# Is Portal Hypertension due to Liver Cirrhosis a Major Factor in the Development of Portal Hypertensive Gastropathy?

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***Özet:*** *KARACİĞER SİROZU NEDENİYLE GELİŞMİŞ PORTAL HİPERTANSİYON PORTAL HİPERTANSİF GASTROPATİ GELİŞMESİNDE ÖNEMLİ BİR FAKTÖRMÜDÜR?*

*Portal hipertansiyonu olan hastaların midelerinde endoskopiyle tespit edilebilecek lezyonlar sıkça gözlenir. Önemli kanama odakları olan bu lezyonlar mukozadaki bazı bozukluklara bağlı olarak gelişir. Çoğu araştırmacı portal hipertansif gastropati (PHG) gelişiminde portal hipertansiyonun önemli etiopatojenik rolü olduğuna inanır, anak kesin sebep veya sebepler belli değildir. Bu sebepleri orataya çıkarmak için, dört değişik hasta grubu prospektif olarak araştırıldı. Karaciğer sirozuna bağlı portal hipertansiyonu olan 37 hasta, portal ven obstrüksiyonu (PVO) nedeniyle portal hipertansiyonu olan 26 hasta, sirozu ve ek olarak PVO olan 9 hasta, ve 57 kişiden oluşan kontrol grubu araştırma grubunu oluşturdu. Sirotik hastalarda yılan derisi, skarlatina döküntüsü, hiperemi, ve peteşiler en sık görülen lezyonlardı. Bu lezyonlar PVO nedeniyle portal hipertansiyon gelişmiş hastalarda daha az gözlenirken, sirotik grupta genellikle birlikte görülmeleri dikkati çekti. Sirotik hastalarda PHG insidansı PVO hastalarına göre daha fazlaydı ($p < 0.0001$). Bu lezyonların en sık görüldüğü grup siroz ve PVO olan gruptu ($p < 0.0001$). Endoskopik bulgular, sirozun derecesi (Child's-Pugh sınıflaması) ve özefagus varislerinin büyüklüğü (Beppu skoru) arasınada korrelasyon gözlendi. Gastrik mukozada karakteristik inflamatuar değişiklik saptanmadı. Sirotik hastalarda genellikle hipergastrinemi tespit edilirken, PVO nedeniyle portal hipertansiyon gelişen grupta hiçbir vakada hipergastrinemi saptanmadı.*

*Sonuçlar PHG'nin önemli ölçüde karaciğer hastalığının şiddetinden, portal hipertansiyonun etiolojisinden ve birlikte görülen PVO dan etkilendiğini, ancak gastrik varis olup olmamasıyla ilişkili olmadığını gösterdi. Gastroözefajeal lezyonların gelişmesinde sadece portal hipertansiyonun değil, ayrıca kronik parankimal karaciğer hastalığının da önemli bir faktör olduğu belirlendi.*

**Anahtar kelimeler:** Portal hipertansiyon, portal ven obstrüksiyonu, portal gastropati, karaciğer sirozu, özefagus varisi, gastrik varis.

***Summary:*** *The stomachs of patients with portal hypertension are frequently subjected to a number of alterations visible by endoscopy, the presence of visible lesions which are important sources of bleeding indicates a disturbance in the mucosa. Although the majority believe that portal hypertension plays an etiopathogenetic role in the development of portal hypertensive gastropathy (PHG), the factors that influence it are not clearly understood. To investigate these, four groups of subjects were studied prospectively. 37 patients with portal hypertantion due to liver cirrhosis, 26 patients with portal hypertension due to portal vein obstruction (PVO), 9 cirrhotic patients with PVO, and 57 control subjects. Snake skin, scarlatina rash, hyperemia, and petechia were the most frequent endoscopic finding in cirrhotic patients. These findings were less frequent in patients with portal hypertension due to PVO and were most frequently present in association with each other in cirrhotic groups. The incidence of PHG was higher in cirrhotic patients than in PVO patients ($p<0.0001$). The highest incidence was in cirrhotic patients with PVO ($p<0.0001$). There was a correlation between the endoscopic findings, the clinical gravity of liver cirrhosis (Child-Pugh grade), and the severity of esophageal varices (Beppu score). There were no charecteristic inflammatory findings in the gastric mucosa. Hypergastrinemia was often observed in cirrhotic patients but never in patients with portal hypertension resulting from PVO.*

*The results suggested that PHG is significantly affected by the severity of liver disease, etiology of portal hypertension and coexisting PVO, and is not correlated with coexisting gastric varices. Developement of the gastroesophageal lesions requires not only portal hypertention but also chronic paranchymal liver disase.*

**Key words:** Portal hypertension, portal vein obstruction, portal gastropathy, liver cirrhosis, esophageal varices, gastric varices.

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The stomachs and esophagus of patients with portal hypertension regardless of etiology are frequently subject to a number of alterations, detectable by endoscopy, which indicate a disturbance in the mucosa. In addition to variceal bleeding from the stomach and the esophagus, which is a well-known manifestation in cirrhotic patients that may precipitate the clinical condition, gastric mucosal lesions are an important cause of upper gastrointestinal hemorrhage. These mucosal lesions have been variously called erosive gastritis (1,2), congestive gastropathy (3), mosaic pattern, and portal hypertensive gastropathy (4). Less is known about the factors responsible for these manifestation, although several workers believe that portal hypertension has an etiopathogenetic role (4,5). The incidence and profile of portal hypertensive gastropathy has been variably reported in different studies, ranging from 100% mild to severe gastropathy (4) to 50% of the patients. In the literature most studies are about portal hypertension due to liver cirrhosis, but few studies comparing the results obtained from non-cirhotic portal hypertension with cirrhotic portal hypertension cases have carried out (6). The aim of this study is to determine whether the severity of liver disease, etiology of portal hypertension and the presence of gastric varices have a role in the occurence of PHG, to determine whether there is a link between the endoscopic condition of the mucosa and the degree of severity of the varices; and to compare the morphologic alteration present at the level of the gastric and esophageal mucosa in the four groups. In addition, we compared the levels of fasting serum gastrin in four groups.

## MATERIALS and METHODS

The study included 72 patients and 57 controls presenting at the Hacettepe University Hospital between January 1987 and June 1994. The patients were divided into four groups. Group 1,37 patients (23 male, female 14; mean age 43 years) with portal hypertension due to liver cirrhosis caused by mostly viral infections; group 2, 26 patients (15 male, 11 female, mean age 37 years) with portal hypertension due to portal vein obstruction; group 3,9 patients (3 female, 6 male, mean age 40 years) with liver cirhosis complicated with portal venous obstruction among 457 cirrhotic patients diagnosed at the same period, group 4(control group), 57 patients (26 female, 31 male, mean age 42 years) who complained of various upper abdominal symptoms but had no history of peptic ulcers. Discomfort, fullness, belching, burning and bloating were the basic symptoms of the control group. They had no symptoms characteristic of either biliary colic or typical heailburn and did not report weight loss, severe systemic illness, peptic ulcer complications or multisystem diseases, and their general conditions were good. Before endoscopy (esophagogastroduodenoscopy, EGD), the clinical, laboratory, and instrumental tests demrastrate neither significant illness as evidence of hepatic pathology nor signs of portal hypertension. The EGD was performed to define dyspepsia precisely. Table I shows the endoscopic findings of the patients. At the time of entry into the study no patient in any group had remarkable signs or symptoms of bleeding from the gastroentistinal system. None of the patients were on any drugs that might damage the gastric mucosa. All patients were subjected to EGD with an optical-fiber endoscope (Olimpus models Q10, Q20). No clinical or anamnestic information was given to the endoscopist before the examination.

The diagnosis of liver cirrhosis was always based on clinical examination, laboratory tests and liver biopsy. The presence of portal hypertension was confirmed using the clinical and instrumental criteria proposed by Pagliaro et al (7): 1) splenomegaly (>13 cm of longitudinal axis on ultrasonography (US), 2) thrombocytopenia (<100 000/μL); 3) leucopenia (<40 000/ μL 4) portal vein larger than 14 mm in diameter; 5) esophageal varices at endoscopy. Other signs of portal hypertension are the presence of ascites and gastric varices. All patient groups except for controls had esophageal varices and portal vein enlargement, greater than 14 mm on ultrasonographic examination if portal vein was patent, and some of them had more than two these indicators. Wedged hepatic venous pressure and portal vein pressures were not measured. In all groups, the degree of severity of the esophageal varices was expressed in terms of Beppu's score (8). We paid extra attention to the following five discriminant categories: 1) basic color of the varices (white or blue); 2) red color sign (positive or

**Table I:** Frequency of Endoscopic Findings in the Stomach, Duodenum and Esophagus.

| Findings | Portal Hypertension Cirrhosis(C) n: 37(%) | CTPV n: 26(%) | C+CTPV n: 9(%) | Control n: 57(%) |
|---|---|---|---|---|
| Duodenum | | | | |
| Ulcer | 8 (21.6) | 3 (11.6) | 1 (11.1) | - |
| Varices | 1 (2.7) | 4 (15.4) | 2 (22.2) | - |
| Duodenitis | 10 (27) | 5 (19.2) | 3 (33.3) | 6 (10.5) |
| Stomach | | | | |
| Ulcer | 3 (8.1) | 1 (3.8) | 1 (11.1) | - |
| Snake skin | 21 (56.8) | 4 (15.4) | 8 (88.9) | 1 (1.8) |
| Scarlatina rash | 13 35.1 | 5 (19.2) | 7 (77.8) | - |
| Petechia | 14 37.8 | 4 (15.4) | 9 (100) | 3 (5.3) |
| Hyperemia | 20 54 | 4 (15.4) | 7 (77.8) | 2 (3.5) |
| Fundic Varices | 8 21.6 | 12 (46.2) | 9 (100) | - |
| Esophagus | | | | |
| Esophageal Varices | | | | |
| Forms | | | | |
| F1 | 7 18.9 | 1 (3.8) | - | - |
| F2 | 23 61.1 | 5 (19.2) | - | - |
| F3 | 7 18.9 | 20 (76.9) | 9 (100) | - |
| Extent | | | | |
| L1 (Lower third) | 6 16.2 | 1 (3.8) | - | - |
| L2 (Lower two thirds) | 24 64.9 | 3 (11.6) | - | - |
| L3 (Full extend) | 6 16.2 | 21 (80.8) | 9 (100) | - |
| Color | | | | |
| Blue | 29 78.4 | 19 (73.1) | 5 (55.6) | - |
| White | 8 21.6 | 7 (26.9) | 4 (44.4) | - |
| Red signs | | | | |
| Cherry red spot | 15 40.5 | 2 (7.7) | 8 (88.9) | - |
| Haematocytic spot | 13 35.1 | 1 (3.8) | 7 (77.8) | - |
| Diffuse redness | 11 29.7 | 1 (3.8) | 6 (66.7) | - |
| Esophagitis | 9 24.3 | 2 (7.7) | 4 (44.4) | 3 (5.3) |

F1: varices flattened by insufflation, F2: varices not flattened by insufflation, separated by areas of normal mucosa, F4: confluent varices not flattened by inssufflation.

negative), including red wale marking, cherry red spots, hematocystic spots, and/or diffuse redness; 3) forms of the varices (F1. straight varices; F2, enlarged tortuous varices, F3, very large varices,); 4) Location (locus inferior or lower third of esophagus, L1; medialis or lower two thirds, L2; and/or superior of full extent, L3); and 5) esophagitis (present or absent).

During the same period, in 44 patients (27 male, 15 female mean age 36 years), obstruction of the portal vein was diagnosed by ultrasonography. We prospectively studied these patients. To confirm the diagnosis, either splenoportography or arterial portography with digital subtraction angiography (DSA) was performed on all 44 patients, by using a Phillips DVI device. DSA of the superior mesenteric artery was obtained after selection injection of 35 ml of 50% diluted contrast medium at a rate of 8 ml/s. Digital images were recorded for 25 s at a rate of one frame/s during the arterial, arteriocapillar and venous phases. Sonography guided liver biopsy was done in 38 of the 44 patients with CTPV. In addition to routine blood and urine analyses, serial liver function tests and tumor markers were studied. We included in this study only 26 patients(15 male, 11 female, mean age 37 yrs) with PVCT in whom the liver was normal not only on histologic examination but also in liver function tests and despite full investigation, we could not find any etiologic factor for portal vein thrombosis in these 26 patients. In 9 of the 44 patients with CTPV, there was liver cirhosis. We estimated these patients as a different group and we compared the endoscopic findings of these patients with those of patients with portal hypertension due to liver cirrhosis without portal involvement. In the remining 9 patients whom we excluded from the study there were other pathologies in addition to portal vein involvement.

**Table II:** Characteristics of Patients and Control Subjects.

| | Portal hypertension due to C | Portal hypertension due to CTPV | Portal hypertension Control due to C& CTPV | |
|---|---|---|---|---|
| No. of patients | 37 | 26 | 9 | 57 |
| Male | 23 | 11 | 6 | 31 |
| Female | 14 | 15 | 3 | 26 |
| Mean age(yr) | 43 | 37 | 40 | 42 |
| Cause of cirrhosis(n) | | | | |
| Hepatitis B virus | 23 | - | 7 | |
| Hepatitis C virus | 9 | - | 2 | - |
| Primary biliary cirhosis | 1 | - | - | - |
| Cryptogenic | 4 | - | - | - |
| Child's grade(n) | | | | |
| B | 21 | - | 2 | - |
| C | 16 | - | 7 | - |

Endoscopic definitions

We followed the terminology of the World Society of Digestive Endoscopy (9); When the presence of various signs created complex endoscopic findings requiring very detailed descriptions, the following description were used: confluent measles rash: hyperemic areas of differing sizes, of irregular form, and raised above the surrounding mucosa snake skin (10): fine, white reticular pattern with different shaped areas of red edematous musoa; scarlatina rash: a fine, pink speckling that appeared to be below the surface of the mucosa. We paid extra attention in the evaluation of the duodenal mucosa. We graded the duodenal appearance from I to grade 4.

Biopsies

In all cirrhotic and non cirrhotic patients, ultrasonography-guided liver biopsy was performed, mostly from the right lobe of the livers. In 6 of the 46 cirrhotic patiens, the prothrombin time was high. The diagnosis was based on the history of the disease and laboratory and ultrasonographic findings. Endoscopic biopsies were performed using Olympus forceps in all patients. We obtained four perendoscopic biopsy specimens; one was taken in the antrum within 5cm of the pylorus, one in the the gastric body, one in the anterior wall and one in the upper part of the body.

Histology

All biopsy specimens taken from the livers and stomachs were fixed in buffered formalin, and then stained with H & E. The gastric biopsy specimens were oriented tangentially. We used the histological classification of chronic gastritis of Cheli et al (11). When different levels of gastritis were present, the most severe was chosen.

Serum gastrin

On a different day after an overnight fast, two blood sample were obtained 10 minutes apart from each patient studied. Each sample was stored immediately at 4°C. The serum from all samples was then extracted by centrifugation and stored at -20°C. Gastrin was measured twice by radioimmunoassay using commercially available assay kits. The normal range of gastrin levels was 30-140 pg/mL.

Statistical Analysis

In the study, statistical analysis of the differences with respect to different discrete study variables between the groups was tested by applying $x^2$ analysis and Fisher's Exact Test.

## RESULTS

Endoscopy

Table I. shows the frequency of endoscopic findings in the stomach, duodenum and esophagus. In patients with liver cirrhosis, the most frequent endoscopic findings were snake skin (56.8%), scarlatina rash (35.1%), petechia (37.8%), and hyperemia (54%). In this group the incidence of fundic varices (21%) was less than in non-cirrhotic patients (46%) and the difference was statistically significant ($p<0.001$). When we look at the esophageal varices with re-

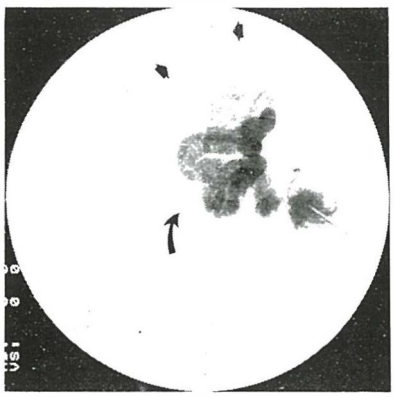

**Figure 1:** Splenoportography demonstrates hepatofugal flow and very large coronary vein (arrows) but no blood flow towards liver in cirrhotic patient with PVO (curved arrow).

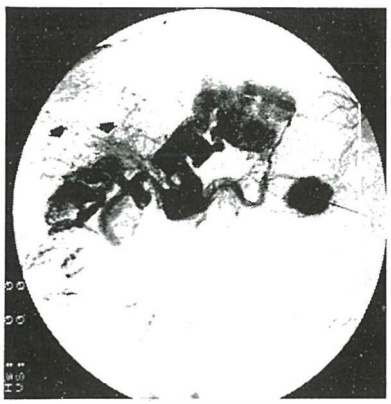

**Figure 2:** Splenoportography reveals tortuous vessels at the porta hepatis, replacing the main portal vein in patients with PVO. Note: there is still blood flow towards liver ( arrows).

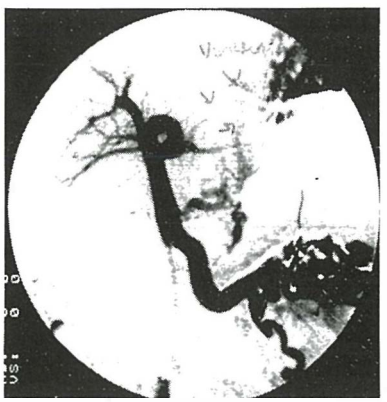

**Figure 3:** Splenoportography shows hepatofugal blood flow but also dilated portal veins passing the blood through the liver in a cirrhotic patient in whom portal pressure was probably less than in the other two cases as shown in Fig. 1-2.

gard to the form and extension, the full extension and F3 forms were more prominent in patients with PVO in comparison to cirrhotic patients ($p<0.001$).

The red signs, which can be predictive factors in estimating the bleeding from varices, were significantly lower in incidence in patients with PVO compared with cirhotic patients($p<0.001$). The highest level was in cirrhotic patients with PVO. When the two cirhotic groups were compared with each other, the difference was statistically significant ($p<0.001$). Table II shows some characteristics of the patients and control subjects.

Splenoportography

In all patients with portal vein involvement, including cirrhotic patients, portography shows multiple collaterals and tortuous vessels at the porta hepatis and a hepatofugal blood flow. The obstruction was almost complete in cirrhotic patients (Fig. 1) compared with patients having only portal venous obstruction (Fig. 2). We also performed portography in ten cirrhotic patients to demonstrate whether there is blood flow through the liver. As seen in Fig. 3, in all cirrhotic cases there was blood flow in the liver although hepatofugal flow had been seen.

Histology

Histology tests did not show any significant difference in type of chronic gastritis between the three groups of patients and the control group. No percentage differences were found in the three groups and, distribution was substantially uniform. However, there were some percentage differences in the topographic distribution. Superficial and chronic superficial gastritis prevailed in the fundic area in the cirrhotic patients whereas atrophic gastitis was more frequently seen in the antrum of the control patients.

Serum gastrin

Fasting serum gastrin values in cirrhotic patients (mean±SD, 198±156 pg/mL) were significantly higher than in the control patients (mean±85±37 pg/mL) and in the patients with PVO (mean±79±45 pg/mL) ($p<0.001$). Although gastrin levels were highest in cirrhotic patients with portal vein obstruction, the difference was not statistically significant (mean±203±185) when compared with patients with cirrhosis. Of course, the same differences had been found among cirrhotic patients with PVO, normal controls and patients with portal vein obstruction. The simultaneous presence of high mean fasting serum gastrin values in both groups of cirrhotic patients with and without PVO did not reveal statistically significant correlation with the presence of characteristic mucosal lesions. In considering the severity of the disease (Child-Pugh grade), we could not find a statistically significant correlation between hypergastrinemia and

the two grades of clinical severity (we had only Child B and C grade patients).

Discussion

The three macroscopic aspects that, either singly or in association, may identify and characterize the gastric mucosa of cirrhotic patients with portal hypertension are petechia, scarlatina rash hyperemia, and snake skin. Papazian et al and McCormack et al have recently investigated this subject (3,4). None of our patients had had episodes of bleeding from the upper gastroentestinal tract during the previous 3 months from either varices or ulcers or portal gastropathy, the major causes of bleeding in patients with portal hypertension, and none had ever undergone sclerotherapy or other procedures related to bleeding at the time of endoscopic evaluation. These criteria for admission to the study were chosen to assess the gastric mucosa in the best condition for endoscopy and to avoid the possibility that the absence or presence of characteristic endoscopic signs would be affected by the altered hemodynamic condition caused by sclerosis or bleeding which can decrease the appearance of the varices as pointed out by other investigators (12). Snake skin was present in 56.8% of the cirrhotic patients, in 89% in cirrhotic patients with portal vein thronbosis and in 15% patients with PVCT but in 2% of control patients; therefore, we felt it may be considered a significant endoscopic sign which influenced not only portal hypertension but also parenchymal liver disease. The other endoscopic signs such as petechia, hyperemia and scarlatina rash were characteristic of cirrhosis. As seen in Table I, all mucosal lesions in the stomach and esophagus had a high incidence in cirrhotic patients when compared with the cases of portal vein obstruction, and this difference was statistically significant. On the other hand, gastric varices and the gravity of the esophageal varices were prominent in patients with portal vein obstruction and cirrhotic patients with PVO. As well known, in cirrhotic patients, there is still blood flow through the liver, but in the case of PVO, hepatopetal flow has been decreased as much as possible (Fig. 2); which means that the portal pressure in these patients might be higher than in cirrhotic patients, although we could not measure the portal vein pressure, it may therefore be possible to say that the greater the portal pressure increase, the greater the increase in esophageal varices and incidence of gastric varices. It seems that additional mucosal lesions require parenchymal liver disease, since almost every cirrhotic patient had mucosal lesions and high degree varices. It is necessary to compare the portal pressure with the gravity of the liver disease in a large series in which portal hypertension should be the result of liver cirrhosis and liver cirrhosis complicated with portal vein thrombosis in order to reach a clear-cut conclusion.

In general, vascular mucosal lesions and esophageal red signs such as cherry red spot, haematocystic spot were correlated with the clinical gravity of the disease (Child-Pugh grade) or with the Beppu's score. Most patients with vascular lesions of the gastric mucosa were in grade C. on the other hand, although the size and extension of varices in patients with CTPV were of the advanced stage, the red signs were significantly decreased in these patients when compared with those of cirhotic patients. It seems that liver disease is the major determinant factor in the development of vascular mucosal lesions located in gastric and esophageal mucosa. This finding was obvious in cirrhotic patients with portal vein involvement due to probable liver disease.

The fasting values of gastrin in our cirrhotic patients were significantly higher than those of the controls and of the patients with CTPV, but was same as those of cirrhotic patients with CTPV, which confirm Lam's (13) and Vigneri's findings (14) but not those of Quintero et al (7). Lam attributed the hypergastrinemia to hypoacidity and accordingly the lack of gastrin-inhibiting feedback(13) and it can result from liver disease in which the metabolism of gastrin may be delayed. In this study we could not measure the gastric acid content in every case and we also did not find any statistical relationship between mucosal histology and fasting gastrin levels in the studied cases. The methodology of histological assessement of the mucosal condition may also have been incomplete and insufficient, although the possibility that other factors beside gastric atrophy and paranchymal liver disease play a role cannot be excluded. The mucosa of

patients with portal hypertension demonstrates particular endoscopic signs that singly or in definitely combination characterize it unequivocally. The signs seem to depend on the patients' clinical condition. The gravity of the esophegeal varices and endoscopic signs are related to the etiologic factors responsible for portal hypertension. The red spots on esophageal mucosa and portal gastropathy findings in cirrhotic patients, particularly complicated with portal thrombosis, were significantly higher than those of patients with extrahepatic portal hypertension. Medium to long-term prospective studies will enable us to determine which factor or factors is effective in the development of these mucosal vascular lesions.

The incidence of portal gastropathy in the West is considerable high(9,15), while a low incidence has been reported a in India(8,9,15,17) where the majority of the cases were of extrahepatic portal hypertension. As seen in Table I, mucosal vascular lesions in the overall estimation are seen in about 50% cirrhotic patients and 20%non-cirrhotic portal hypertensive patients but, although the group is small(nine cases), in almost all cases, gastropathy has been found. Why is there such variability in the incidence of portal gastropathy? The current study provides an excellent opportunity to investigate this clinical problem because there is a large number of patients with different portal hypertension etiologies. The study material includes patients with liver cirrhosis, portal venous obstruction and with cirrhosis complicated with portal venous occlusion. Most study material in the Western world, includes patients with portal hypertension due to liver cirrhosis. During 8 years we diagnosed 26 patients with portal venous occlusion whose livers were histologically normal. In these cases, although the incidence of fundic varices and the extension and forms of the esophageal varices were predominant, the incidence of mucosal lesions was low. Unfortunately, we could not measure the variceal pressure but we believe that the endoscopic appearance of the varices is related to the portal pressure. On the other hand, mucosal vascular lesions are related to both portal hypertension and the gravity of parenchymal disease, because all nine cirrhotic patients with portal hypertension had F3 and L3 and gastric varices, and had at least three of the mucosal lesions.

Is portal pressure the sole determinant factor in the development of portal hypertensive gastropathy? This is suggested by some investigaters (3,7). Some believe that this kind of gastropathy and varices have a common origin. This seems unlikely because not every patient with portal hypertension or variceal obliteration (6) develops portal gastropathy. Moreover, according to the study of Sarin et al(6). the variceal pressure was similar in patients with and without portal hypertensive gastropathy. Therefore, in addition to pressure, other related factors such as development of portosystemic shunts, esophageal varices, and individual factors in the gastric circulation may determine the changes of development of these mucosal lesions in a given patient.

In the present study, the gastric varices seem to be related to portal hypertension rather than liver cirrhosis, because they exist in 21% of the cirrhotic patients, in 46% of the extrahepatic portal hypertension and 100% of portal venous obstruction patients. As seen in Fig. 1, almost complete occlusion of the portal vein with very large collaterals in the fundic area of the stomach were the main radiologic findings of cirrhotic patients with portal venous obstruction. These radiologic findings, which corresponded to endoscopic findings in all 9 patients, were not prominent in cirrhotic patients without portal venous obstruction. On the other hand, as shown in Table I, the incidence of mucosal vascular abnormalities in the gastric or esophageal mucosa was so high that it is possible to say that the development of mucosal lesions necessitates both liver parenchymal disease and portal hypertension.

The other important observation in our study was that the development of portal gastropathy was greatly influenced by the severity of the liver disease. Patients with liver disease of Child's grade C had portal gastropathy significantly more often than those with Child's B liver disease.

The results suggest that, although increased portal pressure is a prerequisite, the development of portal hypertensive gastropathy is

strongly influenced by the severity of the liver disease. On the other hand, the form and extention of the esopgageal varices seem to be related to portal hypertension. More studies including cirrhotic patients with portal venous occlusion are needed to reach a more precise judgement.

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