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Case Report

Syphilitic Hepatitis in Infancy Presenting with Cholestatic Jaundice and Inguinal Hernia: A Case Report

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Abstract:

Background: Congenital syphilis can involve multiple organ systems and, in rare cases, present with syphilitic hepatitis, a cause of cholestatic jaundice in infancy. Early recognition is challenging due to its non-specific presentation and overlap with other etiologies of neonatal cholestasis. This case highlights a rare case of a cholestatic infant with syphilitic hepatitis and concurrent inguinal hernia, emphasizing diagnostic challenges and management in resource-limited settings.

***Case*:** A 1-month-26-day-old infant presented with a left inguinal mass and jaundice. The mother had latent syphilis during pregnancy and received benzathine penicillin G only one week before delivery. The infant had persistent jaundice, pale stools, elevated direct bilirubin, transaminases, and alkaline phosphatase. Abdominal ultrasonography showed normal liver echotexture and gallbladder contractility, with no biliary dilatation. Based on clinical, laboratory, and maternal history, a presumptive diagnosis of biliary atresia with differential syphilitic hepatitis was made. Supportive therapy with ursodeoxycholic acid, fat-soluble vitamins, and antibiotics was initiated. The patient was referred for further evaluation by pediatric gastroenterohepatology.

***Discussion*:** The infant presented with postnatal jaundice, acholic stools, and elevated indirect bilirubin, initially raising suspicion of biliary atresia. However, the maternal history was positive for syphilis, making syphilitic hepatitis a presumptive diagnosis. Careful clinical evaluation and close serial follow-up are essential for establishing the diagnosis and guiding management. Early antenatal screening and timely maternal treatment remain key strategies to prevent vertical transmission.

Conclusion: Syphilitic hepatitis should be considered in the differential diagnosis of neonatal cholestasis, particularly in infants born to mothers with inadequately treated syphilis.

Keywords: congenital syphilis, inguinal hernia, neonatal jaundice, syphilitic hepatitis

Introduction

Congenital syphilis is a serious health problem caused by *Treponema pallidum* infection. It is transmitted vertically from mother to infant. Congenital syphilis infection in babies is often asymptomatic, so it is called "the great masquerader" because the clinical symptoms in babies are unclear and ambiguous, making it difficult to initiate treatment.[1] In Indonesia, syphilis screening is conducted during pregnancy through the antenatal triple elimination program.

The incidence of congenital syphilis in Southeast Asia has decreased by around 8% from the global burden of disease in 2016. However, it is still far from the target of eliminating congenital syphilis by 2030.[2] A research conducted at Wangaya Hospital in 2022 stated that 68.1% of cases were asymptomatic, while jaundice was the second most common manifestation (21.2%).[3] Examination of congenital syphilis in the form of Treponema Pallidum haemagglutinin assay (TPHA) and Venereal Disease Research Laboratory (VDRL) shows non-reactive results for several months in infants. Then, it led to delayed therapy, which affects the progression of the disease.

A rare complication of congenital syphilis is syphilitic hepatitis. The incidence of syphilitic hepatitis only occurs in 3% of congenital syphilis cases. Abnormally elevated liver enzymes and decreased albumin are used as indicators of diagnosis.[4] Complete eradication of *Treponema pallidum* from the liver is the main treatment of syphilitic hepatitis. The first-line treatment is penicillin as an antibiotic. This case report aims to give insight for clinicians in diagnosing syphilitic hepatitis in infants and children, especially in limited-resource settings.

Case

A female infant, 1 month and 26 days old, was brought by her mother to the Wangaya Children's pediatric outpatient clinic with the chief complaint of a lump on her left thigh, which had intermittently appeared and disappeared since September 2024. According to the mother, the infant had previously been treated by a midwife and was given only a topical analgesic spray. However, the lump continued to reappear. The patient also had desquamation on the face, body, and limbs without rash.

During the outpatient examination, the infant appeared yellowish (jaundiced), a previously unrecognized finding by the mother. The mother reported that the jaundice had been present for approximately one month (post-natal jaundice). She also stated that she was unable to provide exclusive breastfeeding for her baby due to insufficient breast milk production and work-related fatigue. Thus, the infant was primarily fed formula milk and cared for by extended family members. The infant occasionally vomited after milk feeds. It was also revealed that the infant has a history of pale-colored stools with a soft consistency. The patient's mother assumed this condition was normal.

The mother attended only two antenatal care (ANC) visits at the Primary Health Care Center during pregnancy. She rarely sought ANC due to her husband's busy work schedule and the absence of someone to accompany her. The syphilis infection was only diagnosed late in the pregnancy, between the 30th and 31st weeks of gestation.

Reactive results were found on the triple elimination tests (HIV non-reactive, HBsAg non-reactive, and TPHA is reactive), which were conducted during the 30th and 31st weeks of gestation. The mother underwent follow-up testing with VDRL and TPHA on June 6, 2024, which showed titers of 1:32 and 1:512, respectively. She was diagnosed with latent syphilis at 39 weeks of gestation and received three doses of Benzathine Penicillin G (2.4 million IU) administered on August 7, 14, and 21, 2024. Post-treatment serological results, obtained before delivery, revealed a TPHA titer 1:1.

The patient was the first child of the family, delivered via caesarean section at 39 weeks of gestation. The patient was diagnosed with latent syphilis infection with suspected intrauterine growth retardation (IUGR) due to comorbid maternal infection. The APGAR score was 6-7-8-9. The patient was born with a Finstrom score corresponding to 37 weeks and 4 days of gestational age, a low birth weight (LBW) of 2020 grams, and a birth length of 43 cm. At birth, the infant did not cry spontaneously and required neonatal resuscitation. The patient was admitted to the Neonatal Intensive Care Unit (NICU), where supportive care was provided. After the patient's condition stabilized in the NICU, a prophylactic injection of Benzathine Penicillin Antibiotic 100.000 IU was administered in the anterolateral aspect of the right thigh on August 30, 2024.

The examination for syphilis was conducted using the VDRL test, which showed a titer of 1:2. The IT-Ratio level was 0.06, indicating a possible risk of infection. The patient was hospitalized from birth until 7 days of chronological age for monitoring and supportive care until clinical improvement was observed. The patient's immunization record was incomplete, with the last documented vaccine being Hepatitis B.

The patient appeared moderately ill at the pediatric outpatient clinic visit on October 25th, 2024. At the time of examination, the infant was chronologically 1 month and 26 days old, with a body weight of 3.0 kg and a body length of 49 cm. Assessment of weight gain from birth to the current age, based on WHO weight increment plotting for 1 month, indicated growth faltering. The patient's anthropometric assessment showed a Body Height-for-Age (BH/A) z-score of -3.79 SD, indicating stunted (short stature), The Body Weight-for-Age (BW/A) z-score was -3.75 SD, consistent with underweight status. Meanwhile, the Body Weight-for-Height (BW/BH) z-score was -0.56 SD, suggesting a well-nourished condition.

On physical examination, the head showed no frontal bossing. The infant appeared lethargic but cried moderately. The conjunctiva was not pale, while the sclerae appeared icteric. The patient exhibited regular spontaneous breathing. The hair appeared to be slightly brownish. There was a secret positive on the nose with slimy consistency. No saddle nose deformity was found. The pharynx was not hyperemic, and the tonsils were not enlarged. No coated tongue was observed. Thoracic examination revealed bronchovesicular breath sounds, with no rhonchi or wheezing detected. No abnormalities were found in the abdominal area, hepatomegaly, or splenomegaly were not present. Skin was icteric (Kramer grade III-IV). Extremities appeared warm, with a capillary refill time (CRT) of less than 3 seconds. The patient was subsequently hospitalized for further examination.

Laboratory examination showed leukocyte levels of 9.50 x 10^3/µL, erythrocytes 4.21x 10^6/µL, hemoglobin 14.0 g/dl, hematocrit 40.5%, and platelet levels increased by 496 x 10^3/µL. Bilirubin analysis showed findings consistent with cholestasis, with total bilirubin levels of 11.46 (Ref: 0.2-1.0), direct bilirubin levels of 9.9 (Ref: 0.1-0.4), and indirect bilirubin levels of 1.56. The patient was also examined for coagulation factors, such as activated partial thromboplastin time (aPTT), prothrombin time (PT), and International Normalized Ratio (INR). The aPTT level was 31.2 seconds (Ref: 25.5 to 42.1), PT was 10.9 seconds (Ref: 9.5-11.7), and the INR level was 1.02. Coagulation factor studies were conducted due to the risk of gastrointestinal bleeding or perforation in congenital syphilis. Liver function tests revealed markedly elevated transaminase levels, with AST level 476 (Ref: 0-37) and ALT level 316 (Ref: 0-42). Serum albumin was within normal limits at 3.9 g/dl (Ref: 3.8-5.1). The alkaline phosphatase examination result was elevated at 439 U/L (Ref: 53-128). The C-Reactive Protein (CRP) levels were <5.0 mg/dl. Occult blood analysis was performed on the patient. Erythrocyte components were normochromic normocytic with poikilocytosis cell shapes. Leukocytes were dominated by mature lymphocytes. Platelet evaluation showed an increased count with the presence of giant platelets, indicating thrombocytosis. These findings were suggestive of an underlying liver disorder.

Urinalysis examination was performed to rule out other causes of intrahepatic cholestasis. The urinalysis examination showed yellow-colored urine, with negative results for leukocyte esterase, nitrite, ketones, urobilinogen, and bilirubin. The titers of leukocytes were 0-1 (Ref: 0-1), and no bacteria were found on the sample.

Three stool portion samples were collected from the patient. The first sample exhibits soft, pale in colour, and acholic. The second sample was also soft, pale-yellowish, and acholic. The third stool sample appeared soft with a pale-yellowish colour, and reduced acholia.

Two-phase ultrasonography showed a normal liver size and echotexture, normal echoparenchymal, sharp angles, flat edges, without visible widening of the Intra Hepatobiliary Bile Duct (IHBD) and Extra Hepatobiliary Bile Duct (EHBD), normal portal and hepatic veins, and with no visible masses/nodules/cysts. The gallbladder was normal in size, the walls were not thickened, there was no irregularity, and there were no stones or sludge. In the first phase, the size was 1.6x0.54 cm, and in the second phase, the size was 1x0.32 cm with a contraction index level of 77.61%. No free echo fluid was seen in the abdominal cavity. The results of the USG showed normal contractility of the gallbladder, no visible obstruction in the intra- or extrahepatic biliary tract, and the liver and gallbladder did not show any abnormalities.

Based on the findings from the patient's history, physical examination, and supporting examinations, the patient was diagnosed with extrahepatic cholestasis, suspected biliary atresia, and left lateral inguinal hernia, with differential diagnoses of intrahepatic cholestasis, suspected syphilitic hepatitis, and suspicion of urinary tract infection (UTI), accompanied by failure to thrive, good nutritional status. When hospitalized, the patient received D10% fluid rehydration and Cefotaxime antibiotic to treat the infection. After using antibiotics, the patient had improvement, such as a reduction to Kramer Stage III and more adequate breastfeeding. Supportive therapy with ursodeoxycholic acid (UDCA) was given 3 times daily (30 mg/kg/day) alongside multivitamin drops containing amino acids and vitamins A, D, E, and K.

After four days of inpatient treatment, the patient was referred to a higher-level hospital for further evaluation and management by a pediatric gastroenterologist and a pediatric surgeon, particularly to address other differential diagnoses of cholestasis and the suspected hernia defect.

Discussion

Most published cases of syphilitic hepatitis are from Western countries, where diagnostic resources are more readily available. According to the guidelines from the European Centre for Disease Prevention and Control, the prevalence of syphilitic hepatitis is still unclear, ranging from 0.1% to 39.8% cases per 100.000 live births.[2, 5] The condition of syphilitic hepatitis is one of the conditions of re-emerged disease besides non-communicable disease in low- and middle-income settings. According to a study by Luo et al in China, the prevalence of syphilis during pregnancy in Eastern China is around 0.3%.[2, 6] At present, many early-phase congenital syphilis cases have hidden clinical manifestations and abnormalities that are found only on laboratory examination. The pathogenesis of syphilitic hepatitis remains unclear, but it may involve immune-mediated injury induced by Treponema pallidum, which causes damage to liver cells and the intrahepatic duct system, leading to the onset of hepatitis.[4, 7] The importance of syphilis testing is to eliminate mother-to-child transmission of syphilis. If a pregnant woman has latent stage and asymptomatic

syphilis, appropriate management can prevent disease progression that may cause irreversible organ damage and other clinical manifestations. Based on a case-series study by Salome et al. in Italy, proper antenatal screening and early initiation of therapy were protective against vertical transmission to children (95% CI: 1.2–1.4; $p < 0.001$).[5]

This case differs from recently published reports and provides crucial insight into the diagnosis of syphilitic hepatitis presenting with postnatal jaundice in limited resources. The presumptive diagnosis for syphilitic hepatitis on limited source can be made using the diagnostic criteria of syphilitic hepatitis proposed by Mullick 2004 such as abnormal liver enzyme levels, evidence of syphilis infection, and exclusion of other diseases.[8]

In this case, the patient was born to a mother diagnosed with latent syphilis. The patient presented with jaundice, which was likely triggered by hepatocellular injury leading to bile duct obstruction. If left untreated, disease progression may result in liver cirrhosis. In this case, physical examination did not reveal hepatosplenomegaly. This is consistent with the clinical course, as hepatosplenomegaly in congenital syphilis most commonly presents during the neonatal period.[4, 9] Other stigmata of syphilis hepatitis, such as saddle nose deformity or chancre lesions, were not found in this patient.

The presence of acholic stools is suggestive of cholestasis, in which biliary atresia must remain the primary suspicion until definitively excluded. Although the ultrasound did not reveal a triangular cord sign as a sign of biliary atresia, three consecutive acholic stool examination must be considered to diagnose biliary atresia as a differential diagnosis. Due to cost-benefit considerations, the gamma-glutamyl-transferase (GGT) examination and intraoperative cholangiography were not assessed on this patient. Instead, a two-phase ultrasound examination was performed to examine bile contractility, with the patient was fasted for 4-5 hours before the examination. No abnormalities were reported from the ultrasound examination, suggesting a lower possibility of biliary atresia (BA).[10] However, further work-up is required to exclude BA as highlighted in previous publications.[11, 12]

Nevertheless, A study in China found that clinicians could diagnose cases of syphilitic hepatitis by relying on AST examination (median 123.7 U/L) and a decrease in albumin level (hypoalbuminemia). In this case, the AST was high (476), and the albumin level remained within the normal limit but closer to the lower end of the reference range. Hypoalbuminemia may indicate extensive hepatocellular damage, as albumin plays a key role in the transport of indirect bilirubin. A decreased albumin level also shows a chronic course of jaundice, and in this patient, early signs of hypoalbuminemia were already observed.[5] In this case, alkaline phosphatase was elevated, at 439 U/L. Elevated alkaline phosphatase is a marker of hepatocellular

damage related to spirochete dissemination in the liver, and it may also indicate bile duct obstruction, manifesting as jaundice.[5, 13]

Evaluation of the infant using TPHA or VDRL titers is essential to support the diagnosis of congenital syphilis. Rapid plasma reagen (RPR) as an indirect biomarker of damage to the cells of the spirochete and TPHA examinations in infants are recommended to be postponed, as testing too early may result in false-negative results due to "incubating congenital syphilis." Confirmatory examination is therefore advised at around 3 months of age. Cerebrospinal fluid analysis examination was not available at health facilities, so it was not performed.

Based on the clinical findings and supporting findings, the physician made a working diagnosis of possible congenital syphilis. In developed countries, liver biopsy is the diagnostic modality of choice to exclude other potential causes of liver disease. However, the diagnosis of syphilitic hepatitis can often be established based on clinical manifestations and basic laboratory examinations. *Treponema pallidum*, the causative agent of syphilis, is classified as a non-hepatotropic pathogen but has been recognized as a rare etiology of unidentified hepatitis in children.[9]

The patient received a single dose of prophylactic Benzathine Penicillin G 50 mg per kg/day after birth, in accordance with the guidelines from the Indonesia Ministry of Health. Benzathine penicillin injection was also given to the mother less than four weeks before delivery despite her TPHA titer being 1:1. In this patient, a Jarisch-Herxheimer reaction following penicillin administration was ruled out, as no transient worsening occurred within the typical 24-hour window.[9] In congenital syphilis, VDRL results rarely become seronegative following therapy; therefore, monthly clinical follow-up is recommended. The patient's parents were advised to come every month to evaluate the TPHA and VDRL titers.[7]

Therapy also focuses on cholestasis, including supplementation with fat-soluble vitamins A, D, E, and K for three months. This approach has been shown to support the resolution of jaundice, as measured by either the Kramer scale or bilirubin levels. Additionally, the administration of UDCA was beneficial as a hepatoprotector, aiding in bile flow stimulation and fat absorption, particularly in cases of liver injury related to syphilis infection.[5, 14]

Jaundice with elevated indirect bilirubin in infants older than 14 days should prompt consideration of abnormal underlying conditions, with cholestasis being an important differential to exclude. In infants under one year of age, cholestasis may occur due to immaturity of the hepatobiliary system, such as underdeveloped bile canaliculi and impaired bile flow. However, pathological causes must be carefully excluded, particularly vertically transmitted infections like those in the TORCH group, which

can be screened during antenatal care. The condition of intrahepatic cholestasis, besides congenital syphilis, must be considered, such as congenital hypothyroidism or TORCH infection, especially CMV infection, with prominent manifestations like blueberry muffin rash, hepatosplenomegaly, microcephaly, congenital heart defect, intellectual disability, or lethargy. These manifestations were not identified in the patient's clinical findings. Nevertheless, the possibility of CMV infection cannot be entirely excluded. Therefore, further work-up for CMV infection or other potential infectious etiologies should still be performed.

Clinicians should still consider urinalysis examination to rule out the cause of cholestasis due to urinary tract infection (UTI). The condition of urinary tract problems in infancy tends to be asymptomatic and is often skipped from evaluation.[15]

Several studies also mention that patients who experience intrahepatic cholestasis tend to experience growth failure. Research by Serra in Palermo, Italy, showed growth failure in affected infants was associated with feeding difficulties and an inadequate sucking reflex, which compromised the adequacy of nutritional intake and the ability to meet dietary needs. In this patient, evidence of growth faltering was observed.[15]

The lump in the left thigh was clinically suspected to be an inguinal hernia, with findings limited to one side. This is noteworthy because inguinal hernias are more commonly bilateral in preterm infants, whereas the patient in this case was born at term (39 weeks of gestation). The etiology of inguinal hernia involves a defect during embryogenesis, with the persistent processus vaginalis during the formation of the labia majora or minora in girls. On physical examination, the lump was associated with mild erythema of the overlying skin. However, the absence of a pediatric surgeon at our facility restricted confirmatory diagnosis and further evaluation. An abdominal CT scan was also not performed due to its limited diagnostic facility.[16] In this case, the inguinal hernia may represent an incidental finding, and a direct causal relationship with congenital syphilis cannot be established.

This case demonstrates that congenital syphilis diagnosed in the late third trimester may affect the developmental process of fetal organs. The cause of the defect in the formation of the inguinal ligament, which leads to an inguinal hernia in this patient, remains unclear. Salome's study in Italy indicated that when syphilis treatment is given in the third trimester, there is limited time for nontreponemal titers to decrease before delivery.[5, 17] Managing early latent syphilis in the mother is crucial to reduce the transmission of maternal syphilis to the fetus by 98%.

The prognosis in this patient has not been determined. It depends on the adequacy of the therapy, whether the jaundice lasts for a long time and leads to decreased consciousness (kernicterus), and whether the biopsy examination shows massive

inflammation. According to the study by Yang et al, the liver function test significantly improved and decreased gradually compared to before treatment.[7, 14] From this case, we can learn that we must evaluate liver function every two weeks to maintain the adequacy of post-treatment antibiotics.[7]

Clinical implication for this case is that it can be a lesson learn for clinicians, mainly for the condition of syphilitic hepatitis, which is diagnosed at a late stage, because in the early phase, it is asymptomatic. Syphilitic hepatitis in many recent studies manifested with a multiorgan involvement until a fatal outcome. Today, the incidence of syphilitic hepatitis is still increasing because of the lack of antenatal screening with various and unspecified clinical manifestations. It's necessary for early recognition of an infant with probable congenital syphilis.

The key limitation of this case report is that the diagnosis of syphilitic hepatitis is still a presumptive diagnosis. Diagnostic tools for supporting diagnosis and the absence of follow-up measurements of bilirubin, ALT, and AST levels after therapy limited the ability to assess treatment response. This case report also did not perform GGT, CMV-PCR, or thyroid-stimulating hormone (TSH). Additionally, a liver biopsy could not be performed due to restricted resources. However, several literatures indicate that liver biopsy in similar cases may reveal microscopic findings such as localized leukocyte infiltration, lobular inflammation, and hepatocellular necrosis.[7, 15 11,14] The patient was referred to the referral hospital for further examination and comprehensive management by a pediatric gastroenterohepatologist.

Conclusion

This is a rare case of infant syphilitic hepatitis that was initially confounded with biliary atresia in a resource-limited setting. Syphilitic hepatitis should be considered as a differential diagnosis in neonatal cholestasis born to mothers with syphilis. The liver function should be monitored regularly in a patient with a history of syphilis treatment. Syphilis hepatitis is rarely diagnosed and often overlooked by clinicians. Its manifestations may mimic other causes of neonatal jaundice, leading to delays in appropriate management. As syphilis is a re-emerging but preventable and treatable disease, it is important to increase awareness and adherence to antenatal screening protocols, as outlined by the Indonesia Ministry of Health.

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Conflict of Interest

None declared

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References

1. Sankaran D, Partridge E, Lakshminrusimha S. Congenital syphilis-an illustrative review. Children (Basel). 2023;10(8). https://doi.org/10.3390/children10081310
2. Gilmour LS, Walls T. Congenital syphilis: A review of global epidemiology. Clin Microbiol Rev. 2023;36(2):e0012622. https://doi.org/10.1128/cmr.00126-22
3. Mahadewi IGA, Hsieh PP, Beatrice C, Indrawan IGDK. Gambaran karekteristik kejadian sifilis kongenital di rsud wangaya, kota denpasar. Intisari Sains Medis. 2022;12(2):362-66.
4. Supronowicz Ł, Rogalska M. Syphilitic hepatitis. Clin Exp Hepatol. 2024;10(1):9-13. https://doi.org/10.5114/ceh.2024.136235
5. Salomè S, Cambriglia MD, Montesano G, Capasso L, Raimondi F. Congenital syphilis: A re-emerging but preventable infection. Pathogens. 2024;13(6). https://doi.org/10.3390/pathogens13060481
6. Luo H, Qiu L, Wu Y, Zhang X. Growth in syphilis-exposed and -unexposed uninfected children from birth to 18 months of age in china: A longitudinal study. Scientific Reports. 2019;9. https://doi.org/10.1038/s41598-019-40134-3
7. Yang H, Zhang H, Wang C, Pang L. An analysis of the clinical features of children with early congenital syphilis and syphilitic hepatitis. BMC Pediatr. 2021;21(1):498. https://doi.org/10.1186/s12887-021-02932-5
8. Mullick CJ, Liappis AP, Benator DA, Roberts AD, Parenti DM, Simon GL. Syphilitic hepatitis in hiv-infected patients: A report of 7 cases and review of the literature. Clin Infect Dis. 2004;39(10):e100-5. https://doi.org/10.1086/425501
9. Ringoringo H, Tambunan K, Khalis F, Nawati F, Permana Y. Amoxicillin as an option in congenital syphilis management: A case report. Open Access Macedonian Journal of Medical Sciences. 2022;10:208-11. https://doi.org/10.3889/oamjms.2022.10191
10. Padua-Zamora AP, Dios MPRd, Gregorio GEV. Neonatal cholestasis secondary to congenital syphilis. Acta Medica Philippina. 2023;57. https://doi.org/https://doi.org/10.47895/amp.vi0.3787
11. Ogawa K, Kawase K, Sugiura T, Yasui T, Yamagata S, Watanabe T, Kawabe Y. Case report: Management of cholestasis associated with congenital syphilis. Frontiers in Pediatrics. 2020;8. https://doi.org/10.3389/fped.2020.607506
12. Munoz A, Crane T, Hazboun R, Banerji A, Baerg J. Surgical consultation in neonates with congenital syphilis. Journal of Pediatric Surgery Case Reports. 2019;42:4-8. https://doi.org/https://doi.org/10.1016/j.epsc.2018.12.013
13. Isella V, Dewi MR. Kolestasis neonatal di rumah sakit umum daerah wangaya, bali. Cermin Dunia Kedokteran. 2021;48(9):346 - 9. https://doi.org/10.55175/cdk.v48i9.126
14. Delle Cave V, Zito Marinosci G, Ferrara D, Esposito F, Lo Vecchio A, Sciveres M, et al. Syphilitic hepatitis in infants, the forgotten disease that hepatologists have to brush up on: From a case series to a revision of literature. Eur J Pediatr. 2024;183(11):4939-49. https://doi.org/10.1007/s00431-024-05698-x
15. Serra G, Carta M, Di Pace MR, La Sala E, Piro E, Salerno S, et al. Congenital syphilis in a preterm newborn with gastrointestinal disorders and postnatal growth restriction. Ital J Pediatr. 2022;48(1):205. https://doi.org/10.1186/s13052-022-01404-5
16. Yeap E, Nataraja R, Pacilli M. Inguinal hernias in children. Australian Journal of General Practice. 2020;49:38-43.
17. Pereira TN, Walsh MJ, Lewindon PJ, Ramm GA. Paediatric cholestatic liver disease: Diagnosis, assessment of disease progression and mechanisms of fibrogenesis. World J Gastrointest Pathophysiol. 2010;1(2):69-84. https://doi.org/10.4291/wjgp.v1.i2.69