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SONOGRAPHIC CORRELATION BETWEEN PORTAL VEIN DIAMETER AND SPLEEN LENGTH IN PATIENTS WITH CHRONIC LIVER DISEASE

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Abstract

Background: *For several decades, ultrasonography (US) and Doppler US have been widely used to diagnose splenomegaly and portal vein diameter. It has become the most common and valuable diagnostic modality for detecting not only parenchymal disease but also anomalies in liver hemodynamics via Doppler imaging due to its low cost, ease of use, and high patient compliance. As a result, previous studies have evaluated several US indices used to identify cirrhosis, such as nodular liver surface, blunted liver edge, coarse parenchymal echogenicity, atrophic change of the right lobe, splenomegaly, and enlarged splenic vein diameter (SV). Furthermore, Doppler US indices, which have been widely used to assess PH, include measurements of portal and splenic venous blood velocity.*

Objective(s): *To determine the correlation between portal vein diameter and spleen size (craniocaudal)*

Methodology: *T he study was conducted at the National hospital and medical center, Lahore Pakistan. All the individuals who referred for abdominal sonographic examination, including male, female, older and younger were conveniently included in the study, voluntarily, irrespective of the disease state. Ultrasonographic measurements of the caudocranial length of the spleen and portal vein were carried out on all of the one thousand subjects. The subject position for spleen was supine or right posterior oblique during suspended inspiration and right anterior oblique position for portal vein diameter with quiet respiration.*

Results: *In this study total of 65 patients were include in which 30 patient were*

female and 35 were male and out of 65 in46 patients, Hypertension was diagnosed in 29 males 17 female and out 65 in 52 patient diabetes mellitus was seen 30 male and 22 female . Patient with CLD 23 male were chain smoker and 3 male were consume alcohol regularly. In this study 34 patients was obese 18 male and 16 female. Out of 65 patient chronic liver diseases 50 patient has hepatitis C and 6 presented with NAFLD other patient with CLD caused by alcoholic fatty liver and hepatitis B and frequency of alcoholic fatty liver and hepatitis B was 4. Ten patients present with the sharp edge of liver and 8 presented with blunt edge. Out of 65 patient , in 26 patients splenomegaly was scene. It was also observed that 17 patients out of 65 present with dilated portal vein diameter. When portal vein diameter was correlate with the splenomegaly it was observed that 1 patients with splenomegaly have normal portal vein diameter and 25 patients out of 26 splenomegaly patients have increased portal vein diameter. Mean portal vein size was 13.457 mm, minimum size measured was 6.5 mm and maximum 21.5 mm ,the average size of spleen measured in 65 patients was 13.458 cm and minimum size observed was 7.0 cm and maximum size of spleen observed was 21.0 cm. when the correlation of spleen size and portal vein was checked was found between the caudocranial length of the spleen and portal vein diameter with P-value (0.001), the correlation was significant at less than 0.01 level and R-value (0.997), which is near to 1, represents a strong positive relationship and he concluded that Portal vein diameter and splenic craniocaudal length are linearly correlated.

Conclusion(s): *The diameter of the portal vein and the splenic craniocaudal length are linearly related. In the case of chronic liver parenchymal disease, an increase in spleen size may predict an increase in portal vein diameter (liver congestion).*

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INTRODUCTION

The liver is located in the upper right quadrant of the abdominal cavity, beneath the diaphragm, above the stomach, right kidney, and intestines. The liver is a cone-shaped, dark reddish-brown organ that weighs about 3 pounds. When dangerous compounds are broken down, their byproducts are expelled into the bile or blood. The primary channel of the portal venous system (PVS), carries blood from the spleen, gallbladder, pancreas, and digestive tract to the liver, is the portal vein (PV). Normal portal venous pressure (PVP) is between 5 and 10 mmHg, while normal portal vein diameter (PVD) ranges between 7 and 15 mm.[1]

Because the portal vein also drains blood from the spleen, chromosomal liver illness can occasionally induce an obstruction in the portal vein, which may result in an increase in the width of portal vein and the spleen. While normal arterial structure of celiac trunk and its major branches has been described. However, only a few publications have linked splenomegaly to alterations in the splenic artery's diameter. Patients with liver cirrhosis have shown a substantial correlation between the grade of splenomegaly and the width of portal vein. Recent research has shown that chronic liver illness causes diameter alteration in the hepatic and splenic arteries. Splenic artery alterations occur before clinically obvious portal hypertension manifests. Patients with hepatic cirrhosis had much larger gallbladders than healthy individuals. Arithmetical analysis did not find any appreciable difference between people with liver disease and controls in angles created by the longitudinal and transverse paths of the left portal vein. When good liver tissue is changed into scar tissue, chronic liver disease develops. Scar tissue inhibits the blood flow through the liver in cirrhosis.[2] The liver loses its

ability to function properly over time. Globally, 1.5 billion cases of CLD are thought to exist (inclusive of all disease severity stages). NAFLD (59 percent) is the leading cause of prevalent illness, followed by HBV (29 percent), HCV (9 percent), and ALD (2 percent). 1% of cases of liver illness are caused by, Wilson's disease, alpha-1-antitrypsin deficiency , biliary cholangitis, sclerosing cholangitis, hepatitis, and sclerosing cholangitis.

Chronic alcohol misuse, chronic hepatitis (hepatitis B, C, and D), and liver fat buildup are a few of the causes (nonalcoholic fatty liver disease), an accumulation of iron in the body (hemochromatosis), Poorly developed bile ducts (biliary atresia), Cystic fibrosis, Wilson's disease, a lack in alpha-1 antitrypsin, and inherited problems with sugar metabolism (such galactosemia or glycogen storage disease), immune system-driven liver illness (autoimmune hepatitis), damage to the bile ducts (primary biliary cirrhosis), a disease like syphilis or brucellosis. Indicators of chronic liver disease include a yellowness of skin or the whiteness of eyes (jaundice), rough skin, edema, or swelling of ankles, feet, and legs fluid accumulation in your abdomen or belly (ascites).[3] Your urine is brownish or orange in hue, your faeces are light in colour, confusion, brain fog, memory lapses, personality shifts, stool with blood, Your hands' palms may be red. Loss of sex desire, gynecomastia, and shrinking testicles are all symptoms in men. premature menopause in women (no longer having your menstrual period). Risk factors include excessive alcohol consumption, having viral hepatitis and being overweight. Portal hypertension, swelling in the legs and abdomen, and high B.P in veins supplying liver are among the problems. Bleeding, Swelling of spleen, Malnutrition, Infections, toxic buildup in brain, Jaundice, bone illness, Acute-on-chronic cirrhosis and increased risk of liver cancer.[4] One of the most common side effects of cirrhosis is portal hypertension. Portal hypertension is the most common complication of chronic liver disease and one of the leading causes of death. Increased resistance to portal blood flow caused by liver changes, portal vein narrowing, splenomegaly, and food borne. Cirrhosis causes increased intracranial vascularization, which is most noticeable in the liver sinuses.[5]

Superior mesenteric and splenic vein combine to produce the portal vein (PV), which is located behind to the pancreas at level of L2. The left and right portal veins split at the porta hepatis, 5 to 7 cm from the porto-splenic confluence. The portal vein travels through the initial part of the duodenum's posterior half before passing through the lesser omentum's layers and ending at the porta-hepatis. It splits into its right and left portal veins in liver, then divides again to produce its terminal branches in hepatic sinusoids. Pancreatic, gallbladder, and gastrointestinal tract blood, bile ducts, and spleen is drained by the portal vein to the liver and finally to IVC through hepatic veins.

The rectal venous plexus, esophageal veins and the superficial abdominal veins are anastomosed with the portal vein. The portal vein and the hepatic artery both give blood to the liver. Branching off of the portal vein, hepatic artery, and bile duct make up the portal triad. These structures are encased in a connective tissue sheath, which on sonography gives the portal vein the appearance of an echogenic wall. At the splenic hilum, numerous veins unite to form the splenic vein. The left gastroepiploic and short gastric veins then connect to it. The posteromedial edge of the pancreas is where the splenic vein is located. The portal vein is created when it connects to superior mesenteric vein behind neck of pancreatic, Splenic vein is drained into by additional pancreatic and inferior mesenteric venous veins. Blood is drained into portal vein by the splenic vein from the pancreas, spleen, and stomach.

The primary initiating factor in the normal course of cirrhosis is portal hypertension. When the disease is in its early stages, the liver's increased resistance to portal blood flow—caused by abnormalities including fibrosis and nodules—determines the portal pressure. Extrahepatic vascular alterations take place as the disease progresses. A state known as effective hypovolemia is caused by arterial vasodilation

and neo-angiogenesis at the splanchnic circulation. A hyperkinetic condition that increases portal venous blood flow is brought on by effective hypovolemia. Low peripheral vascular resistance and a high cardiac output are characteristics of the hyperkinetic syndrome. Along with these alterations, neuro-humoral vasoactive mechanisms are activated, which leads to kidney hydroelectrolyte retention.[6]

The end result is a significant increase in the amount of blood moving through the splanchnic circulation. Ascites, which is the result of fluid escaping into the peritoneal cavity due to the liver's failure to handle sinusoidal resistance to high blood flow, develops when the portal pressure rises more (varices). Type-2 hepato-renal syndrome (HSR-2) and/or hyponatremia are the severe effects of vasodilation and hydro-electrolyte imbalance in the most advanced stages of cirrhosis.[7] It has recently been discovered that type-1 HRS is caused by a decrease in cardiac output and the subsequent reversion of the hyperdynamic condition into a (relative) hypodynamic circulatory state. Any comorbidity that can more decrease vascular tone and cardiac role is potentially fatal because it could permanently damage organ perfusion. Splanchnic vasodilation is crucial in development of hypovolemia, the hyperdynamic flow, and subsequent additional increase in portal pressure. This could happen if there is SBP present (or perhaps other illnesses), or if a high volume of fluid is removed during paracentesis deprived of sufficient size expansion with albumin. Overall, the management of vascular homeostasis and organ perfusion deteriorates along with onset of severe portal hypertension, which is one major pathogenic mechanisms driving high death rate of acute and chronic liver failure.[8]

Hepato-cellular deficiency is another significant pathogenic process that manifests clinically in advanced cirrhosis.[9] Splenomegaly is a crucial finding that frequently coexists with entrance hypertension. An extremely high level of steady quality amplification is shown by a cephalo-caudal estimation of 13 cm. Recent investigations have demonstrated that the dynamic component of increased resistance is exacerbated in chronic liver disease, with increased resistance brought on by morphological changes. In liver cirrhosis with portal hypertension, the width is typically increased, and the size of the spleen is also increased.

The patient was placed in the recumbent position to obtain an ultrasonographic estimation of the size of the spleen. Using a 2-5 MHz curved transducer in one of the lower left intercostal gaps, in the segment posterity coronal plane. The normal adult spleen measures about 12 cm in length.[10] The spleen parenchyma is uniformly mid-to-low echogenic and highly homogeneous. The spleen tends to become more echogenic as it expands, The spleen is the biggest single mass of lymphoid tissue and is situated in left upper quadrant of the abdomen, directly below diaphragm. It is a component of the reticuloendothelial system. The body's defense system heavily depends on the spleen (reticuloendothelial system). Despite being frequently impacted by complete illness processes, the spleen rarely becomes the major place of disease. Sonographically, the splenic parenchyma seems to have uniform homogenous mid to low-level echo-pattern, similar to hepatic parenchyma.[11]

Actually, it's thought that the spleen's texture is more echogenic than the liver's. The echogenicity of spleen rises by growth, maturation, and size like that of all other abdominal organs. Delivering lymphocytes and plasma cells, which are used in humoral and cell-safe barriers, is the spleen's primary function. This organ stores around half of the body's monocytes. These cells can effectively differentiate into dendritic cells and macrophages, helping to heal wounds. Additionally, the spleen directs blood and eliminates all waste products from cells and microbes such as viruses, germs, and parasites. When diseases like cancer, anaemia, intestinal illness, tuberculosis, amyloidosis, cirrhosis, hepatitis, and the like interfere with the body's normal functioning, the spleen becomes hyperactive and begins to ensnare and store a large amount of blood cells and platelets. The result is a dramatic decrease in the amount of blood cells and platelets in the circulatory system.

The spleen grows larger as a result of capture, and as it matures, it captures progressively more blood cells and platelets. In the long term, the clogged spleen begins to collect and crush the regular blood cells with the abnormal ones. These blood and platelet components obstruct the spleen's normal function. The trademark indication of spleen expansion is serious irritating pain in the belly and back. When there is hepatic congestion, the portal vein and finally the splenic vein experience resistance to blood flow. The excessive blood pooling causes pressure to build up in the portal vein, which leads to portal hypertension and an eventual increase in portal vein width. Splenomegaly, on the other hand, is brought on by blood clots in the spleen; as a result, splenomegaly and portal vein diameter are connected. Splenomegaly may result from a variety of diseases, including hematologic disorders, rheumatologic disorders, infectious diseases, and infiltrative disorders, in addition to hepatic fibrosis and congestion. However, the most typical cause of splenomegaly and portal hypertension is hepatic congestion.[12]

As a result, we found that increases in Portal vein width also result in proportional increases in splenic caudocranial length. Your blood flow is slowed by cirrhosis, and the portal vein is under stress. Portal hypertension, which is elevated blood pressure, is brought on by this. The progression of liver fibrosis causes an increase in its diameter, which then rises with the onset of cirrhosis, where the mean portal vein diameter is estimated to be around 14 mm. Systemic portal clusters develop at place of splenomegaly and in other sites as a result of portal hypertension. When there is liver cirrhosis, the spleen is also enlarged in size and the portal vein breadth is often increased. Depending on the cause of the condition, a patient's spleen can vary in size with cirrhosis.[13]

A lot of data has been gathered on the sonographic anatomy of venous system, illnesses, and morphologic variations and liver texture alterations because ultrasound is popular noninvasive technique to examining chronic liver disease. There is a wealth of ultrasonographic data describing changes in extrahepatic portal vessels, but the intrahepatic course of the portal veins in ill individuals has never been thoroughly assessed. Sonography is most usually used to evaluate the left upper quadrant in patients with enlarged spleens or in cases of trauma. [14]

The sonographic examination is interactive, real-time, and dynamic in addition to all other benefits. Patients can be easily moved, deep breathed in, squeezed, and subjected to other stimulating techniques. Additionally, the transducer technique might be altered so that deeper access could lead to more superficial access. As a multiplanar modality, ultrasound allows for the acquisition of numerous planes as opposed to just one. As a result, ultrasonography is becoming increasingly significant in regards to the assessment of deep gynaecological, obstetrical, and abdominal systems as well as superficial musculoskeletal structures, nerves, and muscles. The gall bladder, intestinal walls, pancreases, liver, spleen and abdominal vasculature are, among other abdominal structures, are all easily distinguishable using sonography.[15] The spleen's natural texture is fairly uniform and slightly more echogenic than that of liver, so pathology brought on by a splenic rupture is typically obvious. By using sonography, we may also assess the condition of the portal vein in terms of its diameter, hypertension, blockage, thrombus, and collaterals.[16]

The purpose of this study was to compare the length of the spleen and the width of portal vein in individuals with CLD. so that preventive strategies may be used to lower this condition if risk factors were identified.

OBJECTIVE

To determine the correlation between diameter of portal vein and length of spleen in patients with chronic liver disease.

MATERIAL AND METHODS

Study design: Descriptive study

Settings: National hospital and medical center Lahore

Duration of study: 4 months after approval of synopsis

Sample size: 65

Sampling technique: Convenient sampling technique

Sample selection:

Inclusion Criteria

- Patients with chronic liver disease having hepatitis,
- Patients with diffuse liver disease
- Patients with mean age was 51.04 ± 13.53 years, ranging from 20 to 85 years
- Patients with jaundice

Exclusion Criteria

- Uncooperative Patients to participate in research project

Equipment: Ultrasound machine Toshiba with 2.5 to 5 MHz curvilinear multiphase probe was used.

Technique:

Spleen and Liver estimation was estimated ultrasonographically by putting the patient in prone position, utilizing 2 - 5 MHz curvilinear transducer in the coronal plane of segment posteriorly in one of the lower left intercostal spaces. The patient was analyzed in different degrees of motivation to amplify the window to the spleen and Liver. The plane of segment was then cleared posteriorly and anteriorly to see the whole volume of spleen to measure diameter.

RESULTS

In this study total of 65 patients were include in which 30 patient were female and 35 were male ant the mean age 65 year and minimum age included was 25 year and maximum age was 84 of the patients was and out of 65 in46 patients, Hypertension was diagnosed in 29 males 17 female and out 65 in 52 patient diabetes mellitus was seen 30 male and 22 female . Patient with CLD 23 male were chain smoker and 3 male were consume alcohol regularly. In this study 34 patients was obese 18 male and 16 female. Out of 65 patient chronic liver diseases 50 patient has hepatitis C and 6 presented with NAFLD other patient with CLD caused by alcoholic fatty liver and hepatitis B and frequency of alcoholic fatty liver and hepatitis B was 4. Ten patients present with the sharp edge of liver and 8 presented with blunt edge. Out of 65 patient , in 26 patients splenomegaly was scene. It was also observed that 17 patients out of 65 present with dilated portal vein diameter. When portal vein diameter was correlate with the splenomegaly it was observed that 1 patients with splenomegaly have normal portal vein diameter and 25 patients out of 26 splenomegaly patients have increased portal vein diameter. Mean portal vein size was 13.457 mm, minimum size measured was 6.5 mm and maximum 21.5 mm ,the average size of spleen measured in 65 patients was 13.458 cm and minimum size observed was 7.0 cm and maximum size of spleen observed was 21.0 cm. when the correlation of spleen size and portal vein was checked was found between the caudocranial length of the spleen and portal vein diameter with P-value (0.001), the correlation was significant at less than 0.01 level and R-value (0.997), which is near to 1, represents a strong positive relationship and he concluded that Portal vein diameter and splenic craniocaudal length are linearly correlated.

Statistics

age

N	Valid	65
	Missing	0
Percentiles	25	48.50
	50	60.00
	75	70.00

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
Age	65	25	84	60.18	14.302
Valid N (listwise)	65				

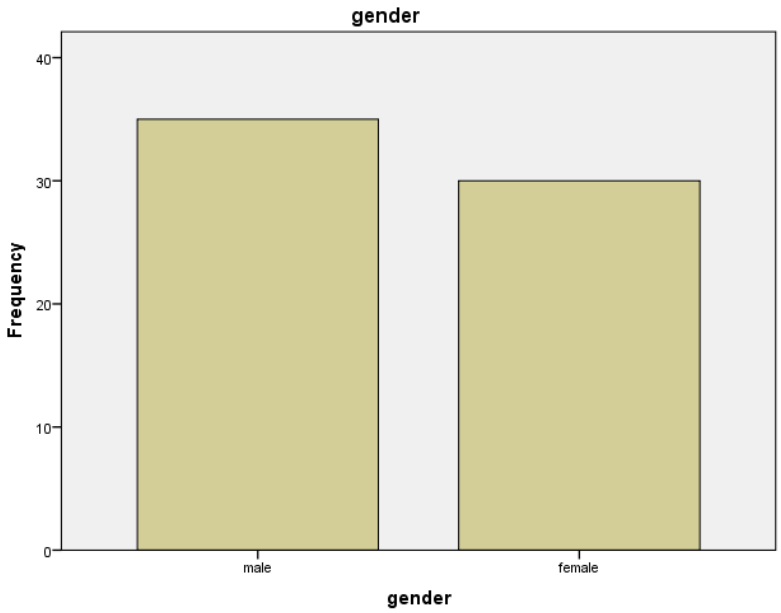
Age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	25	2	3.1	3.1	3.1
	35	1	1.5	1.5	4.6
	38	1	1.5	1.5	6.2
	39	1	1.5	1.5	7.7
	40	2	3.1	3.1	10.8
	44	1	1.5	1.5	12.3
	45	5	7.7	7.7	20.0
	46	1	1.5	1.5	21.5
	48	2	3.1	3.1	24.6
	49	1	1.5	1.5	26.2
	50	1	1.5	1.5	27.7
	51	1	1.5	1.5	29.2
	55	4	6.2	6.2	35.4
	56	3	4.6	4.6	40.0
	58	1	1.5	1.5	41.5
	60	7	10.8	10.8	52.3
	65	9	13.8	13.8	66.2
	68	1	1.5	1.5	67.7
	69	1	1.5	1.5	69.2
	70	5	7.7	7.7	76.9
	72	1	1.5	1.5	78.5
	73	1	1.5	1.5	80.0
	75	4	6.2	6.2	86.2
	76	1	1.5	1.5	87.7
	80	5	7.7	7.7	95.4
	82	1	1.5	1.5	96.9

	84	2	3.1	3.1	100.0
	Total	65	100.0	100.0	

Gender

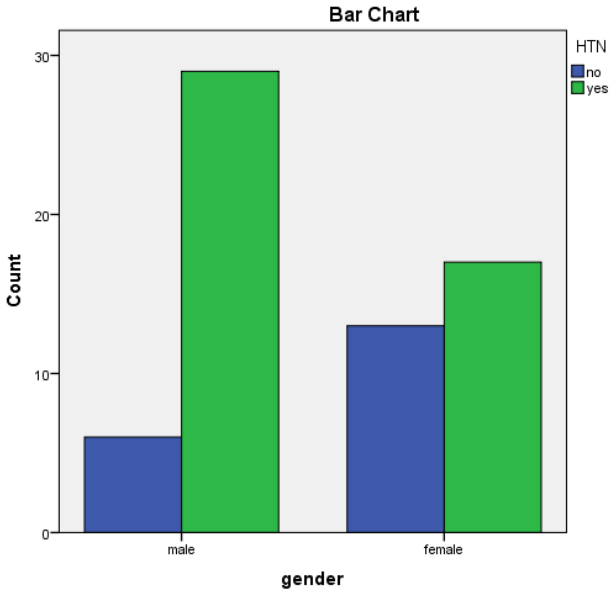
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	35	53.8	53.8	53.8
	Female	30	46.2	46.2	100.0
	Total	65	100.0	100.0	



gender * HTN Cross tabulation

Count

		HTN		
		No	Yes	Total
gender	Male	6	29	35
	female	13	17	30
Total		19	46	65

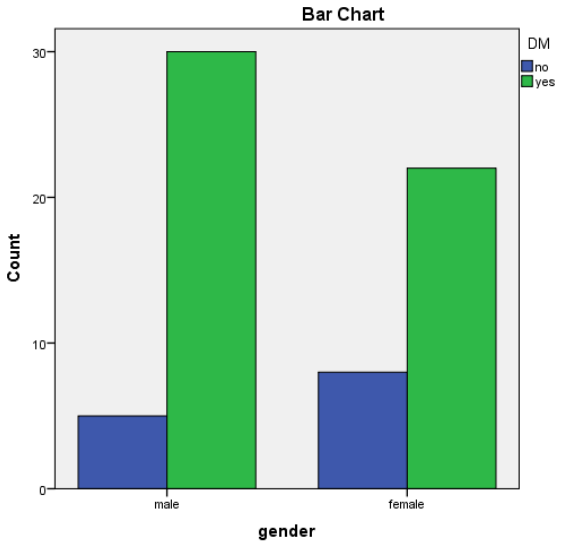


gender * DM Cross tabulation

Count

		DM		
		No	yes	Total
gender	Male	5	30	35
	female	8	22	30
Total		13	52	65

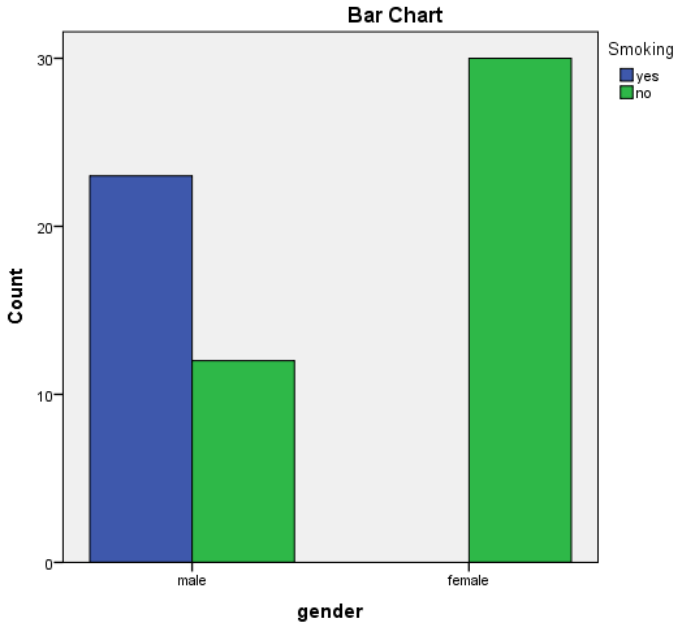
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gender * Smoking Cross tabulation

Count

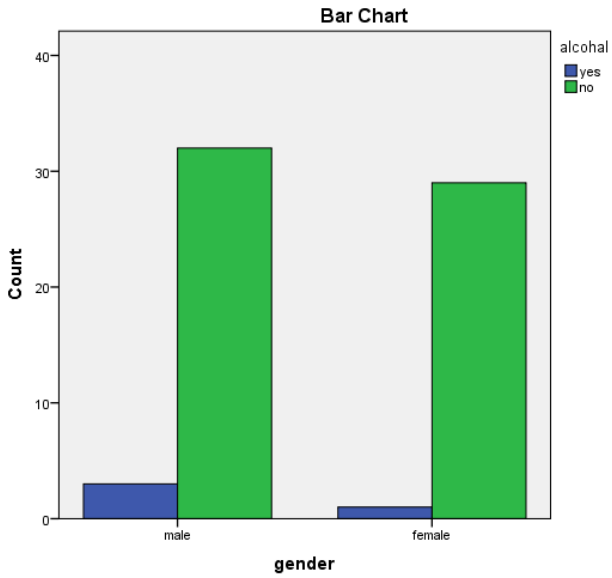
		Smoking		
		Yes	no	Total
gender	Male	23	12	35
	female	0	30	30
Total		23	42	65



gender * alcohol Cross tabulation

Count

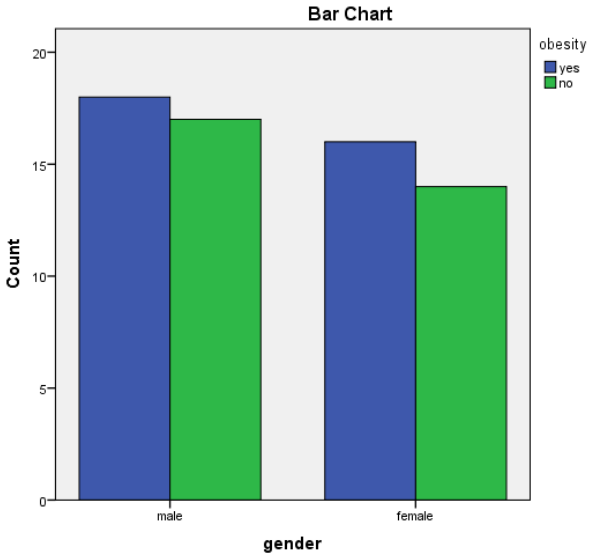
		alcohol		
		Yes	no	Total
gender	Male	3	32	35
	female	1	29	30
Total		4	61	65



gender * obesity Cross tabulation

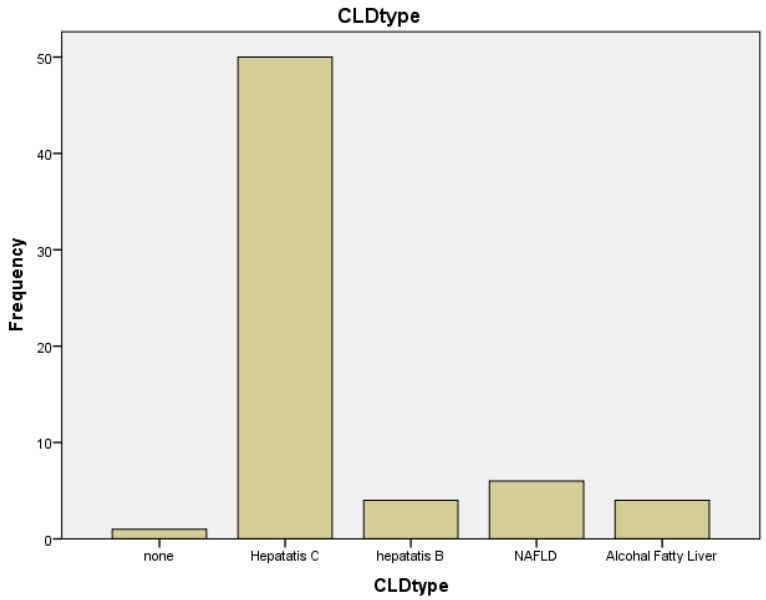
Count

		obesity		
		Yes	no	Total
gender	Male	18	17	35
	female	16	14	30
Total		34	31	65



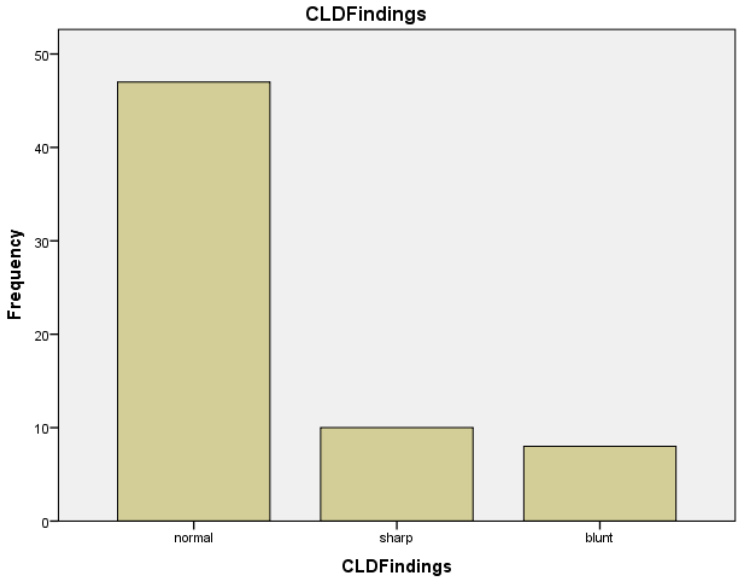
CLD type

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	None	1	1.5	1.5	1.5
	Hepatatis C	50	76.9	76.9	78.5
	hepatatis B	4	6.2	6.2	84.6
	NAFLD	6	9.2	9.2	93.8
	Alcohal Fatty Liver	4	6.2	6.2	100.0
	Total	65	100.0	100.0	



CLD Findings

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	normal	47	72.3	72.3	72.3
	Sharp	10	15.4	15.4	87.7
	Blunt	8	12.3	12.3	100.0
	Total	65	100.0	100.0	



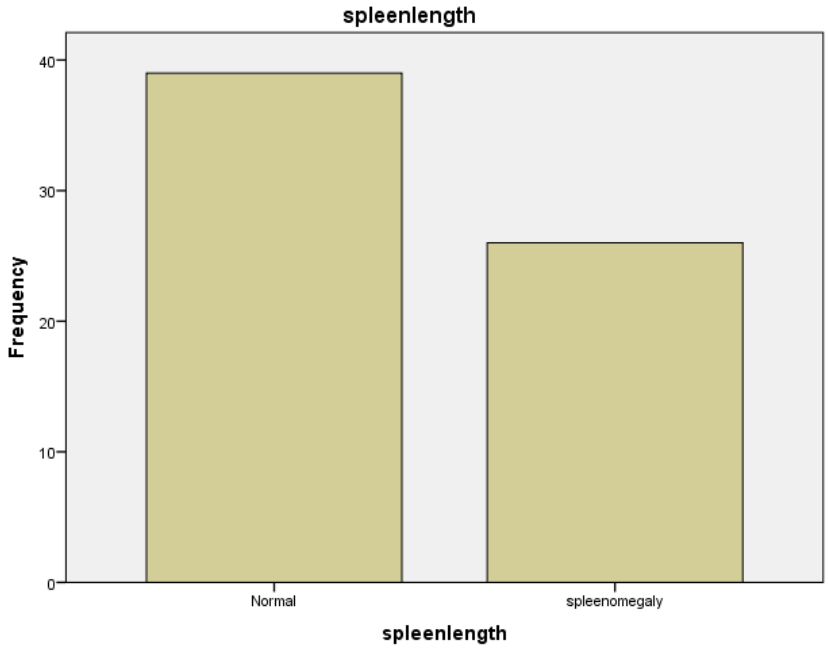
Statistics

Spleen length in CM

N	Valid	65
	Missing	36
Mean		13.458
Std. Deviation		5.1092
Minimum		7.0
Maximum		21.5

Spleen length

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	39	60.0	60.0	60.0
	Splenomegaly	26	40.0	40.0	100.0
	Total	65	100.0	100.0	



SLCM

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	7.0	5	5.0	7.7	7.7
	8.0	10	9.9	15.4	23.1
	9.0	7	6.9	10.8	33.8
	10.0	7	6.9	10.8	44.6
	11.0	3	3.0	4.6	49.2
	12.0	2	2.0	3.1	52.3
	13.0	2	2.0	3.1	55.4
	14.0	2	2.0	3.1	58.5
	14.5	1	1.0	1.5	60.0
	17.0	1	1.0	1.5	61.5
	17.5	1	1.0	1.5	63.1
	18.0	4	4.0	6.2	69.2
	18.5	2	2.0	3.1	72.3
	18.6	1	1.0	1.5	73.8
	19.0	4	4.0	6.2	80.0
	19.2	1	1.0	1.5	81.5
	19.5	3	3.0	4.6	86.2
	20.0	3	3.0	4.6	90.8
	20.5	2	2.0	3.1	93.8
	21.0	3	3.0	4.6	98.5
	21.5	1	1.0	1.5	100.0
	Total	65	64.4	100.0	

Missing	System	36	35.6		
Total		101	100.0		

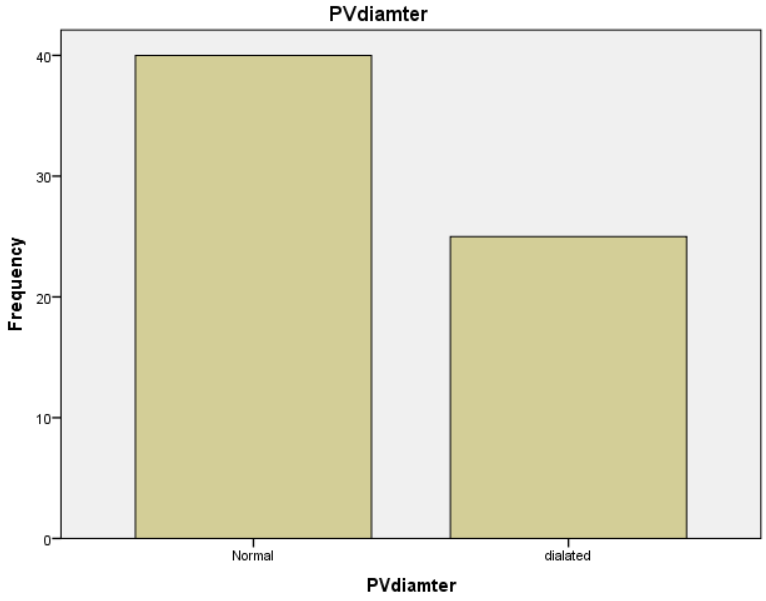
Statistics

PV diameter in MM

N	Valid	65
	Missing	36
Mean		13.457
Std. Deviation		5.0792
Minimum		6.5
Maximum		21.5

PV diamter

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	40	61.5	61.5	61.5
	dilated	25	38.5	38.5	100.0
	Total	65	100.0	100.0	



PV in MM

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	6.5	1	1.0	1.5	1.5
	7.0	4	4.0	6.2	7.7
	8.0	8	7.9	12.3	20.0
	8.5	1	1.0	1.5	21.5
	9.0	8	7.9	12.3	33.8
	10.0	7	6.9	10.8	44.6
	11.0	3	3.0	4.6	49.2
	12.0	2	2.0	3.1	52.3
	13.0	2	2.0	3.1	55.4
	14.0	2	2.0	3.1	58.5
	15.0	1	1.0	1.5	60.0
	16.0	1	1.0	1.5	61.5
	17.5	1	1.0	1.5	63.1
	18.0	4	4.0	6.2	69.2
	18.5	4	4.0	6.2	75.4
	19.0	3	3.0	4.6	80.0
	19.2	1	1.0	1.5	81.5
	19.5	2	2.0	3.1	84.6
	20.0	5	5.0	7.7	92.3
	20.5	2	2.0	3.1	95.4
	21.0	1	1.0	1.5	96.9
	21.5	2	2.0	3.1	100.0
	Total	65	64.4	100.0	
Missing	System	36	35.6		
Total		101	100.0		

Spleen length * PV diameter Cross tabulation

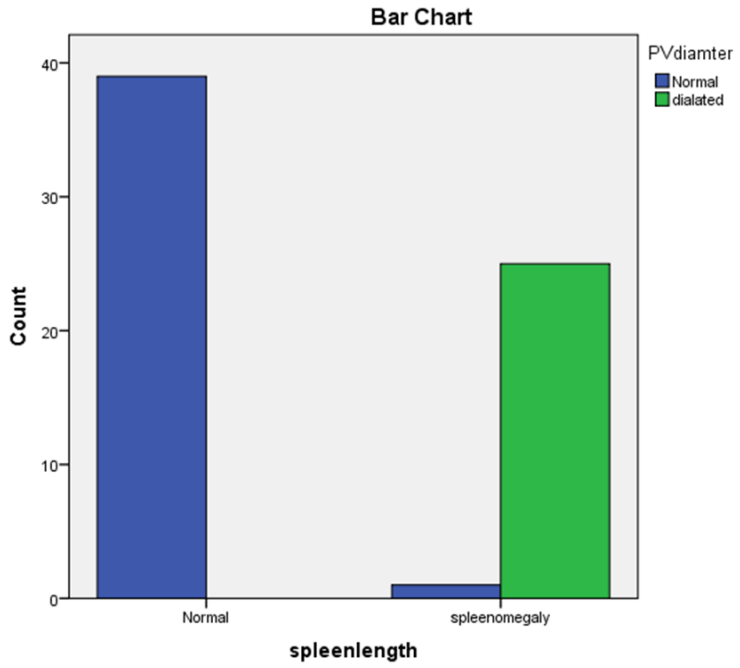
Count

		PV diameter		
		Normal	dilated	Total
Spleen length	Normal	39	0	39
	Splenomegaly	1	25	26
Total		40	25	65

Correlations

		SLCM	PVMM
SLCM	Pearson Correlation	1	.997**
	Sig. (2-tailed)		.001
	N	65	65
PVMM	Pearson Correlation	.997**	1
	Sig. (2-tailed)	.001	
	N	65	65

**. Correlation is significant at the 0.01 level (2-tailed).



Variables Entered/Removed[a]

Model	Variables Entered	Variables Removed	Method
1	SLCM[b]	.	Enter

a. Dependent Variable: PVMM
b. All requested variables entered.

Model Summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.999[a]	.997	.997	.2794

a. Predictors: (Constant), SLCM

ANOVA[a]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	1646.200	1	1646.200	21080.177	.000[b]
	Residual	4.920	63	.078		
	Total	1651.119	64			

a. Dependent Variable: PVMM
b. Predictors: (Constant), SLCM

Coefficients[a]

Model		Unstandardized Coefficients		Standardized Coefficients		
		B	Std. Error	Beta	t	Sig.
1	(Constant)	.097	.098		.989	.326
	SLCM	.993	.007	.999	145.190	.000

a. Dependent Variable: PVMM

Linear regression analysis was performed to determine the effect of spleen length on portal vein diameter in patients with chronic liver disease. The overall regression model was statistically significant ($F(1,63) = 21080.17$, $p < 0.001$), indicating that spleen length is a significant predictor of portal vein diameter. The model demonstrated an extremely high coefficient of determination ($R^2 = 0.997$), showing that 99.7% of the variability in portal vein diameter could be explained by spleen length. The correlation coefficient ($R = 0.999$) indicated a very strong positive relationship between the two variables, suggesting that an increase in spleen size is directly associated with an increase in portal vein diameter.

The unstandardized regression coefficient for spleen length was 0.993 ($p < 0.001$), which means that for every 1 cm increase in spleen length, the portal vein diameter increased by approximately 0.993 mm. The standardized beta value ($\beta = 0.999$) further confirmed that spleen length is a very strong independent predictor of portal vein diameter. The constant was not statistically significant ($p = 0.326$), indicating that the predictive value of the model primarily depends on spleen length. Overall, these findings demonstrate a highly significant and strong positive linear relationship between spleen length and portal vein diameter in patients with chronic liver disease.

DISCUSSION

The purpose of this research was to sonographically assess the relationship between spleen craniocaudal length and portal vein diameter, which has been observed frequently in ultrasound practise. The most accurate and dependable method of determining portal vein diameter is real-time sonography. Ultrasound is frequently used to assess the size and texture of the spleen; it is a simple, non-invasive technique that can be used safely at any age. Ultrasound has been shown in studies to be highly reliable and accurate in measuring the diameter of the portal vein and the size of the spleen. The size can help with disease diagnosis and prognosis, whereas the portal vein diameter can help with splenic-portal conditions.

In our descriptive study total of 65 patients were. Ten patients present with the sharp edge of liver and 8 presented with blunt edge. Out of 65 patient , in 26 patients splenomegaly was scene. It was also observed that 17 patients out of 65 present with dilated portal vein diameter. When portal vein diameter was correlate with the splenomegaly it was observed that 1 patients with splenomegaly have normal portal vein diameter and 25 patients out of 26 splenomegaly patients have increased portal vein diameter. Mean portal vein size was 13.457 mm, minimum size measured was 6.5 mm and maximum 21.5 mm ,the average size of spleen measured in 65

patients was 13.458 cm and minimum size observed was 7.0 cm and maximum size of spleen observed was 21.0 cm. when the correlation of spleen size and portal vein was checked was found between the caudocranial length of the spleen and portal vein diameter with P-value (0.001), the correlation was significant at less than 0.01 level and R-value (0.997), which is near to 1, represents a strong positive relationship and he concluded that Portal vein diameter and splenic craniocaudal length are linearly correlated.

The results of our study were closely related with Muhammad Muazzam research work on Correlation Between Diameter of Portal Vein and Length of Spleen in Patients with Chronic. In his study the average of portal vein diameter was 14.023 ± 2.86 mm, ranging from 7 to 22 mm, while the mean of splenic length was 14.278 ± 2.93 cm, ranging from 7.6 to 21 cm and in our study the average portal vein diameter was 13.45 and splenic length was 13.45 cm. Significant relation (at 0.01 level) was found between portal vein diameter and splenic length, and between splenic length and patient age. But at 0.05 levels significant relation was found between portal vein diameter and patient age among the patients of chronic liver parenchymal disease.[17]

In another study conducted by Shah Zaman on Correlation between portal vein diameter and craniocaudal length of the spleen. A statistically strong correlation was found between the caudocranial length of the spleen and portal vein diameter with P-value (0.000), the correlation was significant at less than 0.01 level and R-value (0.98), which is near to 1, represents a strong positive relationship as compared tour study in which nearly same co relation was find out between spleen length and portal vein diameter, where P-value was 0.001 and the co relation was significant at less than 0.01 level with R-value 0.97. According to his study conclusion Portal vein diameter and splenic craniocaudal length are linearly correlated. An increase in the spleen size may linearly predict an increase in the portal vein diameter in the case of chronic liver parenchymal disease, [18] similarly in our study it was concluded that increase in spleen size linearly predict the portal vein size in case of chronic liver disease.

CONCLUSION(S)

The diameter of the portal vein and the splenic craniocaudal length are linearly related. In the case of chronic liver parenchymal disease, an increase in spleen size may predict an increase in portal vein diameter (liver congestion).

RECOMMENDATIONS

1) More advanced ultrasound an doppler equipment with high frequency process should be used to reduce the false positive and false negative results.
2) Long study duration is recommended for more accurate results.

LIMITATION

The role of confounding factors in determining the mean portal vein diameter variation by age and gender was not controlled for in this study. As a result, interpretation and application of the findings should be done with caution.

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