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HISTOPATHOLOGICAL DIAGNOSIS OF BILIARY ATRESIA AND ITS CORRELATION WITH CLINICAL PRESENTATION: A RETROSPECTIVE STUDY

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ABSTRACT

Biliary atresia is a rare and life-threatening liver disease that primarily affects infants. Early diagnosis and timely intervention are crucial for improved outcomes. This retrospective study aimed to diagnose cases of biliary atresia through histopathological examination and confirm the diagnosis using immunohistochemical stains. Additionally, the study sought to correlate the histological findings with the clinical presentation. In 3 years (2019- 2022),11 cases of biliary atresia were analyzed, comprising 6 male and 5 female patients. All patients presented with elevated bilirubin levels (both direct and total) along with deranged liver enzymes. The key histopathological finding that consistently led to the diagnosis of biliary atresia in all 11 cases was characterized by portal tract expansion and oedematous fibroplasia, accompanied by bile ductular proliferation. Inflammatory cell infiltrates were also noted in the liver biopsies. Therefore, biliary atresia is a complex disorder with variable outcomes, underscoring the importance of prenatal screening and early postnatal detection. Timely surgical intervention and histological examination play crucial roles in managing this condition effectively. Further research and awareness are essential to optimize diagnostic and therapeutic strategies for biliary atresia, ultimately improving the prognosis for affected infants.

Keywords: Biliary Atresia, Bile Duct Proliferation, Neonatal Jaundice, Kasai Procedure, Liver Biopsy.

INTRODUCTION

Biliary atresia is a rare disease that is characterized by biliary obstruction of unknown origin.[1] It results in progressive fibrosis and obliteration of extrahepatic and intrahepatic bile ducts.[2] Biliary atresia occurs exclusively in the neonatal period.[1] It has a slight predominance in females occurs in 1 per 10,000-16,700 births.[3] Incidence of neonatal cholestasis is noted to be 1 in 2500 newborn children It is the commonest surgical cause for neonatal cholestasis.[4] Among group of children with neonatal cholestasis, about 34-42% have been noted to be biliary atresia.[5,6] Progressive liver disease with development of portal hypertension and liver failure resulting in death within first 2yrs of life is seen in affected children.[1] There is a noted regional variation in the incidence of BA, with higher rates observed in Asia and the Pacific region. In Taiwan, the disease is diagnosed in about 1 in every 5,000 individuals, while in Japan, it occurs in 1 in 10,000. In the Netherlands, the incidence is between 1 in 17,000 and 19,000, and in the USA, it is 1 in 15,000 live births. Hawaii, an island in the Pacific Ocean, also reports higher incidence rates. Similarly, increased occurrences are found among Inuits and Native Americans.[7-13] A recent study highlights a correlation between the incidence of BA and the frequency of the most common human leukocyte antigen (HLA) haplotype, suggesting a link to human colonization.[14] This particular ethnicity is more prone to maternal microchimerism, which could reasonably account for this genetic predisposition if immunological uniformity is maintained. Biliary atresia remains the most common indication of liver transplant in young children. Besides liver transplant,

Kasai procedure remains only treatment that can be offered to these patients.[15,16] Disease concordance in twins is unusual with familial clusterings being exceedingly rare.[17] No single agent or abnormality has been implicated as a cause of biliary atresia in humans. Biliary atresia is believed to result from interaction of genetic, environmental, immune and infectious factors individually or in combination. No single agent or abnormality has been implicated as a cause of biliary atresia in humans.[3] Biliary atresia is broadly classified into two main forms based on the organs involved. One is the early or syndromic form associated with high frequency of additional congenital malformations and is referred to as biliary atresia–splenic malformation (BASM) syndrome comprising 10 to 20 % of cases. Other is the late or non-syndromic form usually occurring as an isolated abnormality. It represents 80-90% of cases.[18] Its diagnosis is mostly made at 2-6weeks of age presenting with cholestasis-jaundice, acholia,[19] and possibly choluria and hepatomegaly.[3] BA can be classified into different types based on the area affected .TYPE 1 where common bile duct is being affected, TYPE 2 that is of two types: 2a in which common hepatic duct is affected, 2b in which common hepatic duct, cystic duct, common bile duct are affected, TYPE 3 BA involves entire bile pathway which includes intrahepatic duct, common hepatic duct, cystic duct, and common bile duct. BA can be classified as early-onset BA, if symptoms appear within 2 weeks of birth and late-onset BA, if symptoms appear after 2 weeks of birth based on the time of onset of disease.[18] In suspected patients of biliary atresia, histopathological examination of liver biopsy specimens represents a crucial element in the diagnostic evaluation. Biliary obstructive features must be confirmed histologically and distinguished from various non-obstructive etiologies of neonatal cholestasis.[19] A well-coordinated multidisciplinary approach is essential for appropriate patient management. Diagnosis of BA can be done by performing prenatal tests, clinical features, laboratory and imaging studies along with certain invasive procedures. Prenatal testing is done when BA is suspected in such cases,a cystic structure is observed in porta hepatis which needs further investigations to confirm the diagnosis. Clinical features of BA include conjugated bilirubin which persists beyond 2 weeks of life, acholic stools, dark urine and hepatomegaly. Diagnosis is mostly made at 2-6 weeks of life. Diagnosis of BA can be confirmed by various laboratory investigations which include biochemical liver function tests which are typically those of cholestasis with elevated levels of total and conjugated bilirubin, increased gamma-glutamyl transpeptidase, alkaline phosphatase and occasionally higher levels of transaminases. Imaging procedures for diagnosis include abdominal ultrasonography, the one being the gold standard and initial choice in suspected cases of BA. Hepatobiliary scintigraphy provides dynamic assessment of both parenchymal liver function and biliary excretion. MRCP allows for a visualization of major biliary structures, hence excluding the diagnosis of BA. Cholangiography is the gold standard procedure to diagnose BA. The cholangiogram can be obtained percutaneously, by laproscopy, via an open laparotomy or through ERCP. Above all,the definitive diagnosis of BA is established by histopathological examination of a liver biopsy.[20,21] Therefore, the aim of this study was to identify cases of biliary atresia by analyzing tissue samples through histopathological examination. Furthermore, the study aimed to validate the diagnosis by utilizing immunohistochemical stains and to establish a relationship between the observed histological characteristics and the clinical symptoms and presentation of the disease.

MATERIALS AND METHODS

The study was conducted in the department of pathology at Sher-i-Kashmir Institute of Medical Sciences, Jammu & Kashmir India. A total of 11 cases were collected retrospectively over a period of 3 years from the month of June 2019 to June 2022. Our study included wedge and core needle biopsies sent after surgical procedure of Hepatoportoenterostomy (Kasai Procedure). Only 2 cases were diagnosed as Biliary Atresia preoperatively on HIDA scan. Rest of the cases were operated after clinical suspicion and relevant clinical and radiological findings. The biopsies were confirmed on histopathological examination and immunohistochemical stains.

RESULTS

The study comprised 6 males (54.5%) and 5 females (45.5%). Ten cases were in the age group of 1 to 3.5 months, while one case studied was of 23 months of age as illustrated in [Table 1].

Majority of the cases had complaints of either jaundice, dark colored urine or acholic stools at the time of admission while 2 cases had history of abdominal pain in addition to the above mentioned symptoms.

Serum bilirubin levels were investigated which revealed raised (total and direct) bilirubin levels in all the cases (100%). In addition, other liver enzymes were also deranged in some cases as illustrated in [Table 2].

Hepatobiliary Ultrasonography was performed in 5 cases, all of which showed hepatomegaly. In addition, contracted gallbladder was seen in 2 out of the 5 cases studied. HIDA Scan was possible only in 2 cases which showed features suggestive of Extrahepatic Biliary Atresia and later confirmed on histopathological examination and subsequent immunohistochemistry.

Table 1

S. No	AGE (In Months)	Sex	Jaundice	Dark urine	Acholic stools	HB (gm/dl)	TLC (cu/mm3)
1.	1	M	P	A	P	17	5300
2.	2	F	P	A	A	14	6800
3.	2	M	A	A	P	13	7500
4.	1.5	M	A	A	P	13	9200
5.	1.5	F	P	P	A	14	8400
6.	2	M	P	P	A	13	6900
7.	1.5	M	P	P	P	14	10200
8.	3	F	P	P	P	13	9100
9.	1	F	P	P	A	16	8200
10.	23	F	P	A	A	13	7500
11.	3	M	P	A	A	13	6200

M-Male, F-Female, P-Present, A-Absent, HB-Hemoglobin, TLC-Total Leucocyte Count

Table 2

S.No	AGE (In Months)	UREA (mg/dl)	Cr (mg/dl)	Sr.BIL (Direct) mg/dl	Sr.BIL (Indirect) mg/dl	Sr.BIL (Total) mg/dl	GGT (U/L)	AST (IU/L)	ALT (IU/L)	ALP (IU/L)
1.	1	13	0.6	3.02	0.9	5.9	334	185	140	554
2.	2	15	0.4	3.2	0.9	6.3	350	188	145	560
3.	2	21	0.5	3.6	1.0	6.5	120	30	32	370
4.	1.5	14	0.7	3.1	0.8	5.8	354	25	30	320
5.	1.5	18	0.4	3.0	1.0	5.6	125	28	25	300
6.	2	12	0.3	3.4	1.0	6.5	445	190	142	280
7.	1.5	20	0.7	3.6	0.9	6.4	338	188	145	385
8.	3	23	0.5	3.9	0.7	6.3	54	32	28	250
9.	1	21	0.6	4.0	0.9	6.6	88	25	32	220
10.	23	15	0.5	3.2	1.1	5.9	109	192	142	310
11.	3	18	0.4	3.6	0.9	6.5	40	30	18	408

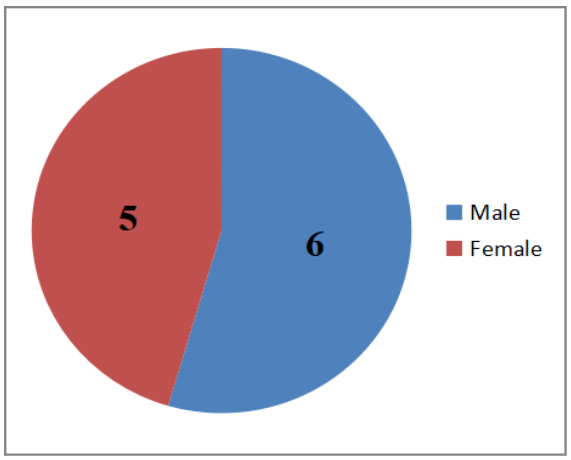
Cr-Creatinine, Sr.Bil-Serum Bilirubin, GGT-Gamma Glutamyl Transferase, AST-Aspartate Transaminase, ALT-Alanine Transaminase, ALP-Alkaline Phosphatase.

Table 3: As per Metavir Score(Assessment of Degree of Fibrosis).

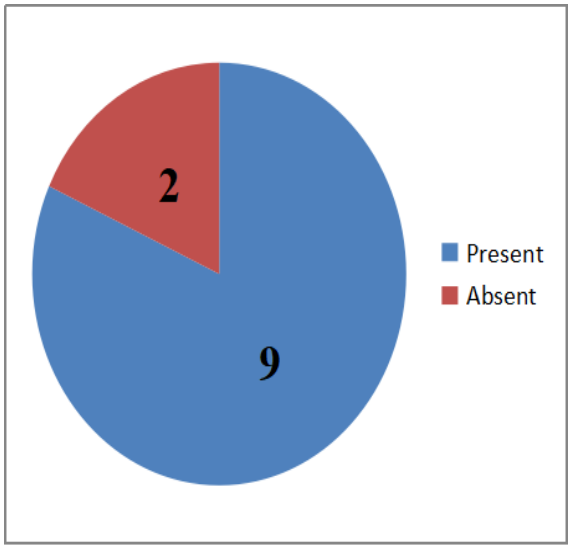
S. No.	Age (In months)	Degree Of Fibrosis (F1-F3)
1.	1	F1
2.	2	F2
3.	2	F1
4.	1.5	F1
5.	1.5	F1
6.	2	F1
7.	1.5	F1
8.	3	F3
9.	1	F2
10.	23	F2
11.	3	F3

F1- Portal fibrosis without septa, F2- Portal fibrosis with few septa, F3- Numerous septa with cirrhosis.

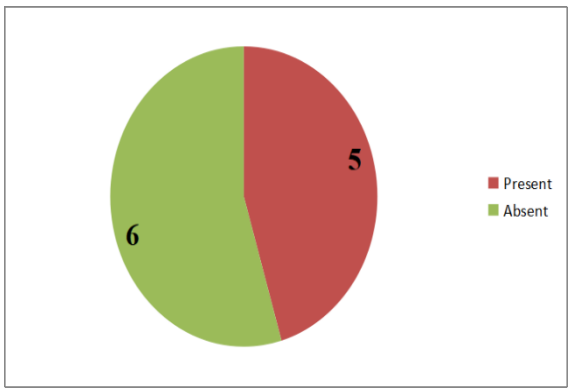
Pie and Bar charts representation of different parameters in Biliary Atresia as per our study:



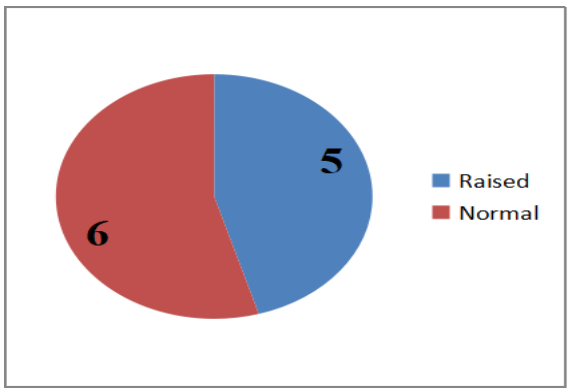
Gender Distribution



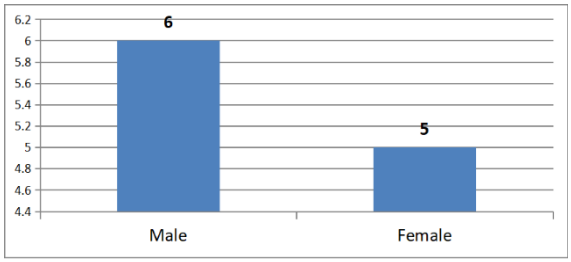
Jaundice



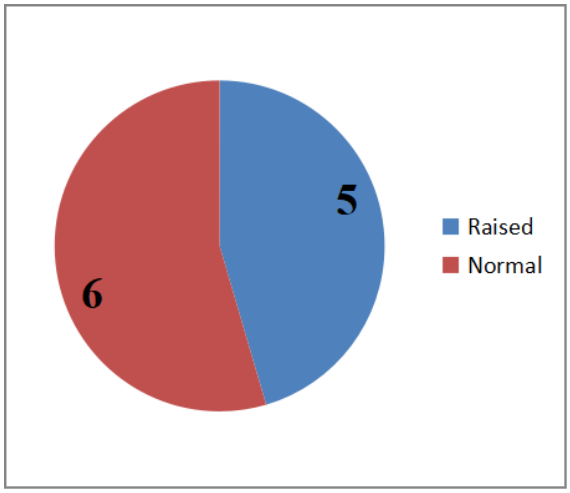
Acholic stools



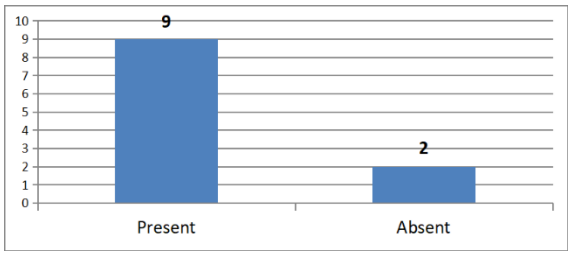
AST



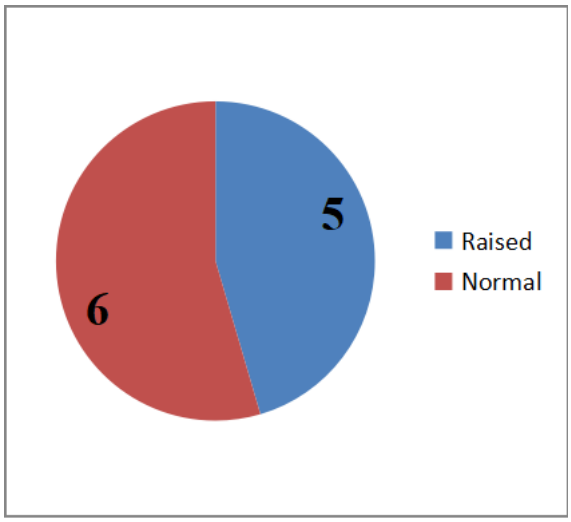
Gender distribution



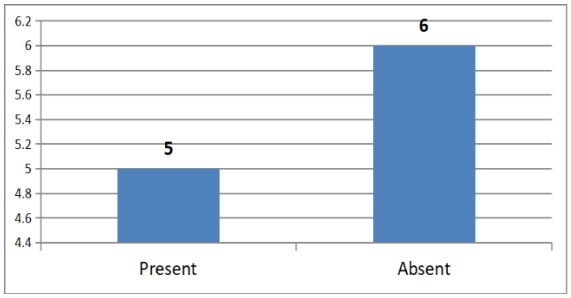
ALT



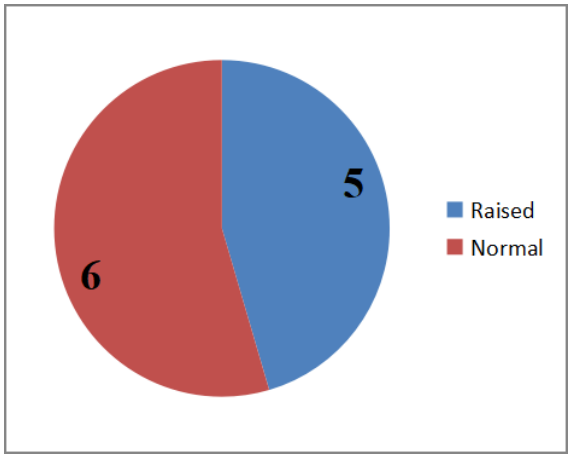
Jaundice



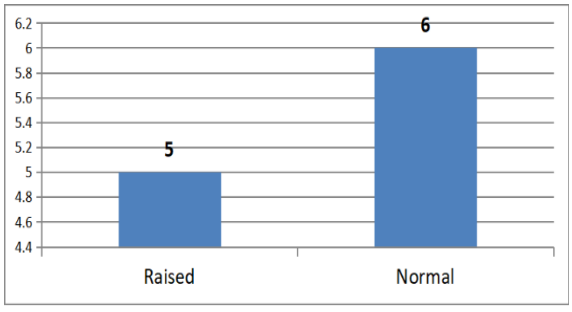
ALP



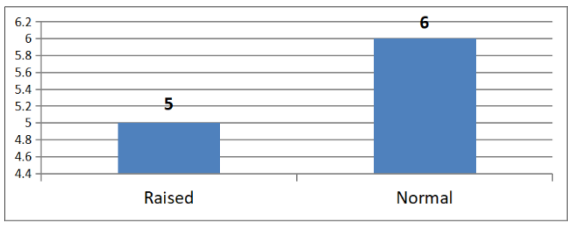
Acholic stools



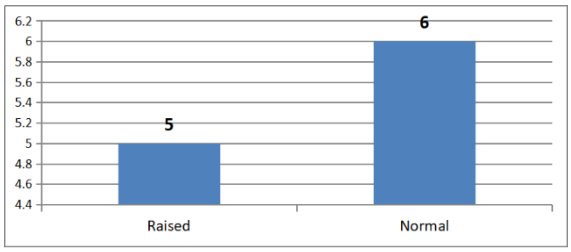
GGT



Gamma-Glutamyl Transferase



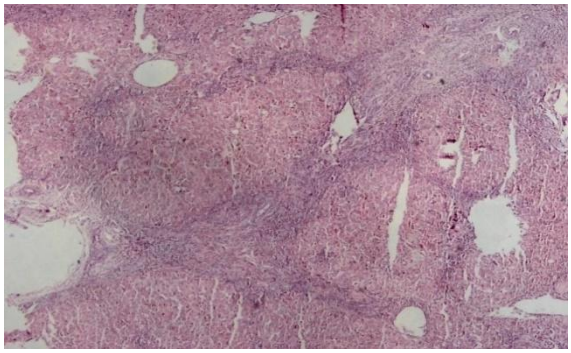
Alanine and Aspartate Transaminases

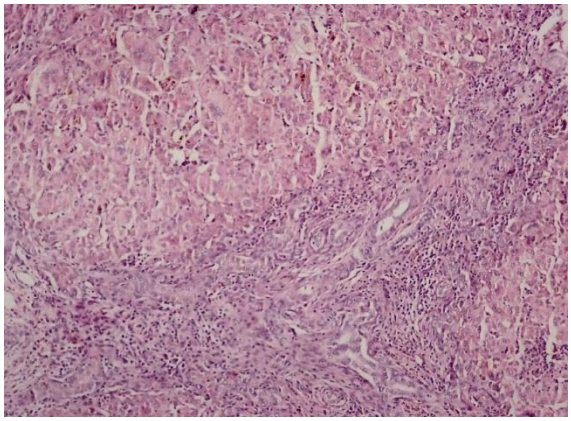


Alkaline Phosphatase

Wedge and core needle biopsies of liver were performed in all the cases and sent for histopathological examination. The routine staining (H&E) was performed for examination.

Key histopathological indicators for diagnosing BA are found in the portal tracts. Microscopic analysis of all cases showed expansion of the portal tracts and edematous fibroplasia accompanied by bile ductular proliferation, characterized by anastomosing ductules situated at the edges of the portal tracts. Observations included ductal bile plugs, portal edema, and varying levels of inflammatory cells, particularly neutrophils. Neutrophils were detected within the duct lumens. Portal tracts also demonstrated hemopoietic elements [Figure 1a, 1b and 1c].





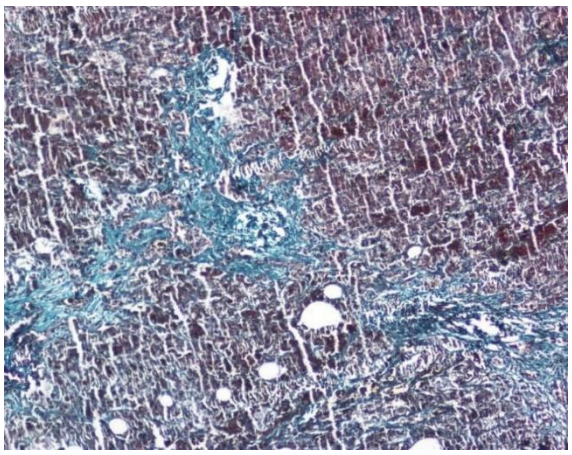


Figure 1: (a) Histological appearance of Biliary atresia, low power (10x), (b) High power view (40x) showing portal tract expansion with bile ductular proliferation along with portal neutrophilic infiltrate, (c) Masson's Trichome Stain positivity in case of Biliary atresia.

Unstained slides were also examined by means of immunohistochemistry for cytokeratin CK7 and CK19 to outline bile ducts and ductular reaction, reactive bile ducts/ductules and ductal plate malformation [Figure 2a and 2b].

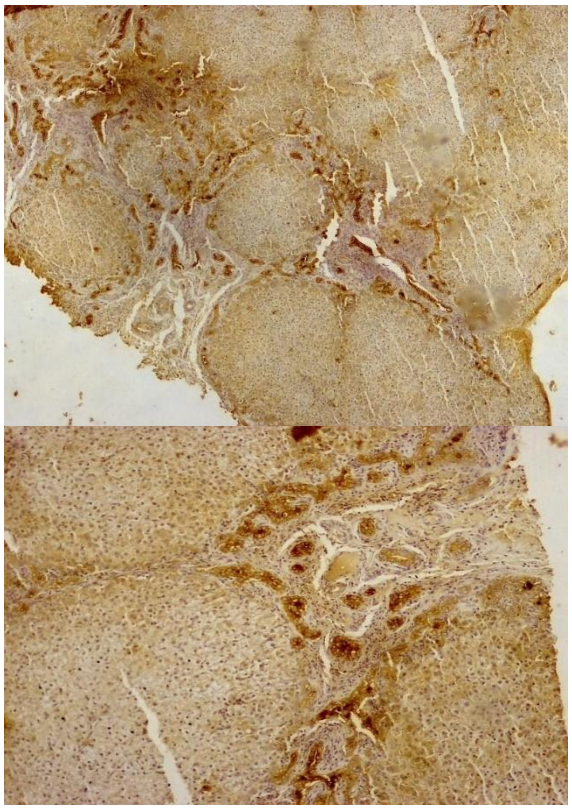


Figure 2: (a) Low power view (10x) and (b) High power view (40x) showing diffuse Cytokeratin (CK7) immunostaining.

DISCUSSION

Biliary atresia can be categorized into several types: TYPE 1, which affects the common bile duct; TYPE 2, which is further divided into 2a, where the

common hepatic duct is impacted, and 2b, where the common hepatic duct, cystic duct, and common bile duct are involved; and TYPE 3, which affects the entire bile pathway, including the intrahepatic duct, common hepatic duct, cystic duct, and common bile duct.[18]

This study presents a 3-year follow-up of 11 cases. A significant limitation in the statistical analysis of the data is the small sample size. All patients were managed by the surgical team, followed by various investigations, and liver biopsies were submitted for histopathological examination. Patients with biliary atresia typically exhibit total bilirubin levels exceeding 58.5mg/L, GGT levels above 100UI/L, and AST and ALT levels ranging from 80 to 200UI/L.

Research by Russo P et al., Batts KP et al., Roskams TA et al., and Kleiner D et al. showed that liver biopsies were assessed for histological features and the degree of fibrosis. Ductular reaction and bile duct proliferation were evaluated to determine if these features were focal (involving less than 50% of portal tracts) or generalized. It was found that duct/ductal bile plugs and portal stromal edema are independent histologic predictors of biliary atresia.[22-25] In our study, bile duct proliferation and ductular reaction were observed along with portal and bridging fibrosis. Similarly, portal stromal edema and ductal bile plugs were noted, suggesting they are independent histologic predictors of biliary atresia.

Another study by Rastogi A et al. and Santos JL et al. identified portal ductal proliferation, bile ducts in the ductules, and portal fibrosis as key indicators of biliary atresia.[26,27] Our study found similar histopathological results.

Bile ductular proliferation is a crucial diagnostic marker for extra-hepatic biliary atresia and is consistently found in biliary atresia except in the very early stages. Biliary fibrosis is an independent prognostic factor for biliary atresia.

Another study by Brough AJ et al. and Ferry GD et al. revealed that the most common specimen submitted for histopathological evaluation in neonatal cholestasis cases is a needle core biopsy sample.[28,29] In our study, we included both needle and wedge biopsy samples.

A study by Lefkowitch JH highlighted that the key to accurately diagnosing biliary atresia lies in the histopathological changes of the portal tracts [30]. Our study revealed similar changes in portal tracts, including portal inflammation, fibrosis, and nodule formation. Portal-based fibrosis is a typical diagnostic finding in this context.

A study by Ferry GD et al., Russo P et al., and Zerbini MC et al. identified that a ductular reaction is a significant characteristic in typical biliary atresia cases, with small, interconnecting ductules proliferating at the edges of portal tracts. This observation has consistently been recognized as a crucial aspect of biliary atresia.[22,29,31] Our research found similar histopathological characteristics of ductular reaction, including bile duct inflammation, periductal fibrosis, and bile duct proliferation.

Another study by Russo P et al., Das P et al., and Davenport M et al. highlighted that damage and loss of intra-hepatic bile ducts can be detected early in some patients through initial biopsy.[22,32,33]

Research conducted by Cocjin J et al. highlighted the effectiveness of immunohistochemical reactions using cytokeratins (CK7, CK19) and neural cell adhesion molecule (CD56) antibodies in diagnosing biliary atresia.[34] In our study, we observed ductular reaction and ductal plate malformation through positive immunohistochemical staining with cytokeratins CK7 and CK19, as well as CD56. CK7 is indicative of ductular proliferation, as it reacts with biliary epithelial cells and progenitor hepatocytes. CD56 is useful for identifying immature biliary epithelial cells and confirming ductal plate malformation. The presence of CK7 and CD56 can suggest newly formed bile structures, which often lack a lumen, indicating inhibited maturation in BA. CK19 positivity is observed in all three stages of the ductal plate: the initial, remodeling, and remodeled stages.[35]

Several studies by Landing BH et al., Azar G et al., Sergi C et al., Raweily EA et al., and Yamaguti DCC noted that intrahepatic bile duct damage and loss can be detected early in some patients through initial biopsy. Intraepithelial lymphocytes and signs of epithelial injury, such as nuclear enlargement, stratification, overlapping, and cytoplasm loss, are often observed, followed by duct destruction [36-40]. Our study found similar features, including intraepithelial lymphocytes with nuclear enlargement, stratification, overlapping, and cytoplasm loss on initial biopsy.

Furthermore, research conducted by Russo P et al., Das P et al., and Davenport M et al. indicated that interlobular bile duct damage or inflammation was observed in 31% to 94% of cases.[22,32,33] In contrast, studies by Lee H et al. and Yamaguti DCC et al. identified bile duct loss in approximately 8% of the initial biopsy samples.[40,41]

In biliary atresia, lymphocytic inflammation within the portal tracts is typically mild, with other inflammatory cells such as eosinophils, plasma cells, and macrophages present but not predominant. A study by Pacheco MC et al. revealed significant portal inflammation in certain patients.[42]

CONCLUSION

Biliary atresia is a complex condition influenced by multiple factors, with outcomes varying based on the timing of surgical intervention and histological findings. It is one of the most significant liver diseases affecting newborns. From a pathological standpoint, it is crucial to identify clear signs of biliary obstruction to make an accurate diagnosis, distinguishing it from Neonatal Giant Cell Hepatitis, which is a closely related differential diagnosis. Early

detection through prenatal screening and prompt identification after birth can help prevent further liver damage. Ultimately, a well-coordinated, multidisciplinary approach to suspected biliary atresia cases is essential for timely diagnosis and achieving the best possible outcomes.

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