SAS Journal of Surgery
Abbreviated Key Title: SAS J Surg
ISSN 2454-5104
Journal homepage: https://saspjournals.com/sasjs/

OPEN ACCESS

A Prospective Study to Evaluate the Correlation between Acute Appendicitis, Its Complications and Hyperbilirubinemia

Dr. Arun Walwekar[1], Dr. Adil Mohamed[2*]

[1]Assistant Professor, Department of General Surgery, Karnataka Institute of Medical Sciences, Hubli, Karnataka, Pin -580022, India
[2]Junior Resident, Karnataka Institute of Medical Sciences, Hubli, Karnataka, Pin- 580022, India

DOI: 10.36347/SASJS.2019.v05i11.003 | **Received:** 08.11.2019 | **Accepted:** 15.11.2019 | **Published:** 29.11.2019

***Corresponding author:** Dr. Adil Mohamed

Abstract **Original Research Article**

Background: In acute inflammatory conditions such as acute appendicitis there is dysfunction of kupffer cells and hepatocytes due to bacterial overload which reach the liver via portal circulation causing increase in the serum bilirubin levels. Acute inflammatory cytokines can also lead to raised serum bilirubin levels without much rise in the serum liver enzymes levels. ***Aim:*** To correlate hyperbilirubinemia with acute appendicitis and its complications such as appendicular perforation and gangrenous appendicitis. ***Material and Methods:*** This is a prospective study conducted at department of general surgery KIMS, Hubli during the period of august 2017 to august 2018. 50 cases of acute appendicitis were selected. All the basics investigations were done such as complete blood count, LFT and ultrasound. Liver function test was done preoperatively. Cases underwent open appendicectomy and intraoperative findings were noted. Clinical examination, laboratory values and operative details were compiled and analysed and following observations were made. ***Results:*** Out of 50 cases 38 had raised serum bilirubin. No associate raise in the liver enzymes. ***Conclusion:*** This study comes to the conclusion that there is correlation between acute appendicitis (and its complications) with the raised serum bilirubin. There is raise in both the direct and indirect bilirubin. There is no raise in the liver enzymes

Keywords: Acute appendicitis, appendicular perforation, Gangrenous appendix, Hyperbilirubinemia.

INTRODUCTION

Acute appendicitis is one of the most common surgical emergencies. Complications like gangrenous appendix and perforation can occur leading to peritonitis and sepsis. Inciting event in most instances of appendicitis is obstruction of lumen (partial/complete) leading to accumulation of secretion and distention of lumen, which rises intra- luminal pressure. There is lymphatic obstruction first later venous obstruction and bacterial over growth and edema. An inflammatory response causes the appendix to become more edematous and ischemic. Complications like gangrenous appendix and perforation can occur leading to peritonitis and sepsis. Bacteria overload reaches liver via portal circulation causing dysfunction of hepatocytes leading to raised serum bilirubin levels.

The mechanism of hepatic injury in sepsis is not completely understood. This is may be due to bacterial infections, its toxin or due to cytokines. In early sepsis with hyper-dynamic circulation bacteria, its toxin or cytokines are involved where as in late sepsis ischemia decreased hepatic blood flow to the liver is the mechanism of hepatic injury. In both above situations the hepatic injury leads to dysfunction of hepatocyte and tubule leading to mixed type of hyperbilirubinemia (hepatocellular and intra hepatic cholestasis). Cholestasis in severe bacterial infection and in post operative period is presumably hepatocellular in nature. It can also be related to cholestatic effect of endotoxin on sodium-potassium-ATPase. All the constituents of bile show an increased level in serum. Conjugation of biliary substance is intact but excretion is defective. Serum alkaline phosphatase is raised. The rise is due to increase synthesis or release of enzymes from liver or biliary plasma membrane. The minimal hepatocellular damage is suspected by noting minimal elevated transaminase value.

MATERIAL AND METHODS

This was a prospective study conducted at department of general surgery KIMS, Hubli during the period of august 2017 to august 2018. 50 cases of acute

appendicitis were selected. All the basics investigations were done such as complete blood count and ultrasound abdomen and pelvis. Liver function test was done preoperatively. Cut off for total bilirubin was taken as <1.2mg/dl, direct bilirubin <0.2 mg/dl, AST <40U/l, ALT <35U/l, ALP< 170 U/L. Cases underwent open appendicectomy and intraoperative findings were noted. Clinical examination, laboratory values and operative details were compiled and data was analyzed with tables and graphs and following observations were made.

INCLUSION CRITERIA

Cases with age group of 13 to 60 years with acute appendicitis without any previous history of jaundice were included.

EXCLUSION CRITERIA

Cases with age less than 13 years and more than 60 years were excluded. Cases with previous history of jaundice and with HBSag positive status were excluded.

RESULTS

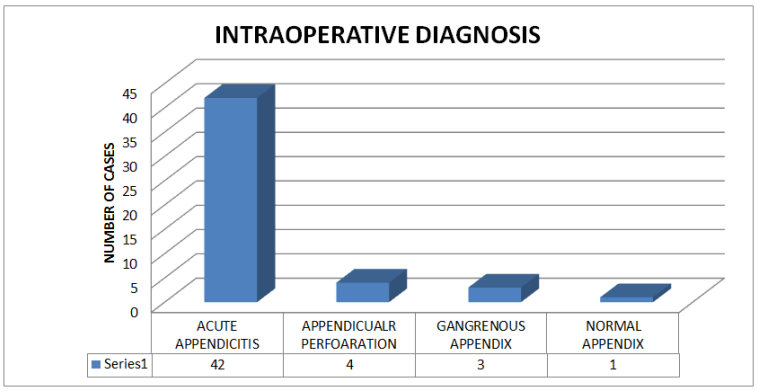
Out of total 50 cases 35 were males and 15 were females with age ranging from 13 to 60 years. Average age was 28.5 years. Among these cases duration of symptoms ranged from 8 hours to 10 days. Out of 50 cases 42 had acute appendicitis, 4 had appendicular perforation, 3 had gangrenous appendix and 1 case had normal appendix. Serum bilirubin was raised in 38 cases and 12 had normal serum bilirubin levels. Serum bilirubin ranged from 0.6 to 5.6 mg/dl with average being 2.272mg/dl.

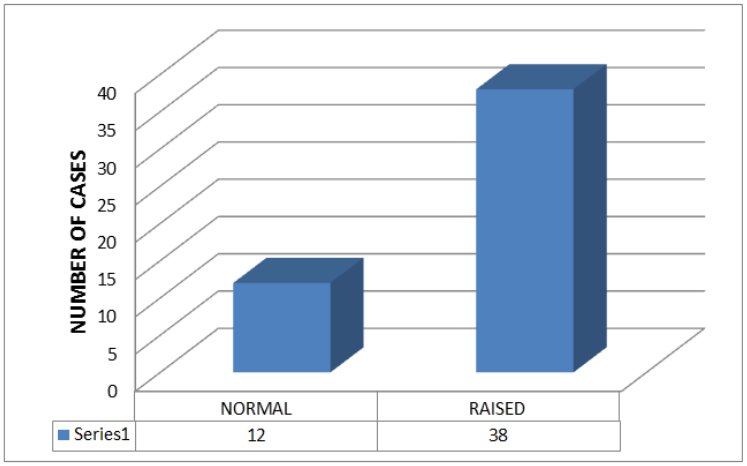
AST levels were normal in 35 (70%) cases and 15 (30%) cases had raised AST levels. AST levels ranged from 16-76 U/L with average being 36 U/L.

ALT levels were normal in 38 (76%) cases and 12 (24%) cases had raised ALT levels. ALT levels ranged from 23 – 105 U/L with average being 41.98U/L.

ALP levels were normal in 43 (86%) cases and 7(14%) cases had raised ALP levels. ALP levels ranged from 88 – 356 U/L with average being 134U/L.

None of the cases had significantly (>3 times) raised liver enzymes.





MASTER CHART

SI NO	AGE	GENDE	DURATION OF SYMP	DIAGNOSIS	TOTAL BILIRUBIN	DIRECT	INDIRECT	SGOT	SGPT	ALP
1	25	M	1	AP	5.4	2	3.4	38	31	112
2	20	M	5	N	0.9	0.3	0.6	26	34	98
3	18	M	2	AA	3.6	1	2.6	18	29	102
4	16	F	4	AA	1.2	0.3	0.9	43	32	135
5	13	M	6	AA	0.6	0.1	0.5	39	30	128
6	22	F	8	AA	1.2	0.4	0.8	35	98	152
7	28	F	5	AA	1.6	0.6	1	70	29	165
8	35	M	7	AA	2.4	0.4	2	38	28	126
9	45	M	4	AA	2.2	0.8	1.4	36	32	101
10	38	M	5	AA	2.5	0.6	1.9	16	25	112
11	60	M	6	AA	1.2	0.1	1.1	21	34	117
12	55	M	4	AA	1	0.1	0.9	34	31	88
13	29	M	5	AA	3.2	1.2	2	42	32	96
14	25	M	2	AG	4.3	1.1	3.2	35	42	94
15	26	F	1	AG	5.6	1.2	4.4	52	31	132
16	27	M	2	AP	4.6	3.5	1.1	35	46	207
17	19	M	10	AA	0.8	0.1	0.7	48	30	96
18	25	M	1	AA	1.6	0.6	1	53	29	154
19	50	F	1	AA	1.8	1.1	0.7	36	33	198
20	46	M	2	AA	2.7	0.8	1.9	24	76	89
21	40	F	3	AA	2.6	1.2	1.4	40	34	138
22	38	F	1	AA	1.2	0.4	0.8	42	88	156
23	16	M	8H	AA	1.1	0.2	0.9	16	32	85
24	52	M	2	AP	4.6	2.5	2.1	40	97	356
25	47	M	1	AA	3.2	1.2	2	18	33	102
26	29	M	2	AA	0.9	0.2	0.7	25	32	98
27	30	M	4	AA	3.8	1.6	2.2	28	46	147
28	31	M	2	AA	4.1	1	3.1	36	91	185
29	41	M	3	AA	3.3	0.9	2.4	52	31	168
30	23	M	2	AA	1.1	0.4	0.7	27	105	145
31	26	F	1	AA	0.9	0.1	0.8	26	23	168
32	29	F	2	AA	1.6	0.6	1	34	68	147
33	21	M	1	AA	0.8	0.3	0.5	29	29	99
34	19	F	2	AA	1.1	0.4	0.7	45	33	105
35	20	M	1	AA	1.8	0.6	1.2	19	34	102
36	14	F	4	AA	1.6	0.6	1	17	29	116
37	17	M	1	AA	0.9	0.3	0.6	31	88	147
38	24	M	2	AA	1.6	0.4	1.2	55	27	111
39	29	F	1	AA	2.1	0.6	1.5	37	95	142
40	33	F	4	AA	1.8	0.7	1.1	38	33	128
41	28	M	2	AA	0.8	0.5	0.3	48	28	205
42	25	M	1	AP	4.3	1.2	3.1	18	30	178
43	24	M	2	AA	3.4	0.9	2.5	22	29	92
44	26	F	1	AA	2.2	0.5	1.7	28	28	136
45	22	F	2	AA	1.3	0.4	0.9	35	27	165
46	13	M	1	AA	1.8	0.6	1.2	42	29	125
47	18	M	3	AA	1.6	0.3	1.3	16	34	185
48	15	M	2	AA	2.4	1	1.4	58	34	102
49	26	M	4	AG	5.6	2.2	3.4	76	31	89
50	28	M	2	AA	1.7	0.5	1.2	63	29	99
AVG	28.5		2.857142857		2.272	0.772	1.5	36	41.98	134

AGE	NUMBER OF CASES
13-20	13 (26%)
21-30	23 (46%)
31-40	6(12%)
41-50	5 (10%)
51-60	3 (6%)
TOTAL	50

DURATION OF SYMPTOMS	NUMBER OF CASES
<24 HOURS	1 (2%)
1- 2 DAYS	30 (60%)
2 – 3 DAYS	3 (6%)
> 3DAYS	16 (32%)
TOTAL	50

SERUM BILIRUBIN LEVELS	NUMBER OF CASES
NORMAL	12 (24%)
RAISED BILIRUBIN	38 (76%)
TOTAL	50

DISCUSSION

Causes for raised serum bilirubin may be prehepatic, hepatic or post hepatic. In prehepatic causes there is raise in indirect bilirubin and in hepatic and post hepatic there is increase in both the type.

In this study there was raise in both the direct and indirect serum bilirubin. There was no significant raise in the liver enzymes. This suggests that there was no damage to hepatocytes but instead dysfunction in permeability of bilirubin and cholestasis causing rise in both the types. The present results are similar to the experimental study of Whiting *et al.,* [1] in which they have demonstrated, depressed excretion of bile in canaliculi. This finding is further supported by demonstrating inspissated bile in dilated proliferated and peri-portal ductules in the histopathological study of liver.

In acute inflammatory conditions such as acute appendicitis there is dysfunction of kupffer cells and hepatocytes due to bacterial overload which reach the liver via portal circulation causing increase in the serum bilirubin levels. Acute inflammatory cytokines can also lead to raised serum bilirubin levels without much rise in the serum liver enzymes.

Indirect evidences of bacterial translocation from inflamed gastro intestinal tract or peritonitis to liver via portal vein and development of hepatitis, pyogenic liver abscess was observed by Fitz and Dieulfoy in their clinical studies [2, 3]. Oshner and Debakey [4] also provided classical treatises on pyogenic infection. These authors revived personal experiences and world literature, and emphasized its pathogenesis and clinical presentation. Bacteria isolated in pyogenic liver abscesses were similar to the bacteria involved in acute inflammation of the gut (i.e. acute appendicitis) and peritonitis. These agents were E. coli, Streptococcus fecalis, Klebsiella and Proteus vulgaris [5]. Commonly the infections were of mixed type. So, it was concluded from indirect evidences that pyogenic liver abscesses developed from the bacteria actually transmigrated from inflamed gut. Recently in one study blood samples from superior mesenteric vein in acute appendicitis showed bacteria in 38% of patients. Similarly; it has also been observed in experimental study done on gonobiotic mouse model, that showed micro-organism moving from gut into lymphatics. Based on the experimental studies it was concluded that the hepatocellular dysfunction in early stages of sepsis is due to release of inflammatory cytokines such as TNF – alpha and IL – 6.

CONCLUSION

This study comes to the conclusion that there is correlation between acute appendicitis (and its complications) with the raised serum bilirubin. There is raise in both the direct and indirect bilirubin. There is no much raise in the liver enzymes.

REFERENCES

1. Whiting JF, Green RM, Rosenbluth AB, Gollan JL. Tumor necrosis factor-alpha decreases hepatocyte bile salt uptake and mediates endotoxin-induced cholestasis. Hepatology. 1995 Oct 1;22(4):1273-8.
2. Fitz RH. Perforating inflammation of the vermiform appendix. Am J Med Sci. 1886 Jul 8;92:321-46.
3. Dieulfoy. Le foie appendiculaire: ab'ces du foi concutifs 'a l' appendicite. Semin Med (Paris) 1898; 18; 449.
4. Ochsner A, DeBakey M, Murray S. Pyogenic abscess of the liver: II. An analysis of forty-seven cases with review of the literature. The American Journal of Surgery. 1938 Apr 1;40(1):292-319.
5. Kauffmann F. Enterobacteriaceae. Collected Studies on Salmonella, Arizona, Ballerup-Bethesda, Escherichla, Alkalescens-Dispar, Klebsiella, Shigella, Providence and Proteus. Enterobacteriaceae. Collected Studies on Salmonella, Arizona, Ballerup-Bethesda, Escherichla, Alkalescens-Dispar, Klebsiella, Shigella, Providence and Proteus. 1951.