ORIGINAL ARTICLE

Diversity of Diseases Causing Neonatal Jaundice - Experience of Pediatric Surgery Unit in Saidu Group of Teaching Hospital, Swat

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Abstract

Background: Rapidly establishing the cause of neonatal jaundice is very important. Delaying an exact diagnosis can have lifelong consequences. Timely treatment, if possible, is a universal solution.

Objectives: This study aimed to report on the prevalence and diagnostic approach of the myriad disorders causing persistent neonatal jaundice in patients presenting to the pediatric surgery unit of Saidu Group of Teaching Hospital (SGTH), Swat.

Materials and Methods: Clinical chemistry and cause of disease were retrospectively analyzed in 40 infants (male n=21, 52.5%) that had presented with neonatal jaundice to SGTH, from January 2020 to December 2021.

Results: The mean age was 45±30 days (range 20-90 days). The most common cause of persistent neonatal jaundice was biliary atresia (52 %) followed by idiopathic causes (15%) and progressive familial intrahepatic cholestasis (10%).

Conclusion: Although in the last 2 decades, new causes such as the progressive familial intrahepatic cholestasis followed by idiopathic cases have been identified biliary atresia still represents the major cause of persistent neonatal jaundice.

Keywords: *Neonatal jaundice, biliary atresia, newborn, diversity, disease, pediatric surgery.*

INTRODUCTION

Word Jaundice (from French, jaune) means yellow. Bilirubin deposition in tissues, leading to a yellowish appearance of skin, sclera and mucus membrane is called jaundice or icterus. It is one of the most common clinical signs in neonates during the initial 14 days of life [1]. This physical sign indicates hyperbilirubinemia (serum bilirubin level > 1.5 mg/dL).

Jaundice in neonates, is a common problem (4 in 10000 live births) [2]. It has many causes, even breastfeeding in some babies [3]. It is abnormal when persistent for more than 2 weeks [4]. Jaundice must be ruled out by determination of the conjugated bilirubin in addition to the total bilirubin level. Jaundice in neonates, longer than 2 weeks is a serious disorder [5]. It requires rapid assessment and investigation. Some of the etiologies can lead to preventable acute and chronic bilirubin encephalopathy or kernicterus and even death [6]. These complications are markedly reduced in the developed world *e.g.*, USA and UK [7]. On the other hand, it is a major public health issue in Africa [8] and the subcontinent [9]. Late presentation and wrong diagnosis are common problems in these areas [10]. Therefore, hyperbilirubinemia is exclusively investigated due to its multiple etiologies [11]. New diagnostic tools and therapeutics have helped to deal with this problem effectively [12].

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A neonate can have persistent jaundice due to several pathologies [13]. One-third is due to biliary atresia [14]. Timely diagnosis of this disease is very important [15]. Surgery (Kasai portoenterostomy) is therapeutic in 33% of diagnosed cases [16]. A delay in exact diagnosis leads to miserable outcomes [17].

Persistent neonatal jaundice needs accurate diagnosis and a centralized data bank [18]. It helps in the identification of its etiology in specific areas. It leads to better patient outcomes [19]. Our part of the world lacks a central data bank regarding this fatal problem. Our study will determine various common causes of persistent neonatal jaundice in our country. It also reviews previous and modern diagnostic approaches. It will also contribute valuable data from northern Pakistan.

MATERIALS AND METHODS

This was a retrospective analysis of data and a single-center study. It includes data of all infants with persistent jaundice, presented to the Pediatric Surgery Unit of Saidu Group of Teaching Hospital (SGTH) Swat. The study was started after approval from the Ethical Review Board (ERB) of Saidu Medical College (SMC), Swat. Data was collected from 1st January 2020 to 31st December 2021. Neonatal jaundice was defined as persistent neonatal jaundice when conjugated hyperbilirubinemia exhibiting a conjugated (direct) bilirubin of more than 1 mg/dL (in combination with a total bilirubin of <5.0 mg/dL or a direct bilirubin fraction of >20% of the total). After 4 weeks, a direct bilirubin >0.2 mg/dL was used as a cut-off value [20]. All patients were given oral A and D vitamins (AD plus drops, 1 drop orally per 24

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hours) and injection of vitamin K (inj. konacain, 1ml/kg) if bleeding disorders were present. Bleeding disorders are a group of disorders that share the inability to form a proper blood clot. Patient with incomplete data, transient jaundice and age >1 year was excluded.

Patients with persistent neonatal jaundice had a thorough workup. It begins with a detailed history (*i.e*., biodata, chief complaints, history of present illness, history, drug history, surgical history, family history, *etc*.) and physical examination (*i.e*., jaundice, anemia, koilonychia, clubbing, edema, abdominal distension). It was followed by hematological (for anemia, coagulation, infection,) biochemical (liver functions, electrolytes, blood glucose), serological tests (viral profile, serum albumin, hepatic functions, radiology (ultrasound assessment of liver, ascites) and liver biopsy (tissue diagnosis). Helsinki Declaration, which is a set of ethical principles regarding human experimentation developed originally in 1964 for the medical community by the World Medical Association, was followed where indicated. An exploration of related anomalies or other diseases was accomplished. Physical examination includes jaundice, face morphology (*e.g*., dysmorphic features), oxygen saturation, and weight. Investigations such as ultrasound abdomen, cardiac echo, cardiac ECG, and x rays (bony deformities), complete blood count or peripheral smear (Ruby, abbot), conjugated and unconjugated bilirubin (cobas, Roche), liver function tests, serum albumin, serum sodium, potassium and chloride (microlab, Merck), serum urea creatinine, uric acid, anemia work up, a workup for metabolic and immunodeficiency. In case of anemia investigations (LDH, haptoglobin, indirect Coombs test) should be done to find immune *versus* nonimmune anemia. Full Coagulation profiles including prothrombin time, international normalization ratio, thromboplastin time, antithrombin III, and fibrinogen level were evaluated. Bacterial, parasitic as well as virological analyses (*e.g*., hepatitis virus, cytomegalovirus, and others) were done.

Many diseases can present like biliary atresia (*e.g*., cystic fibrosis, alpha-1-antitrypsin deficiency (a1AT). These diseases were ruled out through history, examinations and investigations. To diagnose cystic fibrosis, immunoreactive trypsin (for neonates) was measured. enzyme levels were determined for alpha-1-antitrypsin deficiency diagnosis. Except for those patients who have clear diagnoses, a liver biopsy was performed in every case. Histologically, findings of ductular proliferation, portal fibrosis, and bile plugs were diagnostic for biliary atresia. The findings of intrahepatic bile stasis and giant cells were labeled as Neonatal giant cell hepatitis. If no known pathology was identified, these cases were labeled as idiopathic.

Data consisting of age, sex, address, date of hospital visit, disease diagnosis, and interventions was collected. The result was presented using numerical value, texts, percentages and frequencies (categorical variables), tables, mean, median, and standard deviation (numerical variables, based on data distribution). Mann-Whitney U test was used to compare various groups. Statistical Package for Social Science (SPSS) version 23 was used for data analysis. A p-value <0.05 was considered statistically significant.

RESULTS

During this 2-year tenure, a total of 40 infants (male n=22, 55%) were evaluated. Already diagnosed cases were (n=25, 62.5%). These babies had liver biopsies done in parent hospitals. The mean age of these babies was 30±10 days. The rest of the babies were sent for further management. The mean age was 45±30 days (range 20-90 days). Biliary atresia was the most common (n=21; 52.5%). Idiopathic cases (n=6) were 15% and progressive familial intrahepatic cholestasis (PFIC, n=4) was 10%. Amongst the 21 infants of biliary atresia, 4 (12%) were syndromic. In preterm, jaundice starts soon after birth but can present after some time (67±23 days). All of these patients had no history of neonatal (bacterial or viral) infections. It was always regressive. Bilirubin normalized 150±87 days after delivery (median 149, range 85-338 days). Table **1** shows details of other diseases. The most common cause of persistent neonatal jaundice was biliary atresia followed by other diseases.

Table 1: Details of other diseases.

Disease	Percentage	Frequency
Biliary atresia	52.5%	21
Idiopathic	15%	6
PFIC (Progressive familial intrahepatic cholestasis)	10%	4
Giant cell hepatitis	7.5%	3
Biliary sludge	2.5%	1
Alagille syndrome	5%	2
Choledochal cyst	5%	2
Biliary sludge	2.5%	1

DISCUSSION

Persistent neonatal jaundice has many etiologies. Worldwide biliary atresia is most common followed by other diseases [21]. Biliary atresia may account for up to 30% of jaundiced babies [22]. Persistent neonatal jaundice has confusing etiologies. Many were wrongly labeled idiopathic instead of neonatal hepatitis and other causes [23]. Causes Other than biliary atresia are many

but less than 20% individually [24]. This retrospective analysis of data supports this earlier research. There are some deviations from previous research (*e.g*., lower idiopathic cases than Mieli-Vergani [25] 14 *vs*. 30%). New diagnostic investigations *e.g*. liver biopsy, being more sensitive and specific are most probably the reason behind this. Data regarding PFIC (progressive familial intrahepatic cholestasis) follow previous research (10-15) % [26].

Our data analysis showed biliary atresia as the major disease-causing persistent neonatal jaundice (**Table 1**). This was a retrospective analysis and a single-center study, hence having restrictions. Generalization of results should be done in this context (**Table 1**).

In our data, we had two groups of patients *i.e*., already diagnosed and referred ones. The first group may show more severe disease, pointing toward a more severe spectrum neonatal jaundice (*e.g*. atresia). It may be the reason for the variance of results from international research or due to the very vast catchment area (around 250 sq. km) of SGTH. There is a homogeneous distribution of disease burden in the Dir, Buner, and Shangla districts. Idiopathic cases have a declining trend due to recent advances. However further studies are required to determine the etiological origin of persistent jaundice and its therapeutics in our part of the world to get national-level data.

Persistent Neonatal jaundice is a serious problem in early life [27]. It has significant mortality. It is a time-bound disorder [28]. Timely diagnosis and treatment accordingly yield very good results [29]. Globally around half of patients of persistent neonatal jaundice had biliary atresia [30]. Surgical treatment (Kasai procedure) is curative in one-third of cases [31]. Age at surgery is directly related to its success rate [32]. Certain histologic findings and repeated biliary tract infections are also responsible for the failure of the Kasai procedure. Adverse histological findings are small or absent bile ducts at the portal plate. Surgery done before 60 days of life has a much better prognosis than after 60 days.

Presentation of biliary atresia in our setup is quite late (mean age, 62 days). This is more delayed than in Western countries, *e.g*., the United States [33] and Germany (<60 days). The Kasai procedure was done after diagnosis and stabilization. The mean age of surgery was 66 days. It is the effect of delayed diagnosis. Only one-third of our patients get surgical treatment before 60 days. Although efforts are underway, corrective surgery before 60 days of life is still not routine [34]. Initial diagnosis of biliary atresia is still done by general practitioners. Neonatal jaundice is common in premature babies. It is usually self-resolving. Similarly, biliary atresia is asymptomatic in the initial 3 weeks of life. Therefore, accurate and quick diagnostic tools are very helpful. It includes a dipstick test of urine (bilirubin excretion) and a stool color card [35].

Biliary atresia has 2 types *i.e*., the fetal type and the perinatal type. The former is early onset while the latter is delayed onset (14 to 30 days of life) [36]. We are still unable to detect biliary atresia earlier. We are in dire need of awareness regarding this disease. When there is diagnostic suspicion of biliary atresia, ERCP is very sensitive and specific [37]. Unluckily we don't have this facility in our setup and also, it is not universally recommended.

In a nutshell, several conditions can present with persistent neonatal jaundice. One of the major causes is biliary atresia. Patients should be dealt with *via* a systemic approach to achieve accurate diagnosis, leading to timely treatment. This approach is lifesaving, therefore any case of persistent jaundice with or without clay-coloured stool must follow a diagnostic algorithm. If bilirubin is found more than the normal range, referral to a specialized unit is mandatory.

LIMITATION OF STUDY

This is a single-centre study and not all of the patients were referred to our unit but also to other tertiary care units. So, a combined study, collecting data from all major units will be a true representative of this disorder and its various presentations in our region.

CONCLUSION

Jaundice in neonates has multiple etiologies but biliary atresia is the leading cause. an organized approach, timely diagnosis and treatment is crucial. The majority of these diseases can be treated and most of these children can be saved from lifelong disabilities if referred to proper units in due course of time.

ETHICAL APPROVAL

Ethical approval was obtained from the Ethical Review Board of Saidu Medical College (SMC), Swat (REF letter No. 02-ERB/2022 Dated: 27-01-2022). All procedures performed in studies involving human participants were following the ethical standards of the institutional and/ or national research committee and the Helsinki Declaration.

CONSENT FOR PUBLICATION

Written informed consent was taken from the participants.

AVAILABILITY OF DATA

Data presented in this article is obtained from hospital records of SAIDU Group of Teaching Hospital seat. It can be obtained by prior permission from hospital.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORS' CONTRIBUTION

Dr. Muhammad Kabir: Writing and editing the manuscript, Data collection, Data analysis and interpretation, Drafting the article, Critical revision of the article and Final approval of the version to be published.

Dr. Saddar Rahim: Data collection, Data analysis and interpretation and Drafting the article.

Dr. Hafsa: Data collection and Data analysis and interpretation.

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