Original Article

Value of Ascitic Fluid Lipids in the Differentiation between Cirrhotic and Malignant Ascites

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Abstract

Background: The diagnosis of malignant ascites can be difficult. The overall sensitivity of cytology smears for the detection of malignant ascites is 58%–75%. There is a debate about the role of ascitic fluid lipids and their gradients in the differential diagnosis of ascites. This study aims to evaluate the role of ascitic fluid lipids and their gradients in the differentiation of malignant ascites from cirrhotic ascites. **Patients and Methods:** Ninety-six patients were enrolled in this study divided into two groups according to the cause of ascites; Group I: included 48 patients with malignant ascites and Group II: included 48 patients with cirrhotic ascites. **Results:** Ascitic fluid cholesterol, triglycerides, and phospholipids were significantly higher among patients of Group I (77.5 ± 11.5, 82.4 ± 17.04 mg/dl, and 0.92 ± 0.14 mmol/L vs. 27.7 ± 7.5, 56.2 ± 16.2 mg/dl, and 0.33 ± 0.09 mmol/L in succession $P < 0.001$). The cutoff values of cholesterol, triglycerides, and phospholipids were (41.5, 62.5 mg/dl and 0.45 mmol/L successively); they can predict the presence of malignant ascites with sensitivity of (100, 87, and 100% successively) and with specificity of (97.9, 60.4, and 87.5% successively). **Conclusion:** Ascitic fluid lipids are valuable markers in the differentiation of malignant ascites from cirrhotic ascites.

Keywords: *Cholesterol, phospholipids, serum ascites cholesterol gradient, serum ascites triglyceride gradient, triglycerides*

Introduction

Ascites is defined as pathological fluid accumulation within the abdominal cavity. About 50 ml of fluid is normally present in the peritoneal cavity for the purpose of lubrication, but to become clinically evident at least 1500 ml of fluid has to accumulate.[1] Ascites is a common clinical complication to different diseases, the most common cause of ascites is liver cirrhosis (80%) followed by cardiac ascites (10%), tuberculous peritonitis (2%), malignant ascites, nephrotic syndrome, and others (3%).[2]

Malignant ascites results from the accumulation of fluid within the peritoneal cavity caused by the intraperitoneal spread of tumor cells, massive liver metastasis and/or lymphatic obstruction and can be caused by a number of different cancers including ovarian, gastric, endometrial, breast, colon, and pancreatic.[3] The diagnosis of malignant ascites can be difficult. The sensitivity of ascitic fluid cytology in peritoneal carcinomatosis is approximately 100%. However, because not all cases of malignant ascites are associated with peritoneal carcinomatosis, the overall sensitivity of cytology smear for the detection of malignant ascites is 58%–75% hepatocellular carcinoma (HCC) rarely metastasizes to the peritoneum.[4]

Many studies were designed to investigate the role of ascitic fluid lipids in the differentiation of malignant and cirrhotic ascites. The reason for that is, peritoneal carcinomatosis was found to cause excessive secretion of plasma lipoprotein in the peritoneal cavity.[5] Despite that, Runyon and the AASLD practice guidelines commitee, in 2009 updated report on the diagnosis and management of ascites, classified ascitic fluid lipids and especially cholesterol as being useless at the differentiation between malignant and cirrhotic ascites.[6]

In correspondence to that report, Gerbes and Jüngst published a note in 2009[7] that claims that Runyon ignored many studies that not only emphasized the role

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of cholesterol and ascitic fluid lipids in the diagnosis of malignant ascites but also suggested possible mechanisms for the increase in ascitic fluid lipids in this condition.[7-13]

Aim of the work

This study aimed at evaluating the diagnostic reliability of ascitic fluid lipids and the serum ascites lipid gradients in differentiation between malignant and cirrhotic ascites.

Patients and Methods

This case–control stud was performed in tropical medicine, radiotherapy, biochemistry, and clinical pathology departments Zagazig University Hospitals. It included 96 patients with ascites divided into two groups according to the cause of ascites Group I included 48 patients with malignant ascites. Group II included 48 patients with cirrhosis and portal hypertension-related ascites. The patients' diagnosis was confirmed by the combination of clinical, radiological, laboratory data, and cytological study of ascitic fluid.

The exclusion criteria were as follows: patients <18 years, patients who did not give consent to participate in the study, patients with malignant ascites with unknown primary tumor, patients with complicated portal hypertension associated ascites such as spontaneous bacterial peritonitis with ascitic fluid total leukocytic count >500 cell/μL or polymorphonuclear count >250 cell/μL, patients with other causes of ascites such as cardiac, renal, nutritional, local peritoneal inflammatory conditions such as tuberculosis, chylous ascites, pancreatic disease, Meig syndrome, or ovarian hyperstimulation syndrome.

The patients were subjected to the following: full history taking, thorough clinical examination, pelvic-abdominal ultrasonography, and subsequent necessary radiological investigations such as tri-phasic computed tomography, magnetic resonance imaging, magnetic resonance cholangiopancreatography, upper or lower gastrointestinal endoscopy examination to confirm their conditions when needed. Routine laboratory investigations were also done to the patients such as complete blood count, liver and kidney functions tests, viral markers, lipid profile, as well as routine cytological and biochemical examination of ascitic fluid to assess ascitic fluid glucose, protein, albumin, serum ascites albumin gradient (SAAG) as well as ascitic fluid cholesterol, triglycerides, and phospholipids. The ascitic fluid cholesterol, triglycerides, and phospholipids were measured using enzymatic colorimetric method, cholesterol serum level, triglycerides mono SL and phospholipids kits, Biodiagnostic, diagnostic and research reagents, Egypt. They were analyzed using Microlab 300, semi-automated, chemistry analyzer, and VITAL scientific.

The serum and ascitic fluid samples were taken at the same time, and calculation of serum ascites cholesterol gradient (SACG) was calculated by subtracting the ascitic fluid cholesterol concentration from the serum cholesterol concentration. The serum ascites triglycerides gradient was also calculated by subtracting the ascitic fluid triglyceride concentration of the serum triglycerides concentration.

Statistical method

The data were processed using SPSS Epi info version 16 (IBM company, Armonk, New York, USA). The quantitative data were represented as mean and standard deviation, while the qualitative data were represented as number and percentage. The comparison of variables was done using *t*-test for normally distributed quantitative data and Mann–Whitney test for data that were not normally distributed. The comparison of categorical data was done using Chi-square. We used multivariate regression and binary logistic regression analysis to detect the independent correlations between variables and eliminate the effects of possible confounding factors. To study the diagnostic performance, we used receiver operator characteristics curve to calculate cutoff value sensitivity, specificity.

Results

Table 1 represents a summary of the demographic and clinical data of both groups. It shows that there were no significant differences between the studied groups as regards age, gender distribution, viral markers, or grade of ascites. Table 1 also shows that Group I had significantly higher frequency of jaundice than Group II (97.9% vs. 81.2% $P = 0.008$). The frequency of hepatomegaly was significantly higher among Group I as well (35.4% vs. 16.7% $P = 0.04$). The frequency of splenomegaly was higher among patients in Group II (79.2% vs. 27.1 $P < 0.001$) [Table 1].

Table 2 represents the different types of malignancies in Group I. It shows that the most common malignancy in Group II was ovarian malignancy (18.2%) followed by HCC and colorectal carcinoma (14.6%). Pancreatic malignancy comes in third in the list with a frequency of 12.5%. The least common tumor was cancer cervix (2.1%) [Table 2].

Table 3 represents a comparison between the studied groups as regards hematological and serum biochemical parameters. It shows that the Group II had significantly lower White blood cells (WBCs) and platelet counts (5.28 ± 2.56 and $136.9 \pm 110.6 \times 10^3$ cells/μL in Group II vs. 7.56 ± 2.55 and $280.5 \pm 120.3 \times 10^3$ cells/μL in Group I $P < 0.001$). It also shows that serum albumin was also significantly lower in Group II (2.9 ± 0.28 versus 3.5 ± 0.46 g/dl in Group II $P < 0.001$). The total protein level was also significantly lower in Group II (6.4 ± 1.01) versus 6.9 ± 0.76 g/dl in Group II $P = 0.01$.

Comparison between the studied groups also revealed that although alanine aminotransferase showed no significant difference, aspartate aminotransferase level was significantly higher in Group I (66.6 ± 32.9 vs.

Table 1: Difference among both studied groups as regard demographic and clinical data (*n*=48)

	Group I	Group II	*t*-test	*P* (S)
Age, mean±SD	57.2±9.3	55.4±9.7	0.906	0.367 (NS)
	Group I, *n* (%)	**Group II, *n* (%)**	**χ^2/ fisher exact**	***P***
Gender				
Male	23 (47.9)	18 (37.5)	1.06	0.409 (NS)
Female	25 (52.1)	30 (62.5)		
Viral markers				
HCV	14 (29.2)	23 (47.9)	Fisher	0.27 (NS)
HBV	5 (10.4)	3 (6.4)		
Jaundice	47 (97.9)	39 (81.2)	7.14	0.008 (S)
Lower limb edema	17 (35.4)	8 (16.7)	4.38	0.04 (S)
Hepatomegaly	17 (35.4)	8 (16.7)	4.38	0.04 (S)
Splenomegaly				
No	35 (72.9)	10 (20.8)	25.6	<0.001 (HS)
Mild	12 (25)	32 (66.7)		
Moderate	1 (2.1)	5 (10.4)		
Huge	0 (0.0)	1 (2.1)		
Ascites				
Mild	16 (33.3)	17 (35.4)	0.173	0.913 (NS)
Moderate	22 (45.8)	20 (41.7)		
Tense	10 (20.8)	11 (22.9)		

NS: Nonsignificant *P* value >0.05, S: Significant *P* value <0.05, HS: Highly significant *P* value <0.001, SD: Standard deviation, HCV: Hepatitis C virus, HBV: Hepatitis B virus

Table 2: Different types of malignancy among Group I (*n*=48)

Type of malignancy	Group I, *n* (%)
GIT and liver	
HCC	7 (14.6)
Pancreatic	6 (12.5)
Esophagus	2 (4.2)
Stomach	5 (10.4)
Colorectal	7 (14.6)
Gynecological	
Breast	3 (6.3)
Ovarian	9 (18.8)
Cervix	1 (2.1)
Endometrial	3 (6.3)
Others	
Lung	3 (6.3)
Prostate	2 (4.2)

HCC: Hepatocellular carcinoma, GIT: Gastrointestinal tract

51.6 ± 24.4 IU/L $P = 0.03$). The creatinine level was also significantly higher in Group I (1.3 ± 0.36 vs. 1.03 ± 0.47 mg/dl $P = 0.003$) [Table 3].

Serum lipid profile

Comparison between the studied groups as regards serum lipids show that Group I had significantly higher cholesterol and triglyceride levels than Group II (170.9 ± 15.5 and 126.4 ± 14.8 mg/dl successively vs. 131.1 ± 3.5 and 117.8 ± 7.4 mg/dl $P < 0.001$) [Table 3].

Ascitic fluid biochemical parameters

Table 4 represents a comparison between the studied groups as regards ascitic fluid biochemical parameters. It shows that group I had significantly lower glucose level than Group II (85 ± 5.01 vs. 118.8 ± 8.9 mg/dl $P < 0.001$). Group I have also significantly higher protein and albumin concentration in ascitic fluid than in Group II (3.7 ± 0.41 and 2.7 ± 0.46 vs. 2.11 ± 0.31 and 1.0 ± 0.31 g/dl $P < 0.001$). Consecutively, SSAG was significantly lower in Group I than in Group II (0.81 ± 0.11 vs. 1.92 ± 0.29 g/dl $P < 0.001$) [Table 3].

Ascitic fluid lipids

Table 4 shows also that the ascitic fluid cholesterol and triglycerides were significantly higher in group I patients than in Group II (77.5 ± 11.5 and 82.4 ± 17.04 mg/dl vs. 27.7 ± 7.5 and 56.2 ± 16.2 in succession $P < 0.001$). Moreover, phospholipids concentration on ascitic fluid was significantly higher in Group I than in Group II (0.92 ± 0.14 vs. 0.33 ± 0.09 mmol/L $P < 0.001$) [Table 3].

Multivariate analysis

Table 5 represents multivariate logistic regression to assess the independent correlation between ascitic fluid lipids and each other. It shows that ascitic fluid cholesterol was significantly correlated to ascitic fluid phospholipids ($r = 6.8$ $P = 0.01$). It also shows the correlation between the ascitic fluid lipids and serum lipids. It reveals the positive correlation between the serum cholesterol and ascitic fluid cholesterol ($r = 0.9$ $P < 0.001$). It also shows that ascitic fluid phospholipids are significantly positively correlated to serum cholesterol ($r = 0.5$ $P < 0.001$). There is also a significant positive correlation between serum and ascitic fluid triglycerides ($r = 1.04$ $P < 0.001$) [Table 4].

There were so many significant differences between the studied groups. Applying binary logistic regression was important to verify the variables that could be independent predictors of malignant ascites. Table 6 shows that the only independent predictors of malignant ascites in our study were serum cholesterol, serum triglycerides, and ascitic fluid cholesterol [Table 5].

The diagnostic reliability of ascitic fluid lipids

Blotting receiver operating characteristics (ROC) curve for ascitic fluid cholesterol revealed that at a cut off value of 41.5 mg/dl, it can diagnose malignant ascites with sensitivity of 100% and specificity of 79.9% area under the curve (AUC) = 1. The clinical performance of SACG was less satisfactory; it shows that at a cut off value of 101.5 mg/dl or less SACG can predict the presence of malignant ascites with sensitivity of 25% and specificity of 50% AUC = 0.2 [Table 6].

Table 3: Differences between the studied groups as regards complete blood count and serum and ascitic fluid biochemical parameters (*n*=48)

	Group I	Group II	*t*-test\MW*	*P*
Hemoglobin (g/dl)	9.77±1.72	9.67±1.85	0.265	0.97 (NS)
WBC's (×10³ cell/µL)	7.56±2.55	5.28±2.56	4.37*	<0.001 (HS)
Platelet (×10³ cell/µL)	280.5±120.3	136.9±110.6	5.82*	<0.001 (HS)
Serum albumin (g/dl)	3.5±0.46	2.9±0.28	7.5	<0.001 (HS)
Serum protein (g/dl)	6.9±0.76	6.4±1.01	2.6	0.01 (S)
Serum bilirubin (mg/dl)	7.2±9.76	2.8±2.15	3.9*	<0.001 (HS)
ALT (IU/L)	43.9±29.5	35.6±19.4	1.11*	0.279 (NS)
AST (IU/L)	66.6±32.9	51.6±24.4	2.18*	0.03 (S)
INR	1.4±0.22	1.5±0.32	1.15	0.255 (NS)
Serum creatinine (mg/dl)	1.3±0.36	1.03±0.47	3.04*	0.003 (S)
Serum cholesterol (mg/dl)	170.9±15.5	131.1±3.5	17.3	<0.001 (HS)
Serum triglycerides (mg/dl)	126.4±14.8	117.8±7.4	3.6	0.001 (HS)
Ascitic fluid biochemical parameters				
Glucose (g/dl)	85±5.01	118.8±8.9	22.8	<0.001 (HS)
Protein (g/dl)	3.7±0.41	2.11±0.31	22.1	<0.001 (HS)
Albumin (g/dl)	2.7±0.46	1.0±0.31	21.1	<0.001 (HS)
SAAG (g/dl)	0.81±0.11	1.92±0.29	24.5	<0.001 (HS)
Cholesterol (mg/dl)	77.5±11.5	27.7±7.5	25.1	<0.001 (HS)
SACG (mg/dl)	93.02±8.97	103.4±7.96	6.01	<0.001 (HS)
Triglycerides (mg/dl)	82.4±17.04	56.2±16.2	7.7	<0.001 (HS)
SATG (mg/dl)	44.9±14.96	61.7±11.9	6.1	<0.001 (HS)
Phospholipids (mmol/L)	0.92±0.14	0.33±0.09	24.3	<0.001 (HS)

NS: Nonsignificant *P* value >0.05, S: Significant *P* value <0.05, HS: Highly significant *P* value <0.001, WBCs: White blood cells, SAAG: Serum-ascites albumin gradient, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, INR: International normalized ratio, MW: Mann whitney test,
SATG: Serum ascites triglycerides gradient

Table 4: Multivariate regression showing the correlation between the ascitic fluid lipids and serum lipids

	Ascites cholesterol			Ascites triglycerides			Ascites phosophlipids		
	R	SE	*P*	*R*	SE	*P*	*R*	SE	*P*
Serum cholesterol	0.926	0.096	<0.001 (HS)	−0.148	0.415	0.658	0.511	0.231	<0.001 (HS)
Serum triglycerides	0.08	0.112	0.159	1.04	0.144	<0.001 (HS)	0.000	0.001	0.342
Ascites cholesterol									
Ascites triglycerides	−0.023	0.023	0.653						
Ascites phospholipid	6.82	5.37	0.01 (S)	13.6	32.6	0.677			
r, r^2		0.979, 0.958			0.926, 0.863			0.976, 0.956	
ANOVA (*P*)		<0.001 (HS)			<0.001 (HS)			<0.001 (HS)	

HS: Highly significant, S: Significant, SE: Standard error

As for ascitic fluid triglycerides, they can predict the presence of malignancy in ascitic fluid at a cutoff value of 62.5 mg/dl or more with sensitivity of 87.9% and specificity of 60.4% AUC = 0.8. However, serum ascites triglycerides gradient was less reliable as a diagnostic tool the reason for that is, it can predict malignant ascites with sensitivity of 43.8% and specificity of 12.5% at a cutoff value of 47.5 mg/dl or less AUC = 0.1 [Table 6].

Ascitic fluid phospholipids performance was also proved to be reliable in the diagnosis of malignant ascites. It can predict the presence of malignant ascites at a cutoff value of 0.45 mmol/L with sensitivity of 100% and specificity of 87.5% AUC = 1 [Table 6].

Discussion

This study was designed to evaluate the role of ascitic fluid lipids and serum ascites lipid gradient in differentiation between cirrhotic and malignant ascites and as diagnostic predictors of malignant ascites. For this reason, the study included two groups of patients representing the two types of ascites under investigations.

Many differences were noticed between the studied groups. As regards the clinical parameters, Group I had significantly higher frequency of jaundice than Group II along with significantly higher bilirubin level. This finding may be explained by the fact that HCC is the second-most common

Table 5: Binary logistic regression analysis for significant independent predictors of malignant ascitic fluid

Independent variables	*B*	SE	Wald	Significant	OR (95% CI)
WBCs	−0.117	3.21	4.11	0.732	0.89 (0.990-1.65)
Platelets	−0.005	2.93	5.23	1.000	0.995 (1.52-4.12)
Total bilirubin	0.051	1.68	2.12	0.643	1.052 (1.41-2.32)
Serum albumin	5.261	3.92	0.101	1.000	1.68 (1.12-2.24)
Serum total protein	0.989	5.67	0.934	0.101	2.6 (0.941-2.86)
AST	−0.036	6.59	7.43	0.667	0.96 (0.132-2.12)
Creatinine	0.2	1.76	3.23	0.562	1.2 (0.541-1.32)
Serum cholesterol	0.143	1.03	2.112	0.02 (S)	1.14 (0.321-1.86)
Serum triglycerides	1.091	0.67	1.010	0.03 (S)	1.16 (0.301-2.85)
Ascitic fluid glucose	0.311	9.51	7.53	0.222	1.65 (0.122-2.62)
Ascitic fluid albumin	−6.58	3.83	11.23	0.523	0.001 (0.000-0.232)
Ascitic fluid protein	−5.95	1.65	4.76	0.543	0.003 (0.000-0.432)
Ascitic fluid cholesterol	−0.434	3.58	3.650	0.03 (S)	1.648 (0.201-2.42)
Ascitic fluid triglycerides	0.118	9.05	2.12	0.342	1.126 (0.342-1.843)
Ascitic fluid phospholipids	−13.5	2.63	7.0	1.000	0.000 (0.250-0.564)
SAAG	−32.5	1.601	5.02	1.000	0.000 (0.054-0.523)
SACG	2.34	0.234	4.23	0.754	0.541 (0.02-0.832)
SATG	0.234	0.854	2.45	0.986	0.03 (0.002-0.643)

S: Significant *P* value <0.05, SE: Standard error, OR: Odds ratio, CI: Confidence interval, WBCs: White blood cells, SAAG: Serum-ascites albumin gradient, SACG: Serum ascites cholesterol gradient, AST: Aspartate aminotransferase, SATG: Serum ascites triglycerides gradient

Table 6: Clinical performance of ascitic fluid lipids and their gradients as predictors of malignant ascites

Variables	Cutoff	AUC	*P*	PPV (%)	NPV (%)	Sensitivity (%)	Specificity (%)	Accuracy (%)
Cholesterol	>41.5	1.0	<0.001	98	100	100	97.9	98.9
SACG	<101.5	0.205	0.05	32.7	23.7	25	50	29.2
Triglycerides	>62.5	0.848	<0.001	68.8	82.9	87.5	60.4	73.9
SATG	<47.5	0.198	<0.001	33.3	18.2	43.8	12.5	28.1
Phospholipids	>0.45	1.00	<0.001	88.8	100	100	87.5	88.5

SACG: Serum ascites cholesterol gradient, AUC: Area under the curve, PPV: Positive predictive value, NPV: Negative predictive value, SATG: Serum ascites triglycerides gradient

tumor in our study, and that massive liver metastasis is a common finding among patients in Group I. Another thing is that chemotherapy received by those patients also had its hepatotoxic and cholestatic effects.

Lower limb edema was also significantly more frequent in group I than in group II. This can be explained by what was said by Saif *et al*.,[14] that the pathological fluid regulation, electrolyte disturbances, and protein leakage inside the peritoneal cavity lead the patient to anasarca.[14] The hepatomegaly was also more frequent among patients with malignant ascites (36% vs. 17%). This agrees with Kufe *et al*.,[3] who said that 36% of patients develop malignant ascites because of HCC, massive liver metastasis, or combined peritoneal carcinomatosis and liver metastasis.[3] On the other hand, the frequency of splenomegaly was higher in patients with cirrhotic ascites (Group II). Splenomegaly is caused by portal hypertension, which is the cause of ascites in this group.

It is also worth mentioning that patients of Group II had significantly lower WBC's and platelet counts. This finding can be a consequence of the larger spleen size of this group.

In our study, the most common malignancy causing ascites was ovarian malignancy. This agrees with Barni *et al*.[15] The second-most common malignancy was HCC. This comes in agreement with Kufe *et al*.,[3] who said that 13% of malignant ascites develop due to HCC alone.

In our study, the, gastrointestinal tract tumors (esophagus, colorectal, and gastric) caused malignant ascites in 29.5%. This agrees with Smith and Jayson,[16] who said that a large percentage of patients with GIT malignancy develop ascites at a certain stage of their disease.[16] The breast and lung tumors were the only extra-abdominal tumors in our study that caused malignant ascites. This agrees with Saif *et al*.,[14] who said that breast and lung cancer are the main extra-abdominal tumors causing malignant ascites.[14]

Serum albumin and serum total protein are significantly lower in Group II. This is due to impaired synthesis by the diseased liver in this group. It is also worth noticing that, both groups had mean serum total protein of <7 mg/dl, this may be a consequence of protein-energy malnutrition suffered by those patients. This protein-energy malnutrition can be an additional factor that can worsen the ascites condition and the response to conventional treatment.

The serum lipids were significantly higher in Group I patients. This comes in agreement with Gerbes *et al*.,[10] who said that patients with cirrhosis had significantly lower serum cholesterol level.[10]

As regards the biochemical parameters of ascitic fluid, the comparison between the two groups revealed highly significant differences. Malignant ascites had significantly lower glucose concentration. This agrees with Runyon *et al*.,[17] who said that glucose concentration in malignant ascites falls below 100 mg/dl.[17]

The total protein and albumin were significantly higher in malignant ascites than in cirrhotic ascites, and SAAG was also significantly lower in malignant ascites. This agrees with Runyon *et al*.,[18] who said that ascites were initially classified to exudate and transudate according to the protein level and that protein level >2.5 g/dl excludes cirrhotic ascites and that this concept is now replaced by high SAAG >1.1 and low SAAG <1.1 ascites.[18] The ascitic fluid lipids levels were significantly higher in malignant ascites; this finding agrees with Jüngst *et al*.,[8] Gerbes *et al*.,[10] and Gupta *et al*.,[13] who said that the ascitic fluid cholesterol was higher in patients with malignant ascites.[8,10,13] The serum ascites gradients of both cholesterol and triglycerides were found to be significantly lower in malignant ascites group.

Applying multivariate regression model revealed that ascitic fluid cholesterol and triglycerides levels were significantly correlated to the serum cholesterol and triglycerides levels successively, this finding agrees with Mortensen *et al*.,[9] who said that the increased ascitic fluid cholesterol is due to increased vascular permeability not due to increased cellular components in malignant ascites.[9] It also agrees with Jüngst *et al*.,[11] who said that the ascitic fluid cholesterol cannot differentiate positive and negative cytology ascites.[11] We also found that the correlation between the ascitic fluid cholesterol and the ascitic fluid triglycerides level was not significant. This agrees with Jüngst *et al*.,[8] who said that the correlation between cholesterol and triglycerides in ascitic fluid was poor.[8] On the other hand, the ascitic phospholipids level was significantly correlated to ascitic fluid cholesterol and to serum cholesterol as well. Applying the binary logistic regression, we found that the only independent predictors of malignant ascites in our study were serum lipids and ascitic fluid cholesterol.

On plotting the ROC curve for cholesterol level, we found that it can detect malignant ascites at a cut off value of 41.5 mg/dl with sensitivity of 100% and specificity of 97.9%. This comes in agreement with Dessí *et al*.,[12] who said that cholesterol is the most accurate test for malignancy with an accuracy of 97%.[12]

In our study, we also found that the accuracy of triglycerides level in ascitic fluid was lower than that of cholesterol. The cutoff value was 62.5 mg/dl, and sensitivity was 87% and specificity was 60%. This performance was not as accurate as that of cholesterol. However, this disagrees with Jüngst *et al*.,[8] who said that the triglyceride level in ascitic fluid was of no practical value. Jüngst *et al*.'s study did not provide a clear explanation of this finding.[8]

The SACG and SATG showed to be less reliable as a diagnostic marker. This finding is supported by Jüngst *et al*.,[8] who said that the serum ascitic fluid lipid gradients did not provide better discrimination of malignant ascites.[8]

Ascitic fluid phospholipids were proved also to be accurate predictors of malignant ascites. At cutoff value of 0.45 mmol/L, they can diagnose malignant ascites with sensitivity of 100% and specificity of over 80%. This performance can be explained by the significant correlation; it has with ascitic fluid cholesterol level. This also agrees with Jüngst *et al*.,[8] who said that the phospholipids can predict the presence of malignant ascites with sensitivity and specificity that exceed 80%.[8]

Conclusion

Ascitic fluid lipids are significantly higher in malignant ascites than in cirrhotic ascites, and their ascitic fluid levels are correlated to their serum levels. Ascitic fluid lipids are valuable markers for differentiation of malignant ascites from cirrhotic ascites.

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Conflicts of interest

There are no conflicts of interest.

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