REVISIT TO SONOGRAPHY IN DENGUE MANAGEMENT: A REVIEW

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Abstract

Dengue infection (DF) is a mosquito born endemic disease with rising trend in Sri Lanka. Diagnosis of DF is clinical, supported by investigations. The phase of illness, warning signs, hydration and hemodynamic status of the patient determine the need for hospitalization and fluid management. Fluid management is crucial in prognosis. Ultrasonography (USG) is empirical to arrive at management decisions; exact role of USG is reviewed in this article.

Gall bladder wall thickening (GBWT) and pericholecystic fluid are early and consistent sonographic features of dengue haemorrhagic fever, whilst hepatomegaly, splenomegaly, pleural effusions and pericardial effusions are the other features. Among various morphological appearances of GBWT, “honey comb” and “reticular” patterns are most frequent in severe DF. Morphology of GBWT is positively correlated to the disease progression and recovery, thus considered to be a useful sonographic signs in predicting the disease progression. Plasma leakage in DF is frequently depreciated by measuring the ascetic fluid volume, hence not recommended to use as a guideline in fluid management. USS measured inferior vena cava diameter (IVCd) and inferior venacava collapsibility index (IVC-CI) are of value in assessing intra vascular fluid volume. Calculation of IVC-CI is possible with “(IVCdmax – IVCdmin)/IVCdmax” formula. Even though, the liver involvement in DF is yet to be understood, hepatomegaly with elevated liver enzymes are the commonest manifestations. Liver involvement in DF could be ranged from mild transient liver cell dysfunction to acute fulminant hepatic failure.

Bleeding tendency in DF, limits histopathological evaluation of the liver, hence pathogenesis is poorly understood. Future non invasive (sonographic) studies would be of value in the liver assessment. Further studies are recommended to assess IVC-CI pertaining to the volume status and patient outcome to postulate new fluid management guidelines.

Key words: dengue, ultrasound, fluid management, gall bladder, IVC, liver

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Introduction

Dengue infection (DF) is a mosquito born viral disease. Early this century, DF was a periodic disease with predictable out breaks in Sri Lanka; however now the disease shows a more endemic pattern with an inclining trend in incidence. Suspected dengue cases reported in 2017 were 186 101; nearly a 4 fold rise of annual incident compared to the last six years^{1,2}.

Diagnosis of DF is made on clinical findings, supported by investigations. DF could be non severe or severe infection with or without complications. Three phases are described in severe dengue infection; febrile, critical and recovery phase³. The “warning signs” of severe dengue infection are abdominal pain, tenderness, persistent vomiting, clinical fluid accumulation, mucosal bleeding, lethargy, restlessness, liver enlargement more than 2 cm, rise of haematocrit and rapid decrease in platelet count. “Severe infection” is defined by significant plasma leakage and shock (DSS); fluid accumulation with respiratory distress; bleeding manifestations or organ impairment. Organ impairment in DF is manifested by increased liver transaminases (AST or ALT more than

1000 IU/l), altered consciousness and cardiac or other organ failures².

Hospitalization as well as fluid management is mainly decided by the phase of the illness, warning signs, hydration and hemodynamic status of patient. Optimum fluid management is crucial in DF; associates with better patient outcome². Common complications that are seen in critical phase (CP) are bleeding manifestations and organ impairments³.

Role of ultrasonography is imperative in disease stage identification and in assessment of the haemodynaemic status. This article reviews the available literature and management guidelines to re-evaluate the role of sonography in DF.

Sonographic features of dengue infection

Clinically, DF is suspected when a patient is presenting with fever during an epidemic or with a travel history to an endemic area. For diagnosis, investigation findings are complementary to clinical assessment; common laboratory investigation findings are neutropenia, lymphocytosis, thrombocytopenia and elevated liver enzymes².

Sonographic features are helpful to diagnose and to identify phase of the

illness. Venkata Sai et al.⁴ found DF patients initially (day 2-3 of illness) had gallbladder wall thickening (GBWT) & pericholecystic fluid (100%), hepatomegaly (21.8%), splenomegaly (6.25%), right side pleural effusions (6.25%). Subsequent assessment of same patients (day 5-7 of the illness) revealed an increasing frequency of the former signs with emergence of new findings: ascites (53.17%), left side pleural effusion (21.8%) and pericardial effusion (28.12%). When DF patients were assessed in 5-7 days of illness GBWT (100%) and right side pleural effusion (96.46%) was most common, followed by left sided pleural effusion (66.07%), hepatomegaly (60.9%), splenomegaly (30.35%), ascites (7.14%) and pericardial effusion (28.57%). A dengue serology negative subset of fever patients had transient GBWT (35%). Hence, Venkata Sai et al.⁴ reported the gall bladder wall thickening as the most consistent USG feature of DF.

Michels M. et al.⁵. (n=66) grouped sero positive dengue patients in to non severe (83%) and severe (17%) infections according to WHO classification²; GBWT, pleural effusion and ascites were found in both severe and non severe groups. The findings became more frequent in subsequent scans particularly when the infection was severe. in some studies

hepatomegaly was reported as the most common finding, followed by GBWT.⁶ Sonographic findings of DF are summarized in Table 01. All mentioned studies were performed with 6 hours fasting to optimize gallbladder visualization.

To sum up, GBWT and pericholecystic fluid are early and most consistent findings of DF, notably in severe infection. With disease progression, other findings become more frequent; right side pleural effusions and ascites being commonest. Transient GBWT is a non specific feature that can be seen in other infections as well⁵.

Gallbladder morphology in dengue fever

As described earlier GBWT and pericholecystic fluid collections were frequent in DF. Wall thickness more than 3mm was considered as GBWT. Transient uniform GBWT (Figure 2) is a non specific finding seen in biliary pathologies and other systemic diseases; common causes are listed in table 3⁸. Thus, uniform GBWT is not considered as a reliable sign of plasma leakage.

Reticular pattern of GBWT (Figure 2) is described as a typical sonographic feature of plasma leakage in severe dengue infection⁹.

Different morphological patterns of GBWT are described in figure 2¹⁰. “Sachar and Sunder’s sign” or “honeycomb pattern” of GBWT (Figure 2) is a confirmatory sign of DHF that was seen in 95% of patients¹⁰.

Parmar JP et al.¹¹ studied DF sero positive patients (n=93), stratified into three groups: DF without warning signs; with warning signs; severe disease, to find an increasing frequency of GBWT with disease severity (96.5% in severe disease). Rest of the morphological patterns of GBWT are honeycomb pattern; asymmetric thickening; central hypoechoic zone separated by two echogenic layers (“tram track sign”); uniform echogenic pattern (figure 2). Parmar JP et al. also found “honey comb” pattern frequently in severe DF, which was absent in DF without warning signs. A dynamic pattern in GB morphology was observed during the clinical course of DF; honey comb pattern has changed to diffuse echogenic pattern during recovery and vice versa with clinical deterioration. GBWT is positively correlated with the size of pleural effusion or with the ascites¹².

In conclusion, pattern of GBWT can be used to predict the severity, recovery or the deterioration of DF. Additionally, gall bladder thickening can be used to predict

the critical phase as GBWT precedes the development of ascites and pleural effusions.

Assessment of plasma leakage in dengue fever

Febrile phase of dengue haemorrhagic fever (DHF) is usually for 2-7 days; with defervescence the critical phase starts that roughly last for 48 hours. In critical phase of illness localized plasma leakage occurs; mainly in to the peritoneal and pleural cavities. The plasma leakage lasts approximately for 48 hours; resolution is spontaneous and rapid. Mortality in DF is usually related to delayed diagnosis and treatment of plasma leakage.¹³ Fluid management being mainstay of treatment should be carefully monitored, to administer correct amounts of fluid. Excessive fluid can lead to serious complications such as heart failure and pulmonary edema, while inadequacy results in intra vascular volume depletion; dengue shock syndrome (DSS)¹³.

Plasma leakage in to the peritoneal cavity is usually followed by right sided pleural effusion¹⁴. Ascites, pleural effusions and GBWT are less frequent among DSS patients compared to DHF patients ($p>0.05$; Table 4). In another study, pleural effusion was the most frequent indicator of fluid leakage in DHF¹².

Severity of the plasma leakage is positively correlated with the severity of the disease. Pericardial effusion is an uncommon finding in severe DHF¹⁵. Table 4 describes sonographic patterns of plasma leakage in DHF & DSS.

Srikiatkhachorn et al¹². studied children suspected to have DHF (below 15 years, n=139) to delineate the timing, progression and location of plasma leakage by performing chest and abdominal USG daily; right lateral decubitus chest X ray (CXR) findings were compared with erect chest sonographic findings (Table 5). During defeverescence phase pleural effusions were frequent. The magnitude of plasma leakage was positively correlated with the disease severity and with the degree of haemoconcentration.

Sonography in fluid management in dengue

In critical phase, plasma leakage results in intra vascular fluid depletion and systemic hypotension. The known effective treatment to reduce mortality in DSS is optimum fluid resuscitation; however, ideal fluid dosage is yet to be studied in clinical trials. Excessive fluid administration can lead to fluid overload and pulmonary edema. Therefore, fluid resuscitation should be done with extreme care by close monitoring of vital

parameters (pulse rate, blood pressure, pulse pressure and urine output) to determine the required fluid volume. Fluid requirements are reduced during recovery¹⁶.

Hematocrit is widely used to diagnose plasma leakage; a 20% rise in hematocrit values from the base line was defined as hemoconcentration. However, this was not very reliable parameters to predict plasma leakage or the severity of plasma leakage; many other causes such as fever, dehydration and internal or external haemorrhages are associated with high hematocrit values¹².

Free peritoneal fluid with reference to fluid management

Value of sonography to diagnose free peritoneal fluid and pleural effusions in DHF is a widely discussed topic. Ultrasonography could be used to assess the progression of fluid accumulation, hence the severity of plasma leakage.¹³ Sensitivity of detecting ascites using sonography is influenced by the patient position¹⁷.

Verguts J. et al. measured free peritoneal fluid with transvaginal ultrasound scans (n=13) and found any measurement of ascetic fluid in between bowel loops could be an underestimation of the real volume¹⁸.

Though it is possible to determine the temporal progression of plasma leakage by measuring ascetic fluid volume, quantification of leakage in DHF appears to be inaccurate; it frequently underestimates the severity of plasma leakage.

Pleural effusions with reference to fluid Management

The size of the pleural effusion is commonly measured in thoracic ultrasonography. Balik et al.¹⁹ established a positive correlation between pleural fluid volume and the pleural separation by comparing aspirated pleural fluid volume and sonographically measured pleural separation (n=81). The pleural fluid volume can be estimated with a simple formula “pleural fluid volume (ml) = 20 x maximum pleural separation (mm)”. Therefore, quantitative assessment of fluid leakage in to the pleural cavity is possible.

In DHF cumulative plasma leakage is the sum of pleural and peritoneal fluid volumes, which likely to be underestimated when measured ultrasonographically. Thus, ascetic and pleural fluid quantification is not recommended to predict intra vascular fluid deficit.

IVC diameter with reference to fluid management

Inferior venacava diameter (IVCd) is commonly measured (by sonography) in intensive care (ICU) settings to assess the intra vascular volume status. IVCd and inferior vena cava collapsibility index (IVC-CI) are positively correlated with the volume status of the patient and with central venous pressure of spontaneous breathing patients.²⁰ As DHF patients are with haemorrhagic tendency, a non invasive method is preferable to assess volume and the hemodynaemic status^{2,3,4,7}.

IVC-CI is calculated using IVCd that is measured sonographically; in long axis sub costal approach; ideally with 3.5 – 4 MHz probe: intra hepatic portion and right atrial entry point of IVC need to be visualized; measurements to be taken 2cm caudal to right atrial entry point of IVC. The time-motion curve (M-mode) should be recorded for at least for 20s over a single respiratory cycle (in spontaneous breathing); maximum (IVCdmax) and minimum (IVCdmin) diameters of IVC needs to be measured from time motion curve. The IVC-CI equals to $(IVC_{max} - IVC_{min})/IVC_{max}$ (Figure 3)²⁰.

When IVC-CI is >50%, intravascular volume depletion is a possibility²⁰. IVCd

of normotensive patients was recorded as 1.07-2.90 cm; in hypotensive patients IVCd was recorded as 0.56-1.55 cm²¹.

Raman R et al.²² studied 46 children with DF, grouped in to DSS (n=40) and DHF with warning signs (n=6) to evaluate the relationship among IVC-CI, packed cell volume (PCV) and fluid requirements. IVC-CI >50% was positively correlated with high PCV; features of DSS; need for normal saline fluid boluses and colloid requirements; intensive care admission and with mortality. Thus, IVC-CI is recommended as a better non invasive tool to predict intravascular volume status in children with DF.

Most patients admitted to ICU with DF were found to have high IVC-CI and other volume depletion signs (compared to the base line values)²³.

In another study³ (162 adults) hemodynamic changes and IVC parameters were assessed to determine the type of circulatory shock in DHF. High proportion of patients who had IVCd <1.5 cm and IVC-CI >50% during defervescence, subsequently developed DSS (p<0.05). Both cardiogenic and hypovolemic shock appears to have equal contribution (50%) in the pathogenesis of DSS. A dynamic approach to assess the preload during the critical phase would

facilitate optimal fluid therapy and low complication rates in DSS; because both excessive as well as inadequate fluid administration are associated with adverse outcome; a positive fluid balance was found among patients with DSS (p<0.05).

To sum up, IVCd and IVC-CI are important parameters to assess intra vascular volume status in both severe and non severe DF.

Liver involvement in dengue

Liver involvement in DF is yet to understand. Hepatic involvement can vary from transient liver cell dysfunction to acute liver failure^{24,25}. Liver enlargement and elevated enzyme levels are common in DF, particularly in DHF^{3,4,5,6,7}. Liver enzyme levels have shown a positive correlation to the disease severity.²⁶ In contrast to acute hepatitis, DF patients are having higher AST levels than ALT levels^{24,25,26,27,28,29}.

Fulminant liver failure is uncommon in DF compared to DSS^{28,29}. In DSS related acute liver failure, portal venous flow pattern found to be abnormal (reduced portal venous flow that has reversed subsequently); the resistance to intra hepatic portal venous blood flow could be due to hepatic sinusoidal or kupffer cell involvement. Possible causes of liver

injury in DF could be low arterial perfusion in DSS; immune mediated injury; or injury due to direct viral replication within the liver cells.^{25,2730} Liver congestion, steatosis, cell necrosis and apoptosis were recognized histopathological features in postmortem liver specimens³⁰.

Future scopes

The scarcity of knowledge regarding fluid management in DF and DSS, warrants future studies to establish the correlation of IVC-CI and outcome of dengue patients that would be an initiative in developing new guidelines for fluid management.

Pathogenesis of the hepatic dysfunction in DF is poorly understood. Hence future sonographic studies are recommended to bridge this gap.

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Figure 1: Gall bladder wall thickening patterns seen in dengue fever ¹¹

Figure 2: Schematic and ultrasound appearance of gall bladder wall thickening patterns¹²

(A- 1&2 - uniform echogenic pattern, B- 1&2- striated pattern, C- 1&2- asymmetric pattern, D- 1&2- tram track pattern, E-1,2&3- honeycomb pattern)

Figure 3: measurement of IVC diameter with USG ²²

Table 1: Sonographic findings in dengue fever

	Liver enlarged	Spleen enlarged	GBWT		Pleural E.		Ascites		Pericardial E.
Venkata Sai et al. ⁴	60.9	NA	100		Right 71.9	Left 21.7	53.2		28.5
Michels M et al. ⁵	NA	NA	Severe 100	NS 67	Severe 36	NS 7	Severe 36	NS 7	NA
Keshava HK et al. ⁷	16	20	38		22		9		NA

(GBWT- gall bladder wall thickening, E- effusion, NA- not assessed, NS- non severe, figures are displayed in percentages (%))

Table 2:-Summary of Sonographic Findings in different groups according to platelet counts⁷

Sonographic findings	Group-1 Plt<50 x10 ³	Group-2 Plt 50 -100x 10 ³	Group-3 Plt 100 -150 x10 ³	Group-4 Plt >150 x10 ³	p value
Patient (n)	58	21	9	12	
Hepatomegaly	48 (83%)	14 (67%)	4(44%)	0	<0.001
GBWT	41 (71%)	9 (43%)	1 (11%)	0	<0.001
Splenomegaly	17 (29%)	10 (47%)	2 (22%)	0	<0.034
PE	36 (62%)	2 (9.5 %)	0	0	<0.001
Ascites	33(60%)	4(19%)	0	0	<0.001
Polyserositis	33(57%)	1(5%)	0	0	<0.001

(p value < 0.001 - statistical significant, GBWT- gall bladder wall thickening, Plt- platelet count, PE – pleural effusion)

Table 3: Common causes of gall bladder wall thickening⁸

Diffuse Wall Thickening	Focal Wall Thickening
Cholecystitis	Polyp- adenomatous, cholesterol
Liver disease	Malignancy
Extra cholecystic inflammation – pancreatitis, colitis, peritonitis, pyelonephritis	Focal adenomyomatosis
Systemic diseases - congestive heart failure, renal failure, sepsis, hypoalbuminemia	Focal xanthogranulomatous cholecystitis
Adenomyomatosis	
Pseudothickening (contracted status)	
Malignancy	
Other infections – TB, dengue	

Table 4: Ultrasonographic patterns of fluid leakage in dengue haemorrhagic fever & dengue shock syndrome¹⁴

		DHF (n=215)	DSS (n=11)
Ascites		144 (66.9%)	4 (36.3%)
	Pelvic fluid	39 (27%)	
	Mild AS	68 (47.2%)	
	Moderate AS	33 (22.9%)	
	Massive AS	4 (2.7%)	
PE		110 (51.1%)	7 (63%)
	Right PE	82 (74.5%)	
	Left PE	1 (0.9%)	
	Bilateral PE	27 (24.5%)	
	Mild PE	98 (89%)	
GB Wall Thickening		51 (23.7%)	1 (9.1%)
Pericardial effusion		0 (0%)	

(DHF- dengue haemorrhagic fever, DSS- dengue shock syndrome, AS- abdominal ascites, PE- pleural effusion)

Table 5: Sensitivities, specificities, positive and negative predictive values of right lateral decubitus chest roentgenograms and ultrasound scans in diagnosing DHF¹²

	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)
Chest USS(D-1)	100	76	61	100
CXR (D-1)	62	99	93	94
mGBWT>2mm	62	92	54	94
fluid (D-1)	42	100	100	91

(CXR- Right lateral decubitus chest X ray, USS- ultrasound scan, mGBWT- maximum gall bladder wall thickness, D -1: day before defervescence, fluid- free fluid in hepatorenal pouch)