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ORIGINAL ARTICLE

Association of Non-Alcoholic Fatty Liver Disease in Cases of Fitz-Hugh-Curtis Syndrome and its Improvement following Treatment of FHC Syndrome

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Abstract

Pelvic inflammatory disease is common and often neglected and is frequently being missed by clinicians. The ascend of infection around the liver leads to perihepatitis and the combination of two is called Fitz-Hugh-Curtis (FHC) syndrome. We studied the association of non-alcoholic fatty liver disease (NAFLD) with FHC syndrome. Ultrasound was done in 120 cases of FHC syndrome and 40 (33.33%) had NAFLD as compared to an average of 13.7% prevalence in females in India in general population reported in literature, a statistically significant difference (Chi square 10.04, df 1, p 0.001), thereby suggesting a higher prevalence of NAFLD in cases of FHC syndrome. The prevalence of NAFLD in cases of FHC syndrome significantly (chi square 14.605, df 4, p 0.006) increased with increasing age, thereby suggesting the longer the duration of FHC syndrome higher the chances of NAFLD. Treatment of FHC syndrome with four weeks course of doxycycline 100 mg twice daily significantly decreased the mean levels at four weeks and eight weeks follow up respectively of aspartate transaminase (p 0.001 and < 0.001), alanine transaminase (p 0.007 and 0.001) and alkaline phosphatase (p 0.003 and < 0.001) enzymes in cases of NAFLD with FHC syndrome as compared to those with FHC syndrome alone. Five out of 31 (16.12%) cases showed regression of NAFLD after treatment on repeat ultrasonography at 8 weeks. Thirty-four (85%) patients had Grade-1 fatty liver and 6 (15%) patients had Grade-2 fatty liver and none had progressed to grade 3. Therefore, it is very important to promptly diagnose and treat PID/FHC syndrome to prevent progression of NAFLD.

Key words: Fitz-Hugh-Curtis syndrome, Non-alcoholic fatty liver disease, ultrasonography, Aspartate transaminase, Alanine transaminase, Alkaline phosphatase

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Introduction

Pelvic inflammatory disease involves the female upper reproductive tract, it is an infection affecting the endometrium, fallopian tubes, ovaries, and pelvic peritoneum, ovaries, fallopian tubes and endometrium [1]. *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are usually found causative agents in cases of PID, as well in other sexually transmitted infections (STIs). However, they are not the only organisms causing this disease. It is slightly difficult to diagnose cases of PID due to a variety of factors such as variation in clinical manifestations of the disease, many asymptomatic cases with subclinical disease, as well as patients requiring surgical intervention who suffer more severe disease [2,3].

Inflammation of the upper reproductive tract without any signs or symptoms of acute infection is referred to as Subclinical PID [3]. As per 2015 guidelines of CDC on Sexually Transmitted Diseases Treatment, any sexually active female in reproductive age group or any female at risk for STI with unexplained lower abdominal or pelvic pain and with at least one of three clinical criteria like cervical motion/uterine/adenexal tenderness should be presumptively treated for PID [4]. Data of National Survey of Family Growth (NSFG) from year 2006 to 2010 showed that only 5.0% of females get treated for PID during their lifetime [5].

The incidence of subclinical PID can be reduced by screening and early treatment of STIs and studies have concluded that incidence of subclinical PID is quite high [6].

In 1920 Carlos Stajano [7], for the first time described in cases of gonococcal infection adhesions between the liver capsule and anterior abdominal wall in patients complaining of pain in right upper quadrant of abdomen. Subsequently, Fitz-Hugh and Arthur Curtis in 1930s described the syndrome in patients having perihepatitis with “violin-string” adhesions between liver capsule and diaphragm in women with salpingitis [8,9]. *N. gonorrhoeae* was implicated as the only causative agent of this syndrome for a number of years [8,10,11]. However in 1978, Muller-Schoop et al

[12] was the first to demonstrate *Chlamydia trachomatis* antibodies in 9 out of 11 cases of peritonitis diagnosed on laparoscopic examination and perihepatitis was also found in 6. Similar finding have also been described by others [13].

Up to twenty five percent of patients of Fitz-Hugh-Curtis (FHC) syndrome have inflammation of the liver capsule alongwith PID. It classically presents as sharp, pleuritic right upper quadrant pain, along with signs of salpingitis/PID. FHC syndrome can be confused with many other common diseases such as cholecystitis and pyelonephritis [14].

Pain in the right upper quadrant (RUQ) pain along with fever, lower abdominal pain, vaginal discharge is the usual presentation of FHC syndrome [15]. The RUQ pain is classically sharp, pleuritic, aggravated with movement, and frequently presents as a referred pain to the right shoulder or the right arm. Nausea, vomiting, hiccupping, chills, fever, night sweats, headache, and malaise might be associated [15] This pain in the right upper quadrant is due to adhesion of the anterior hepatic surface to the abdominal wall [15]. The differential diagnosis of RUQ pain can be hepatobiliary, gastrointestinal and urogenital diseases [16].

Non-alcoholic fatty liver disease (NAFLD) can progress from fatty liver to non-alcoholic steatohepatitis, fibrosis, and ultimately to cirrhosis. Non-alcoholic fatty liver (NAFL) is usually asymptomatic and is characterized by increased deposition of fat on ultrasonography in 20-30% of the general population and is largely asymptomatic [17-20].

Only 5-6% of patients with NAFL progress to non-alcoholic steatohepatitis (NASH), fibrosis, or cirrhosis [21]. In these few cases there is enhanced risk of fatality from liver failure or carcinoma of liver, or requiring a liver transplant [17]. In cases of severe form of NAFLD there has been increase in hospital admissions for treatment of liver damage [22].

Frequency of the diagnosis of Fitz-Hugh–Curtis syndrome depends on the diagnostic criteria used. It is quite possible that patients with no symptoms have extended perihepatic adhesions recognized through laparoscopy, while others with clinically diagnosed PID and right upper quadrant pain might not have any laparoscopic signs or evidence of perihepatitis [14].

FHC syndrome poses diagnostic challenge in resource-limited settings where advanced investigations are not available. However, in a well-equipped centers, the causative organism can be isolated and other differential diagnosis can be excluded [23]. This involves the use of non-invasive and invasive measures including laparoscopy and laparotomy. Definitive diagnosis of perihepatitis can only be established by laparoscopic examination or by performing laparotomy and demonstrating characteristic "violin-string" adhesions [24]. However, in the absence of invasive techniques, systemic examination will reveal tenderness in the right upper quadrant and on auscultation friction rub that can be heard as was described by Fitz-Hugh as "beautiful new snow creaking frictions" [25].

Ultrasonography can rule out cholecystitis, cholelithiasis, hepatitis and other common causes in cases of right upper quadrant pain. USG can also detect ovarian abscess and collection of fluid in Pouch of Douglas suggestive of PID. In addition, typical ultrasonographic findings have also been found in cases of FHC syndrome [26-31].

It has been observed that patients with FHC syndrome had elevated transaminases levels. The raised enzyme levels came to normal on treatment with antibiotics, thereby suggesting that the perihepatitis may be responsible for elevated hepatic enzymes. Our aim was to establish any relationship between FHC syndrome and non-alcoholic fatty liver disease.

Material and Methods

Inclusion and Exclusion Criteria

The inclusion criteria were newly diagnosed cases of FHC syndrome aged 18-60 years. The exclusion criteria were patients with history of alcohol ingestion, patients with underlying liver disease (abscess, metastasis, etc) and patients already on antibiotics.

Study Procedure

Clearance from the institute's ethical committee was taken. After the written informed consent, as per Helsinki declaration all patients were interviewed according to a pre-designed questionnaire to collect the core behaviour information and details about the presence of any complaints related to PID such as lower abdomen pain, painful coitus, discharge per vaginum, irregular and/or painful menstruation. History of any systemic disease, relevant past history and family history was also recorded.

Patients were diagnosed with PID using CDC Guidelines (2015) - if one or more of the minimum clinical criteria like cervical motion tenderness or uterine tenderness or adnexal tenderness were present on pelvic examination.

Patients with PID who in addition had right hypochondrial tenderness were diagnosed as FHC syndrome. All patients clinically diagnosed with FHC syndrome using the above criteria were evaluated by doing whole abdomen ultrasonography on empty stomach along with liver function tests.

The patients diagnosed with FHC syndrome using the above criteria were treated with doxycycline 100 mg twice a day for 4 weeks.

All the patients with enzymatic abnormalities and fatty liver were followed up by doing repeat liver function tests after complete treatment of FHC syndrome i.e. at the end of 4 weeks and 8 weeks respectively and a repeat ultrasound scan was done at the end of 8 weeks after initiation of treatment.

Results

A total of 120 patients of FHC syndrome were analysed with USG and liver enzyme studies before treatment and after treatment. Age distribution of study population is as shown (Table 1). Of the study population, majority belonged to 26-35 years (30.6%) followed by 16-25 years and 36-45 years (24.8%), a sexually active population. The mean age of the study population was 35.52±11.25 years.

On USG 40 (33.3%) out of 120 patients showed fatty liver. Age group distribution of the study population having fatty liver is as

Table 1: Distribution of the study population having FHC syndrome according to Age groups

Age groups	Frequency	Percentage
16-25 years	30	24.8%
26-35 years	37	30.6%
36-45 years	30	24.8%
46-55 years	16	13.2%
56-65 years	7	5.8%
Total	120	100.0

shown in Table 2. A significant (chi square 14.605, df 4, p 0.006) increase in prevalence of fatty liver NAFLD was found with increasing population age, from 10% in age group of 16-25 years to 75% in age group of 56-65 years. Thirty-four (85%) patients had Grade-1 fatty liver and 6 (15%) patients had Grade-2 fatty liver and none had progressed to grade 3.

On liver enzymes study the mean levels of AST, ALT and ALP were found to be within normal range in cases of FHC syndrome (Table 3). However, 3 (3.7%) out of 80 without fatty liver as compared to 5 (12.8%) out of 40 patients with fatty liver had raised AST levels, a statistically significant difference (Chi square 4.098, df 1, p 0.010). Similarly, 4 (4.9%) patients without fatty liver as compared to 7 (17.9%) with fatty liver had raised ALT levels, a statistically significant difference (Chi square 5.679, df 1, p 0.005). Likewise, 7 (8.6%) patients without fatty liver and 9 (23.1%) patients with fatty liver had significantly raised ALP levels (Chi square 6.801, df 1, p <0.001), thereby indicating that all 3 enzymes were significantly raised in some cases with fatty liver as compared to those without fatty liver.

The mean AST before treatment, after 4 weeks and after 8 weeks was compared between subjects without fatty liver using the repeated measures ANOVA test with post-hoc Bonferroni test or inter-interval comparisons (Table 4). No significant difference was reported for the inter-interval comparison of mean AST in cases of FHC syndrome without fatty liver. However, the mean AST levels decreased significantly at 4 weeks (p<0.001) and after 8 weeks (p<0.001) as compared to pre-treatment levels in patients with fatty liver (Table 4).

The mean ALT levels before treatment, after 4 weeks and after 8 weeks was compared between subjects without fatty liver. No significant difference was found at 4 weeks and 8 weeks after treatment as compared to pre-treatment levels (Table 5). However, the same decreased significantly from before treatment to at 4 weeks and after 8 weeks in those with fatty liver (Table 5).

Similarly, the mean ALP levels before treatment, after 4 weeks and after 8 weeks were not found to be significant in subjects without fatty liver (Table 6). However, the same decreased significantly in those with fatty liver (Table 6).

Nine out of 120 patients were lost to follow up for repeat ultrasonography after 8 weeks. Therefore, a repeat USG could be performed in only 31 out of 40 cases of NAFLD. Five (16.12%) out of 31 patients showed regression of fatty liver on repeat USG scan at 8 weeks after treatment.

Discussion

Fitz-Hugh-Curtis syndrome is characterized by perihepatic inflammation along with PID, mainly among women of child bearing

Table 2: Age distribution of study population having fatty liver on USG

Age group (years)	Fatty liver on USG		
	Present (%)	Absent (%)	Total (%)
16-25	3 (10.0)	27 (90.0)	30 (100)
26-35	10 (27.0)	27 (73.0)	37 (100)
36-45	13 (43.3)	27 (56.7)	40 (100)
46-55	8 (56.7))	7 (43.3)	15 (100)
56-65	6 (75.0)	2 (25.0)	8 (100)
Total	40 (33.33)	80 (66.67)	120 (100)

Chi-square value-14.605, df-4, p-value- 0.006*

Table 3: Descriptive statistics of liver enzymes among study population

Liver enzyme	Minimum	Maximum	Mean	Std. Deviation
AST (10-40)	12	131	29.83	19.64
ALT (7-56)	6	118	32.39	23.74
ALP (20-140)	27	236	100.59	39.15

Table 4: Comparison of mean AST level before treatment, after 4 weeks and after 8 weeks in patients with and without fatty liver

AST	Mean		SD		p-value	
	Without fatty liver	*With fatty liver*	*Without fatty liver*	*With fatty liver*	*Without fatty liver*	*With fatty liver*
Before treatment	31.39	59.66	22.63	15.93		
After 4 weeks	26.11	43.02	17.37	11.41	0.655	0.001*
After 8 weeks	21.44	26.89	16.28	10.63	0.230	<0.001*

Repeated measures ANOVA test * Significant difference

Table 5: Comparison of mean ALT level before treatment, after 4 weeks and after 8 weeks in patients with and without fatty liver

ALT	Mean		SD		p-value	
	Without fatty liver	*With fatty liver*	*Without fatty liver*	*With fatty liver*	*Without fatty liver*	*With fatty liver*
Before treatment	30.77	55.12	24.23	19.90		
After 4 weeks	23.14	42.25	16.16	13.10	0.378	0.007*
After 8 weeks	24.53	29.29	11.12	10.90	0.520	0.001*

Repeated measures ANOVA test * Significant difference

Table 6: Comparison of mean ALP level between before treatment, after 4 weeks and after 8 weeks in patients with and without fatty liver

ALP	Mean		SD		p-value	
	Without fatty liver	*With fatty liver*	*Without fatty liver*	*With fatty liver*	*Without fatty liver*	*With fatty liver*
Before treatment	87.34	159.26	37.33	43.68		
After 4 weeks	79.60	120.24	29.72	31.98	0.726	0.003*
After 8 weeks	65.98	98.39	28.35	28.12	0.097	<0.001*

Repeated measures ANOVA test * Significant difference

age. It is seen in all the age groups in women, as a complication of pelvic inflammatory disease. The peak prevalence of FHC syndrome was found to be highest in the age group of 15-35 years by Basit et al [32] and 15-24 years in a study by Risser et al [33] as was also seen in our study. Woo et al [34] found the mean age of 31.0 + 8.1 years (range 19-49 years) in a study of 22 patients of FHC syndrome as compared to 35.52±11.25 in our study. This signifies that majority of affected patients fall in the sexually active group of female population.

In our study, we found that 33.3% patients of FHC syndrome had associated NAFLD which is much higher as compared to the prevalence of fatty liver in general Indian population (18.9%) as reported by Amaraparkar et al [35]. In the study the authors recruited 730 subjects of both sexes above the age of 20 years with a mean age of 39.08 ± 12.3 years. Prevalence of NAFLD was found to be 24.6% in males as compared to 13.6% in females. Similarly, Singh et al [36] also found a prevalence of NAFLD in 13.8% in general female population. So, from the available Indian literature the prevalence of NAFLD in females was average of 13.7% as compared to 33.3% found in cases of FHC syndrome in our study, a statistically significant difference (p=0.001). Thereby, suggesting that FHC syndrome is one of the important causes of NAFLD in females. Although the majority of studies are among people aged 30 to 70 years, the general trend of increased prevalence is observed with age, with peak prevalence of NAFLD noted between age 50-60 in men.[36] According to a study by Lazo et al [37] in women, prevalence of NAFLD has been seen to increase with age especially after menopause; with 11.1% in ages 30 to 40 years old, 15% in 41 to 50 years old, 20.5% in 51 to 60 years old, and 25.7% in over 60 years old. In our study we found a mean age of 41.57± 11.95 years in patients of FHC syndrome having NAFLD. This signifies that in cases of FHC syndrome a comparatively younger age group is having a higher prevalence of NAFLD as compared to that found out in various studies done in general population. Thereby, further strengthening that FHC syndrome has a possible role in causation of NAFLD in few of the cases.

Five to six percent of NAFLD patients progress to NASH fibrosis and cirrhosis leading on to high mortality and morbidity. NAFLD may manifest as fatty liver, steatohepatitis (NASH), fibrosis and cirrhosis [21]. In our study 34 (85%) patients had Grade-1 fatty liver and 6 (15%) patients had Grade-2 fatty liver. We didn't find any case with fibrosis or cirrhosis liver. A progressive increase in the prevalence of NAFLD was found with increasing population age groups from 10% in 16-25 years ages to 75% in 56-65 years. This indirectly indicates that the patients having FHC syndrome for a longer period are at a much higher risk for the development of NAFLD. Through extensive literature search, to the best of our knowledge we could not find any study showing association of FHC syndrome with NAFLD. In our study one patient was found to have an echogenic fatty focus in the periportal region. This could be the start of an inflammatory process which can potentially progress to involve the whole liver.

Maharjan et al [38] found a significant increase in liver enzymes in a comparative study of 75 cases of NAFLD with 70 controls. Although in our study the mean values of AST, ALT and ALP were not found significantly raised in cases of NAFLD as compared to those without it, however a significant number of cases of FHC syndrome with NAFLD had raised levels of AST, ALT and ALP as compared to those without it. Litt and Cohan [39] also reported a high rate of elevation of transaminase levels in cases of FHC syndrome.

The treatment of FHC is same to that of PID [40,41]. Its management includes the use of antibiotics like doxycycline and azithromycin in cases of Chlamydia associated FHCS [42]. The treatment is cheap and safe without the requirement of interventional procedures unless there are adhesions. Hence early diagnosis and treatment of FHC is important. This therefore calls for a need for early diagnosis and treatment of FHCS in resource-limited settings where interventional procedures are not readily available [43]. In our study, fatty liver regressed back to normal in 12.5% patients after treatment. This highlights chlamydia infection to be a causative factor in some cases of NAFLD and therefore treating it can lead to regression of both PID and fatty liver. This is possibly also the reason for significant reduction in levels of liver enzymes (AST, ALT and ALP) post-treatment. Litt and Cohan [39] also reported the resolution of all findings including hepatic transaminase levels, right upper quadrant tenderness and hepatic enlargement following treatment for gonococcal perihepatitis. This signifies that NAFLD is associated with deranged biochemical parameters in patients of FHC syndrome. Therefore, its early diagnosis will help prevent its further progression. In the large number of cases of FHC syndrome there are improvement in symptoms, laboratory abnormalities and imaging with appropriate antibiotic treatment [15, 44], as was also documented in a study by Rivero Sánchez et al [45].

Conclusion

In females, PID is common and often neglected and is frequently being missed by clinicians. The chronicity of PID may lead to spread of the infection to involve organs in the upper reproductive tract leading to complications. The ascend of infection around the liver leads to perihepatitis and perihepatic adhesions to anterior abdominal wall. We suggest that perihepatic inflammation can cause NAFLD in a few cases as we found a significantly higher proportion of NAFLD in patients of FHC syndrome when compared to the healthy population. Almost one-sixth (16.12%) cases showed regression of NAFLD and there was also significant fall in elevated hepatic enzymes after treatment of FHC syndrome. Therefore, it is very important to promptly diagnose and treat PID/FHC syndrome to prevent the above-mentioned complications.

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Ethics:	There is no ethical violation as it is based on voluntary anonymous interviews
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