Postprandial Evaluation of Possible Collateral Pathways in Chronic Mesenteric Ischemia with Duplex Ultrasound

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Abstract

Objective: Duplex scanning may be used to detect high-grade stenoses in the mesenteric vessels in patients with suspect Chronic Mesenterial Ischemia (CMI). The aim of the study was to evaluate reserve and collateral vascular function in relation to bowel symptoms in patients with CMI.

Methods: Fourteen consecutive patients, referred to the vascular laboratory with suspicion of CMI, 7 men (average 79 years), and 7 women (average 66 years), were investigated by duplex ultrasound (DUS) of the visceral arteries. All patients were examined both fasting and 30 min after a standard meal. A reference group of 10 patients with diffuse bowel symptoms (5 men and 5 women, average 75 years) were investigated by the same DUS protocol, all showing normal mesenteric arteries. Possible collateral reserve capacity was defined as a marked increase of flow velocities (>20%) in vessels with lesions and/or in non-affected vessels.

Results: All patients had a significant stenosis (>70%) in one or several visceral arteries at the baseline DUS investigation. Six patients had abdominal pain after the test meal, whilst 8 patients experienced no pain. In patients without postprandial pain a possible collateral reserve capacity was detected. In this group superior mesenteric artery (SMA) peak systolic velocity (PSV) increased significantly (baseline PSV 2.6 ± 1.3 m/s vs. postprandial PSV 4.1 ± 1.6 m/s, P < 0.05) whereas patients with postprandial pain showed no signs of collateral reserve capacity in any vessel except one patient who showed possible collateral reserve in one vessel. In the reference group a significant increase of PSV was seen in SMA only, (1.7 ± 0.6, vs. 2.5 ± 0.9, P < 0.05).

Conclusion: Postprandial duplex adds information about the functional flow limitation of the stenosis and possible collateral reserve flow capacity. Further studies have to be performed to establish criteria for selection of patients appropriate for intervention.

Keywords: Chronic mesenteric ischemia; Duplex ultrasound; Postprandial duplex ultrasound

Introduction

Duplex scanning may be utilised to detect high-grade stenoses in the mesenteric vessels in patients with suspect vascular abdominal angina. Flow velocity criteria have been established concerning the Coeliac Artery (CA), Superior Mesenteric Artery (SMA) and the Inferior Mesenteric Artery (IMA) to detect high grade stenoses [1-3].

The finding of visceral artery high-grade stenosis, however, does not always correlate with the clinical entity of chronic mesenteric ischemia and one reason may be the presence of individual collateral capacity.

Chronic Mesenteric Ischemia (CMI), also called postprandial abdominal angina, was first described by Councilman in 1894 and seems to be a rare disorder, accounting for less than 0.01% of hospital admissions in the US [1,4]. The mortality is relatively high due to delayed diagnosis or with the disease presenting late, when severe bowel ischemia already is present [5,6]. Atherosclerosis (AS) is the main cause of CMI; rarely fibromuscular dysplasia, artery dissection or inflammatory artery disease such as Takayasu’s disease may be detected. In more than 70% of the patients; AS seems to be disseminated. Symptoms may occur late when stenosis or occlusion of one or several of the mesenteric arteries occurs, usually the SMA but the CA and the IMA may also be involved. In typical patients abdominal pain is presented after food intake, from 15-30 min after meal, persisting as long as 5-6 hours. The composition of the meal may influence the duration and quality of the bowel symptoms, and the symptoms may lead to cibophobia (fear for food) and weight loss in as many as 90% of patients with abdominal angina [7].

The patients usually are elderly with female preponderance, and a history of smoking may be an important factor. Catheter based angiography has been considered the reference for the diagnosis of stenosis of the visceral arteries to visualise and measure flow and assess collaterals.

Advances in non-invasive tests include Computed Tomography Angiography (CTA) and Magnetic Resonance Angiography (MRA) where the latter also includes possibilities for quantification of blood flow [8-10].
Radiological methods, however, are not commonly used to assess data about the reserve capacity of collateral pathways and thus assess the functional flow limitation of the stenosis of the mesenteric arteries. The definition of degree of visceral stenosis and collateral capacity inducing clinical symptoms at CMI is still a controversial issue [5,11].

The use of Duplex scanning was first described in the mid 1980’s and may be used as a screening method to detect the presence of mesenteric stenosis, both on the 2D image and with Doppler flow detection [2,3,8].

The Doppler may be used both to measure velocities in stenoses, to evaluate flow patterns in different vessels, and to evaluate the effects of symptom provocation, for example a meal [12]. In this study we performed duplex scanning on the three main mesenteric vessels before and after food intake, with the intention to evaluate collateral capacity of both stenotic and non-stenotic vessels.

The hypothesis was that postprandial Duplex might give additional information to the fasting duplex scanning concerning the hemodynamic significance of the visceral stenoses.

Material and Methods

Fourteen patients, referred to the vascular laboratory, with suspicion of CMI, agreed to take part in the study. There were 7 men (average 79 years, range 62-89), and 7 women (average 66 years, range 32-81). Other signs of cardiovascular disease was present in at least 64% of the patients, and 50% were smokers or ex-smokers. The cause of the detected mesenteric stenosis was probably AS in 12/14 patients due to the presence of plaques by ultrasound (Table 1).

Table 1: Demographic values and site of disease in the patient group of CMI (n=14)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Symptoms</th>
<th>Weight loss</th>
<th>Test symptoms</th>
<th>Site of disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>F</td>
<td>Severe</td>
<td>7 kg</td>
<td>No pain</td>
<td>CA</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>F</td>
<td>Moderate</td>
<td>None</td>
<td>No pain</td>
<td>CA, SMA</td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>F</td>
<td>Severe</td>
<td>30 kg</td>
<td>pain, illness</td>
<td>CA, SMA, IMA</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>F</td>
<td>Severe</td>
<td>Slight</td>
<td>No pain</td>
<td>CA</td>
</tr>
<tr>
<td>5</td>
<td>77</td>
<td>M</td>
<td>Moderate</td>
<td>5 kg</td>
<td>pain, illness</td>
<td>CA, IMA lig</td>
</tr>
<tr>
<td>6</td>
<td>74</td>
<td>F</td>
<td>Severe</td>
<td>Slight</td>
<td>No pain</td>
<td>SMA occl, IMA</td>
</tr>
<tr>
<td>7</td>
<td>76</td>
<td>F</td>
<td>Moderate</td>
<td>Slight</td>
<td>Pain</td>
<td>CA, SMA occl, IMA</td>
</tr>
<tr>
<td>8</td>
<td>75</td>
<td>M</td>
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<td>No</td>
<td>No pain</td>
<td>CA, SMA</td>
</tr>
<tr>
<td>9</td>
<td>60</td>
<td>M</td>
<td>Severe</td>
<td>A lot</td>
<td>No pain</td>
<td>CA, SMA, IMA</td>
</tr>
<tr>
<td>10</td>
<td>88</td>
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<td>not reported</td>
<td>Slight</td>
<td>CA</td>
</tr>
<tr>
<td>11</td>
<td>83</td>
<td>M</td>
<td>Moderate</td>
<td>No</td>
<td>No pain</td>
<td>CA, SMA occl, IMA</td>
</tr>
<tr>
<td>12</td>
<td>77</td>
<td>M</td>
<td>Moderate</td>
<td>No</td>
<td>Pain</td>
<td>IMA</td>
</tr>
<tr>
<td>13</td>
<td>63</td>
<td>F</td>
<td>Moderate</td>
<td>Slight</td>
<td>pain, illness</td>
<td>IMA, SMA</td>
</tr>
<tr>
<td>14</td>
<td>80</td>
<td>M</td>
<td>Severe</td>
<td>not reported</td>
<td>No pain</td>
<td>IMA</td>
</tr>
</tbody>
</table>

The reference group (5 men and 5 women, average 75 years, range 68-82), had a final diagnosis of gastritis/reflux and/or irritable bowel syndrome. The reference group had normal mesenteric vessels at the Duplex examination on 2D image and normal Doppler velocities.

All individuals were examined in the prone position both fasting and 30 min after a standard meal, consisting of 600 ml of a high-caloric drink containing 900 kcal. All investigations were performed by the same experienced technician having a variation coefficient of <10% in repeated studies.

Blood flow velocities were measured in the CA, SMA and IMA (proximal, mid and distal part of the main vessel). Flow velocities were also measured in branches of the vessels as well as collaterals if identified. In CA flow velocities were measured including the bifurcation of hepatic and ilineal artery.

The Doppler angle was adjusted in the flow direction, normally with minimal angles towards the Doppler beam. The investigation was performed both during inspiration and expiration and the PSV value of expiration was used for comparison. Care was taken that the examination was performed at the same site and the same Doppler angle in all examinations in the individual patient. If possible, flow patterns were studied in the internal iliac artery as well, to evaluate if flow changes might be present after caloric load. Possible reserve capacity was defined as a large vessel (>1.0 cm), more easily detected than normal, and detected increase in flow velocities (>20%).

Possible collateral reserve capacity in the mesenteric arteries was defined as significant increase of flow velocities (>20%) in either vessels with lesions and/or in non-affected vessels.

Duplex findings of the visceral arteries were also correlated to bowel symptoms after the meal. In the study an ACUSON S2000 TM ultrasound system (Siemens Medical Solutions USA, Inc) was used,
and the scans were performed with a semi-linear (curved) 5-2 MHz transducer. The “Portland criteria” for >70% stenosis were used for fasting peak systolic velocities (PSV) i.e. PSV of >2.75 m/s in SMA and >2.0 m/s in CA [1,13]. IMA stenosis was defined as a PSV >2.0 m/s [3].

The reference for detecting mesenteric artery stenosis was the final diagnosis of CMI verified by Computed Tomography (CT)/intervention including a sub group of patients who underwent intervention of the mesenteric stenoses.

Statistical analysis

Data were analysed using Statview® (ver. 5.01, SAS Institute Inc., UK). Values are presented as mean ± SD and Statistical significance was set to P<0.05. Wilcoxon signed rank test was used for paired comparisons and the Mann & Whitney test for unpaired comparisons.

Results

As seen in table 1 multi vessel disease, defined as stenosis or occlusion in 2 or 3 vessels (CA, SMA or IMA), was found in 9 patients. IMA was ligated in one of these patients and in 5 patients single vessel disease was found. All patients had a significant stenosis (>70%) in one or several visceral arteries at the baseline DUS investigation. Twenty-six significant stenosis or occlusions were found whereas all stenosis were located in the proximal part of the vessel.

Six patients had postprandial abdominal pain, whilst 8 patients experienced no pain. In SMA and IMA significant flow velocity increase was seen postprandially in the whole group of patients (baseline PSV in SMA 2.2 ± 1.1 m/s, vs. postprandial PSV 3.2 ± 1.8 m/s, P<0.05, and in IMA (3.7 ± 2.3, vs. 4.3 ± 2.3, P<0.05).

In the group of patients without pain (n=8); two patients had 3-vessel disease, 3 patients had 2-vessel disease and 3 patients had 1-vessel disease. In this group possible reserve capacity (PSV increase >20%) was seen in either two vessels (n=6) or in one vessel (n=2).

Patients with collateral reserve capacity in only one vessel had signs of possible collateral flow in the internal iliac artery.

In the group of patients with postprandial pain (n=6); two patients had 3-vessel disease, two patients had 2-vessel disease and 2 patients had 1-vessel disease. In this group four patients with 1-2 or 3-vessel disease had no flow velocity increase in any vessel.

One patient with 3-vessel disease had slight flow velocity increase (<20%) in only one vessel indicating the absence of significant collateral reserve capacity. One patient with stenosis in CA and a ligated IMA, who experienced pain immediately after the meal, had a >20% postprandial increase of flow velocity in SMA, indicating possible collateral reserve capacity.

As seen in table 2 possible collateral reserve capacity was seen in patients without postprandial pain in SMA, (baseline PSV 2.6 ± 1.3, vs. postprandial PSV 4.1 ± 1.6 m/s, P<0.05) whereas patients with postprandial pain showed no collateral reserve capacity in SMA, (baseline PSV 1.8 ± 1.0, vs. postprandial PSV 2.0 ± 1.2 m/s, NS). In the reference group collateral reserve capacity was seen in SMA only (PSV 1.7 ± 0.6, vs. 2.5 ± 0.9, P<0.05). The flow velocity increase postprandially in SMA was significantly higher in the reference group compared to the patient group with postprandial pain (P<0.05). A similar but lower velocity increase was detected in CA and IMA (change of PSV in patients without pain vs. those with pain) (0.7 ± 0.9 vs. 0.2±0.7 m/sec and 0.6 ± 0.5 vs. 0.1 ± -0.3 m/sec, both NS).

<table>
<thead>
<tr>
<th></th>
<th>PSV</th>
<th>change</th>
<th>EDV</th>
<th>change</th>
</tr>
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<tbody>
<tr>
<td>CA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>3.2±1.2</td>
<td>0.5±0.8</td>
<td>1.0±0.8</td>
<td>0.2±0.5</td>
</tr>
<tr>
<td>Pain</td>
<td>3.3±1.6</td>
<td>0.2±0.7</td>
<td>1.1±1.1</td>
<td>0.6±0.6</td>
</tr>
<tr>
<td>No pain</td>
<td>3.1±0.7</td>
<td>0.7±0.9</td>
<td>0.9±0.6</td>
<td>-0.1±0.4</td>
</tr>
<tr>
<td>Ref group</td>
<td>1.5±0.7</td>
<td>0.0±0.4</td>
<td>0.3±0.2</td>
<td>0.0±0.1</td>
</tr>
<tr>
<td>SMA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>2.2±1.1</td>
<td>0.9±1.1</td>
<td>0.3±0.3</td>
<td>0.6±0.6</td>
</tr>
<tr>
<td>Pain</td>
<td>1.8±1.0</td>
<td>0.3±0.4</td>
<td>0.3±0.2</td>
<td>0.2±0.2</td>
</tr>
<tr>
<td>No pain</td>
<td>2.6±1.3</td>
<td>1.5±1.3*</td>
<td>0.3±0.3</td>
<td>1.0±0.7</td>
</tr>
<tr>
<td>Ref group</td>
<td>1.7±0.6</td>
<td>0.8±0.4*</td>
<td>0.2±0.1</td>
<td>0.3±0.3</td>
</tr>
<tr>
<td>IMA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>3.7±2.3</td>
<td>0.6±0.5*</td>
<td>0.6±0.8</td>
<td>-0.1±0.3</td>
</tr>
<tr>
<td>Pain</td>
<td>3.9±1.8</td>
<td>0.1±-0.3</td>
<td>0.5±0.6</td>
<td>0.0±0.3</td>
</tr>
<tr>
<td>No pain</td>
<td>3.5±2.7</td>
<td>0.6±0.5</td>
<td>0.8±1.0</td>
<td>-0.2±0.4</td>
</tr>
<tr>
<td>Ref group</td>
<td>1.5±0.6</td>
<td>0.1±-0.3</td>
<td>0.1±0.1</td>
<td>0.2±0.4</td>
</tr>
</tbody>
</table>

Table 2: Peak Systolic and End-Diastolic Velocities (PSV, EDV) in Coeliac Artery (CA), Superior Mesenteric Artery (SMA) and Inferior Mesenteric Artery (IMA) before and change after a test meal

The normal reference group had a significant velocity increase in SMA only (1.7 ± 0.6 change 0.8 ± 0.4, P<0.05), and minimal changes in CA (0.0 ± 0.4 m/sec and IMA (0.1±-0.3 m/sec, NS). Diastolic velocities did not change significantly in any of the studied groups.

Figure 1a: Plaque in proximal coeliac artery (CA) (Panel A=left) and a high grade stenosis in proximal superior mesenteric artery (SMA) (Panel B=right). Figure 1b: Normal arteries in one of the controls in the reference group.
Figure 1 shows a typical patient with possible collateral reserve capacity and no postprandial pain despite a 3-vessel disease. This pictures show plaque and stenosis in CA and SMA (PSV in CA: 2.6 m/sec and no flow velocity increase after the test meal). A high grade stenosis is shown in SMA (PSV in SMA: 3.9 m/s, and 5.5 m/s after the test meal). Pictures of IMA are not shown. (PSV in IMA: 4.0 m/s and 5.0 m/s after the test meal). Thus the presence of a significant flow velocity increase in SMA and IMA, despite high grade stenosis in both vessels, indicates possible collateral reserve capacity. Computed tomography (CT) in this patient showed a 70% stenosis in CA, a 90% stenosis in SMA and a 60% stenosis in IMA.

Open collateral pathways were detected; ( gastroduodenal collateral to SMA as well as open collateral of Reolans; SMA to IMA).

Discussion

This study was performed to investigate changes of flow velocity in the main mesenteric arteries after a meal load, in patients with chronic mesenteric ischemia (CMI). All patients had duplex-verified stenosis or occlusion in at least one of the mesenteric arteries, and most patients had multi vessel disease. The caloric load resulted in typical gastric symptoms in almost half of the CMI patients, in whom the peak systolic velocity (PSV) after caloric load did not increase significantly in SMA compared to baseline values, whereas a significant increase of PSV was detected in SMA in the group of patients without postprandial pain. The flow velocity increase in SMA was also significantly higher in the reference group with normal mesenteric arteries compared to the patient group with postprandial pain.

Thus it seems that the abdominal vessels may have an important reserve flow capacity in spite of the presence of high grade stenosis in several vessels, in patients with clinical CMI. This is probably explained by the presence of arterial communications in the whole mesenterical territory.

In patients with postprandial abdominal angina, obliterative changes in the mesenteric arteries are often diagnosed, but there are problems to identify patients who are at risk and could benefit from therapeutic interventions. Collaterals can be visualised by DUS and other radiological methods but the reserve capacity is not possible to assess without performing postprandial studies.

Therefore it seems important to add a functional evaluation of the mesenteric blood flow. Duplex ultrasound examination (DUS) on the issue of CMI may be used in most patients, as it is non-invasive and free of complications. The method seems suitable both for screening and follow up studies [14,15]. Velocity criteria for at least 70% stenosis for all the large mesenteric vessels are well established in comparison to angiography [3,13,16].

However the value of postprandial provocation has not been fully evaluated. In two studies [12,17] the effect on stenosis detection was evaluated but no real improvement was found. The effect on the collateral capacity was not studied. Coeliac studies are considered not suitable for food challenge [1]. Using our method based on ultrasound Doppler it seems, however, that the abdominal vessels may have an important reserve flow capacity even in multi vessel mesenteric disease. We found that patients with possible collateral reserve capacity as indicated by a substantial increase of flow velocities (>20% from baseline velocity) may have a less marked symptoms, in spite of high grade stenoses.

Collateral pathways include connections between CA and SMA, (i.e. the gastroduodenal and pancreatico-duodenal arcade) or between SMA and IMA (i.e. the meandering artery and Marginal artery of Drummond) and between IMA and internal iliac artery (i.e. the hemorrhoidal branches) [18].

Postprandial symptoms may be experienced as abdominal cramping, possibly due to increased tension in the abdominal musculature and/or bowel smooth muscles, due to mesenteric ischemia.

In general, arterial changes are caused by atherosclerosis, but other explanations to bowel syndromes exist such as, fibromuscular dysplasia, inflammatory vessel disease, systemic amyloidosis and carcinoid. Non-vascular causes are for example irritable bowel syndrome and biliary troubles.

Another possible mechanism, that could mimick atherosclerotic disease of the mesenteric arteries, is the artery compression syndrome caused by an arcuate ligament limiting flow after meal and usually affects the coeliac trunk [19]. Atypical postprandial troubles may be explained for example by a vascular steal from the small bowel to the gastric circulation. One of the patients, who experienced pain immediately after the meal, had a high grade coeliac stenosis and a ligated IMA and developed a marked postprandial increase of flow velocity in SMA. This could probably in part be explained by steal to the ventricular area.

When CMI is suspected, radiological methods may be used for diagnostic purposes. Both Computed Tomography (CT) and magnetic resonance imaging (MR) and conventional invasive contrast arteriography may be used [8,9] including also functional testing by MR for quantification of blood flow [10]. Other non-invasive methods are under development to detect gut ischemia such as small bowel tonometry [20].

Duplex ultrasound, however, is often preferred as an initial investigation as it is cheap, non-invasive and without risks, and shows good diagnostic results [1]. The method may be used both for screening of the mesenteric stenoses and for signs of collateral flow patterns, indicating disturbed mesenteric flow. Additionally, as this study shows, a simple caloric load test may help to evaluate collateral flow capacity and thus may be a tool to help in determining which patients that might have an advantage of therapeutic interventions.

A gold standard of the functional flow limitation of the stenosis as well as collateral reserve capacity is difficult to assess with radiological methods. MR perfusion imaging could be used for quantification of visceral blood flow but was not used in this study. In this study the reference for detecting mesenteric artery stenosis was the final diagnosis of CMI verified by CT/intervention including a sub group of patients who underwent intervention of the mesenteric stenoses. CMI is a rare disease and few patients are referred for Duplex Investigation of the mesenteric arteries. The consecutive series of patients in this article include the clinical patient material during four years.

The mortality of CMI is relatively high if delayed diagnosis and intervention should be considered. It is important to note that five patients (2 or 3-vessel disease in 4 of the patients), who were not considered for surgery, died within three years after the Duplex investigation of acute mesenteric ischemia/stroke. Three of these patients had no collateral reserve capacity in any vessel and 2 patients had reserve capacity in only one vessel as judged by DUS.
However the risk for acute mesenteric ischemia could be hard to predict in individual cases but poor collateral capacity might be one factor to be taken in consideration for intervention.

We used established criteria to diagnose high grade stenosis, using maximal baseline velocities to identify >70-80% stenosis. At this level basal flow is considered to diminish, or have minor possibility to increase at demand. By DUS differences in peak systolic as well as end diastolic flow velocities during inspiration and expiration are expected whereas higher velocities are seen during expiration [21]. In addition it is important to be aware of expected differences between functional and anatomical measurements. An important factor for flow and flow velocity is the collateral back pressure. A high back pressure may diminish forward flow and thus the maximal flow velocity may decrease [22,23]. The presence of marked collateral circulation from for example SMA through the gastroduodenal arcade to CA, thus could diminish flow velocities in CA and therefore a high grade CA stenosis could be difficult to detect using flow velocity criteria [16,24]. In addition major calcifications may corrupt both the image and flow detection.

Limitations of the Study

CT was performed only in patients where intervention was possible, including 13 patients in this study. Because of incomplete CT data and variable CT-protocols, systematic comparison between CT and Duplex ultrasound could not be performed. Interestingly, however, CT showed open collaterals in 5/8 patients with no pain after food challenge and no visible collaterals in 4/6 patients with pain. Two patients in this latter group, however, showed collaterals by CT despite Duplex findings of a poor collateral reserve capacity. The conclusion might be that an anatomical assessment of collateral does not tell whether they are functional.

Conclusion

Postprandial duplex adds information about the functional flow limitation of the stenosis and possible collateral reserve flow capacity. Further studies have to be performed to establish criteria for selection of patients appropriate for intervention.

References