antares (Germany) equipped with C5-2 MHz and CH4-1 MHz transducers, or a Toshiba SSA-270A (Japan) equipped with a 3.5 MHz transducer, by an experienced radiologist (A.K.) who was informed of dyspeptic patients but was unaware of the results of endoscopy and histology. The CA was evaluated in a short segment before branching (Figure). The pulse repetition frequency was optimized to record medium-to-high arterial velocities. The Doppler angle was kept at less than 60°. The wall filter was set as low as possible to detect slow diastolic velocity. Peak systolic velocity (PSV), end-diastolic velocity (EDV), resistive index (RI), and pulsatility index (PI) were automatically calculated.

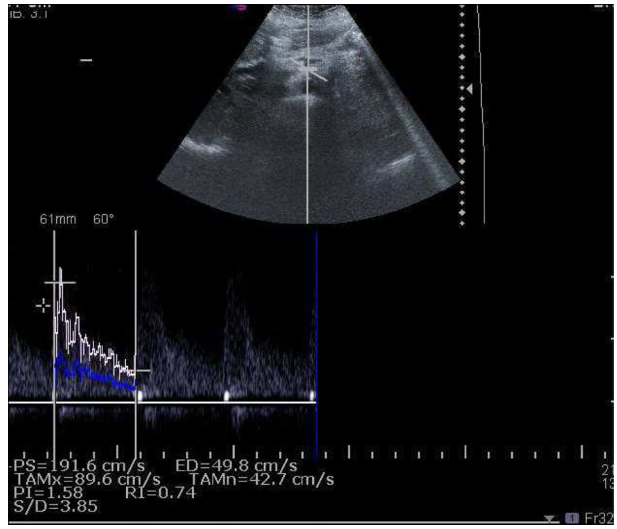

Figure. Duplex sonogram of the celiac artery with normal flow pattern.

Statistical evaluation of the results was performed using the Mann–Whitney U test. A P-value smaller than 0.05 was considered significant.

Results

Doppler parameters of the CA in patient and control groups are shown in the Table. The mean PSV and mean EDV were significantly lower in group 1 (1.52 ± 0.29 m/s and 0.37 ± 0.14 m/s, respectively) than in group 2 (1.84 ± 0.35 m/s and 0.52 ± 0.19 m/s, respectively) (P = 0.002 and P = 0.005, respectively). Although the mean PI in group 1 (1.90 ± 0.92) was higher than in the control group (1.50 ± 0.47), the difference between means was not statistically significant (P > 0.05). Furthermore, although the mean RI was higher in patients than in controls, the difference was not significant (0.74 ± 0.10 and 0.71 ± 0.09, respectively; P > 0.05). The mean PSV and mean EDV were lower in patients with moderate–severe inflammation (group 1b) than in patients with mild inflammation (group 1a). However, these differences were not significant (P > 0.05). The same statistically insignificant difference was seen between group 1c (with *H. pylori*) and group 1d (without *H. pylori*).

Table. Doppler parameters of celiac artery in patients with chronic active gastritis (group 1) and in the control group (group 2). Subgroups of patients are as follows: group 1a = mild gastric inflammation, group 1b = moderate–severe inflammation, group 1c = *H. pylori*-positive, group 1d = *H. pylori*-negative.

Group	Mean PSV, m/s	Mean EDV, m/s	Mean RI	Mean PI
1	1.52 ± 0.29	0.37 ± 0.14	0.74 ± 0.10	1.90 ± 0.92
2	1.84 ± 0.34	0.52 ± 0.19	0.71 ± 0.09	1.50 ± 0.47
1a	1.54 ± 0.23	0.39 ± 0.12	0.75 ± 0.09	1.75 ± 0.72
1b	1.46 ± 0.44	0.34 ± 0.17	0.74 ± 0.15	2.35 ± 1.35
1c	1.49 ± 0.35	0.36 ± 0.15	0.75 ± 0.12	2.14 ± 1.13
1d	1.53 ± 0.24	0.39 ± 0.12	0.74 ± 0.09	1.69 ± 0.68

Abbreviations: PSV, peak systolic velocity; EDV, end-diastolic velocity; RI, resistive index; PI, pulsatility index.

Discussion

From the celiac trunk arise the left gastric artery, the common hepatic artery, and the splenic artery, giving their branches to the stomach and duodenum. However, variant hepatic and celiac arterial anomalies have been reported in the literature (6–8).

Doppler sonography is a valuable tool for the assessment of the blood flow of the mesenteric territory. It is portable, quantitative, and noninvasive (9,10). In patients with inflammatory bowel diseases, such as celiac disease, Crohn's disease, ulcerative colitis, and Behçet's disease, blood flow velocities and volumes have been evaluated (11–13). Doppler studies have shown correlations between splanchnic blood flow (including superior and inferior mesenteric arteries) and inflammatory bowel disease (Crohn's disease, celiac disease, necrotizing enterocolitis) (14–20). It is also used to measure the hepatic artery resistive index, as a Doppler ultrasonography parameter is used to show microcirculatory resistance in fatty liver, adult alcoholic liver disease, and chronic hepatitis, as well as in in obese children to diagnose insulin resistance and in children with cirrhosis secondary to biliary atresia (21–24).

In the superior mesenteric artery (SMA), both the mean flow velocity and the diastolic velocity were significantly higher and the PI was significantly lower in untreated celiac patients than in treated celiac patients or healthy controls. These data showed that intestinal hyperemia and hyperdynamic mesenteric circulation occurs in celiac disease due to the reduction of resistance (11). Erden et al. found that the mean values of PSV and EDV in the SMA were significantly higher in patients with ileocecal region inflammation (25). Bolondi et al. measured the RI of the SMA in patients with active and inactive Crohn's disease and found a statistically significant difference between values in healthy and acutely ill patients but no difference between patients with active and inactive disease (14). Van Oostayen et al. showed that the Doppler SMA flow was significantly increased in the active Crohn's disease group compared to the inactive patient groups and the control groups (26). In one study, a substantial increase in inferior mesenteric artery flow was found in patients with Crohn's disease or with ulcerative colitis including active inflammation of the left colon (27). Symptomatic patients with gastrointestinal Behçet's disease had a significant increase in mesenteric artery flow (12).

The effect of *H. pylori* on gastric mucosal flow and nonsteroid antiinflammatory drug (NSAID)-induced gastric damage is unclear. Elizalde et al. showed gastric mucosal flow changes in mice with *H. pylori* infection and NSAID-induced gastric injury. They found that gastric blood flow increased 1 week after *H. pylori* infection, but it returned to the basal level by 4 weeks. NSAID-induced gastric damage was not seen at an early phase because of this protective hyperemia (28). In another study, it was reported that *H. pylori* infection does not influence the gastric microcirculation in humans (3). On the other hand, Kalia and Bardhan stated that *H. pylori* produces an inflammatory response leading to vascular insufficiency, ischemic lesions, and ulcers (29). Funatsu et al. showed that the presence of gastric

acid is important in the NSAID-induced decrease in gastric mucosal flow detected by a laser Doppler perfusion image system in rats (30). Laser Doppler flowmetry studies including humans showed that gastric mucosal blood flow was significantly slower in an acute ulcer group than in normal subjects (4). Guslandi et al. also showed that chronic gastritis reduced gastric blood flow in patients with or without *Helicobacter pylori* (3). In one study, a reduction in gastric mucosal perfusion was seen in NSAID-treated patients (31). The study of microvasculature in bioptic specimens of gastric mucosa with chronic gastritis showed disorders of terminal blood flow (32). Atuma et al. stated that a factor or a combination of factors, other than the vacuolating cytotoxin and the immunodominant antigen released from *H. pylori*, might affect the defense of gastric mucosa by reducing mucosal blood flow (33).

Previous studies have reported the potential usefulness of Doppler flow in the splanchnic territory. The authors found that inflammatory processes of the bowel tend to produce changes in SMA or inferior mesenteric artery blood flow parameters, reflecting the hyperemia associated with inflammation. In our study, although there was inflammation of the stomach, the blood flow parameters of the CA did not correlate with the results associated with inflammatory bowel diseases. We found reduced flow velocities of the CA in patients with chronic active gastritis. However, this result correlated with previous studies including gastritis that showed a reduced blood flow using the different methods mentioned above (especially laser Doppler flowmetry done during endoscopy). Although the differences were not statistically significant, the flow in patients with moderate–severe inflammation was lower than in patients with mild inflammation, and the flow in patients with *H. pylori* was also lower than in patients without *H. pylori*. This study is the first evaluating Doppler blood flow of the CA in chronic gastritis and showing decreased flow velocities in the CA.

Erden et al. stated that changes in vascular resistance in ileocolic artery branches can change the waveform of the SMA. In ileocecal inflammation, the vascular bed enlarges, vascular impedance decreases, and blood flow velocities increase (25). In our study, the reduced flow velocities of the CA may be related to the increased vascular resistance in the end organ, the stomach. Because of local adverse effects (increased hydrochloric acid level and/or *H. pylori*) on gastric mucosa, microvascularization was impaired. These changes may alter the waveform of the CA.

Limitation of our study include the small size of the study; the possibility that the controls who had not undergone endoscopy might have had chronic gastritis (however, they were asymptomatic and a decrease in flow would be a symptomatic clue); and the fact that reduction of mucosal blood flow should be confirmed by endoscopic techniques.

In conclusion, Doppler assessment of the CA may be helpful in understanding hemodynamic changes in chronic active gastritis. Whether remission or suspected relapse may be evaluated by adding Doppler assessment of the CA to patient follow-up is a question for future studies, to be performed on a large number of patients without awareness of clinical data by the examiner. In addition, Doppler studies of the CA can further our understanding of the pathophysiology of gastritis.

References

1. Whittle BJR. The defensive role played by gastric microcirculation. Meth Find Exp Clin Pharmacol 1989; 11 (Suppl 1): 35–43.
2. Guslandi M. Mucosal blood flow and gastric protection - effect of neurohormonal and pharmacological agents. Int J Clin Pharmacol Ther Toxicol 1986; 24: 143–7.
3. Guslandi M, Sorghi M, Tittobello A. Does *Helicobacter pylori* affect gastric microcirculation? Ital J Gastroenterol 1994; 26: 383–4.
4. Wang Y, Yuan SY, Zhang ZY. A study of gastric mucosal blood flow of peptic ulcer, chronic gastritis and gastric carcinoma. Zhonhua Nei Ke Za Zhi 1993; 32: 239–42.
5. Öberg PÅ. Laser-Doppler flowmetry. Crit Rev Biomed Eng 1990; 18: 125–63.
6. Michels NA. Blood supply and anatomy of the upper abdominal organs with a descriptive atlas. Philadelphia (PA): Lippincott; 1995. p.139–43.

7. Lezzi R, Cotroneo AR, Giancristofaro D, Santoro M, Storto ML. Multidetector-row CT angiographic imaging of the celiac trunk: anatomy and normal variants. Surg Radiol Anat 2008; 30: 303–10.
8. Karakaya AF, Kantarci M, Yalcin A, Demir B, Yuce İ. A rare variation of hepatic arteries (Michels type IV): MDCT angiographic findings. Eurasian J Med 2009; 41: 63–5.
9. Evans DH, McDicken WN, Skidmore R, Woodcock JP. Doppler ultrasound: physics, instrumentation, and clinical applications. New York: John Wiley and Sons; 1989.
10. McDicken WN. Diagnostic ultrasonics: principles and use of instruments. Edinburgh: Churchill Livingstone; 1991.
11. Arienti V, Califano C, Brusco G, Boriani L, Biagi F, Giulia Sama M et al. Doppler ultrasonographic evaluation splanchnic blood flow in coeliac disease. Gut 1996; 39: 369–73.
12. Sığırcı A, Şenol M, Aydin E, Kutlu R, Alkan A, Altınok MT et al. Doppler waveforms and blood flow parameters of the superior and inferior mesenteric arteries in patients having Behçet disease with and without gastrointestinal symptoms: preliminary data. J Ultrasound Med 2003; 22: 449–57.
13. Yekeler E, Danalioglu A, Movasseghi B, Yilmaz S, Karaca C, Kaymakoglu S et al. Crohn disease activity evaluated by Doppler ultrasonography of the superior mesenteric artery and affected small-bowel segments. J Ultrasound Med 2005; 24: 59–65.
14. Bolondi L, Gaiani S, Brignola C, Campieri M, Rigamonti A, Zironi G et al. Changes in splanchnic hemodynamics in inflammatory bowel disease: non-invasive assessment by Doppler ultrasound flowmetry. Scand J Gastroenterol 1992; 27: 501–7.
15. Maconi G, Imbesi V, Bianchi-Porro G. Doppler ultrasound measurement of intestinal blood flow in inflammatory bowel disease. Scand J Gastroenterol 1996; 31: 590–3.
16. Creteur V, Campinne N, Lambert M, Andre PP, Widelec J, Peetrons P. Contribution of Doppler sonography in inflammatory pathology of the large bowel. J Belge Radiol 1996; 79: 1–8.
17. Van Oostayen JA, Wasser MNJM, van Hegezand RA, Griffioen G, Biemond I, Lamers CB et al. Doppler sonography evaluation of superior mesenteric artery flow to assess Crohn's disease activity: correlation with clinical evaluation, Crohn's disease activity index and α_1-antitrypsin clearance in feces. Am J Roentgenol 1997; 168: 429–33.
18. Sığirci A, Baysal T, Kutlu R, Aladağ M, Saraç K, Harputluoğlu H. Doppler sonography of the inferior and superior mesenteric arteries in ulcerative colitis. J Clin Ultrasound 2001; 29: 130–9.
19. Alvarez D, Vasquez H, Bai JC, Mastai R, Flores D, Boerr L. Superior mesenteric artery blood flow in celiac disease. Dig Dis Sci 1993; 38: 1175–82.
20. Deeg KH, Rupprecht T, Schmidt E. Doppler sonographic detection of increased flow velocities in the celiac trunk and superior mesenteric artery in infants with necrotizing enterocolitis. Pediatr Radiol 1993; 23: 578–82.
21. Pierce ME, Sewell R. Identification of hepatic cirrhosis by duplex Doppler ultrasound value of the hepatic artery resistive index. Australas Radiol 1990; 34: 331–3.
22. Colli A, Cocciolo M, Mumoli N, Cattalini N, Fraquelli M, Conte D. Hepatic artery resistance in alcoholic liver disease. Hepatology 1998; 28: 1182–6.
23. Hızlı Ş, Koçyiğit A, Arslan N, Tuncel S, Demircioğlu F, Çakmakçı H et al. The role of ultrasonographic hepatic artery resistive index in the diagnosis of insulin resistance in obese children with non-alcoholic fatty liver disease. Turk J Med Sci 2010; 40: 335–42.
24. Brodie E, Farrant P, Reid F, Baker A, Meire H, Rela M et al. Hepatic artery resistance index can predict early death in children with biliary atresia. Liver Transpl Surg 1997; 3: 604–10.
25. Erden A, Cumhur T, Olcer T. Superior mesenteric artery Doppler waveform changes in response to inflammation of the ileocecal region. Abdom Imaging 1997; 22: 483–6.
26. Van Oostayen JA, Wasser MNJM, Griffioen G, van Hogezand RA, Lamers CB, de Roos A. Activity of Crohn's disease assessed by measurement of superior mesenteric artery flow with Doppler ultrasound. Neth J Med 1998; 53: S3–8.
27. Mirk P, Palazzoni G, Gimondo P. Doppler sonography of hemodynamic changes of the inferior mesenteric artery in inflammatory bowel disease: preliminary data. Am J Roentgenol 1999; 173: 381–7.
28. Elizalde JI, Mendez A, Gomez J, del Rivero M, Gironella M, Closa D et al. Gastric mucosal blood flow changes in *Helicobacter pylori* infection and NSAID-induced gastric injury. 2003; 8: 124–31.
29. Kalia N, Bardhan KD. Of blood and guts: association between *Helicobacter pylori* and the gastric microcirculation. J Gastroenterol Hepatol 2003; 9: 1010–7.
30. Funatsu T, Chono K, Hirata T, Keto Y, Kimoto A, Sasamata M. Mucosal acid gastric mucosal microcirculatory disturbance in nonsteroidal anti-inflammatory drug-treated rats. European J Pharmacol 2007; 554: 53–9.
31. Guslandi M, Foppa L, Fanti L, Sorghi M. Nonsteroidal anti-inflammatory drugs and gastric mucosal blood flow. J Clin Gastroenterol 1999; 28: 258–9.
32. Miller DA, Chernin VV, Tkacher VA, Matiash BL. Microcirculation in patients with chronic gastritis depending on its exacerbation severity and morphological type. Eksp Klin Gastroenterol 2002; 4: 14–7.
33. Atuma C, Engstrand L, Holm L. Extracts of *Helicobacter pylori* reduce gastric mucosal blood flow through a VacA- and CagA-independent pathway in rats. Scand J Gastroenterol 1998; 33: 1256–61.