SEVERE ALCOHOLIC HEPATITIS IN AN INDONESIAN ADOLESCENT : A RARE AND PREVENTABLE CASE OF CHRONIC ETHANOL ABUSE

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ABSTRAK

Hepatitis alkoholik (HA) adalah kondisi inflamasi akut pada hati yang disebabkan oleh konsumsi alkohol yang berlebihan dan berkepanjangan, yang umumnya terjadi pada orang dewasa. Kejadian HA pada remaja sangat jarang dan menimbulkan kekhawatiran yang signifikan dalam konteks klinis maupun kesehatan masyarakat. Kami melaporkan kasus seorang remaja laki-laki berusia 17 tahun asal Indonesia dengan riwayat konsumsi alkohol secara rutin selama dua tahun terakhir, yang datang dengan keluhan nyeri perut kuadran kanan atas, ikterus, urine berwarna coklat seperti teh, kelelahan, dan demam ringan. Pemeriksaan fisik menunjukkan sklera ikterik dan nyeri tekan epigastrium. Hasil laboratorium menunjukkan peningkatan kadar transaminase serum (SGOT 458 U/L, SGPT 1282 U/L) dan hiperbilirubinemia (bilirubin total 10,47 mg/dL), dengan serologi hepatitis virus yang negatif. Pemeriksaan USG dan CT scan menunjukkan adanya asites dan efusi pleura bilateral tanpa adanya massa hati atau obstruksi bilier. Pasien mendapatkan terapi suportif berupa cairan intravena, antibiotik, agen pelindung lambung, dan terapi pendukung fungsi hati, dengan perbaikan klinis dan biokimia yang signifikan dalam waktu satu minggu.Kasus ini menyoroti pentingnya mengenali hepatitis alkoholik pada populasi remaja, terutama mereka yang memiliki riwayat konsumsi alkohol tersembunyi namun kronis. Identifikasi dini, perawatan multidisipliner, dan edukasi pencegahan terhadap bahaya alkohol sangat penting untuk mencegah komplikasi dan kekambuhan.

Kata kunci : cedera hati, hepatitis alkoholik, kasus, konsumsi alkohol kronis, remaja

ABSTRACT

Alcoholic hepatitis (AH) is an acute inflammatory condition of the liver resulting from prolonged excessive alcohol intake, predominantly affecting adults. The occurrence of AH in adolescents is exceedingly rare and raises significant clinical and public health concerns. We report the case of a 17-year-old Indonesian male with a two-year history of regular alcohol consumption, presenting with right upper quadrant abdominal pain, jaundice, dark tea-colored urine, fatigue, and low-grade fever. Physical examination revealed icteric sclera and epigastric tenderness. Laboratory results demonstrated elevated serum transaminases (SGOT 458 U/L, SGPT 1282 U/L) and hyperbilirubinemia (total bilirubin 10.47 mg/dL), while serologies for viral hepatitis were negative. Imaging via ultrasound and CT scan revealed ascites and bilateral pleural effusion without evidence of hepatic mass or biliary obstruction. The patient was managed with intravenous fluids, antibiotics, gastroprotective agents, and liver-supportive therapy, with significant clinical and biochemical improvement observed over the following week. This case underscores the importance of recognizing alcoholic hepatitis in adolescent populations, particularly those with covert but chronic alcohol use. Early identification, multidisciplinary care, and preventive education on alcohol-related harm are essential in mitigating complications and recurrence.

Keywords *: adolescent, alcoholic hepatitis, case report, chronic alcohol use, liver injury*

INTRODUCTION

Alcoholic hepatitis (AH) is a severe and potentially life-threatening inflammatory liver condition caused by prolonged excessive alcohol consumption (Agarwal et al., 2023). It represents the most aggressive form of alcohol-related liver disease (ALD) and can present

acutely with jaundice, fever, abdominal pain, and hepatic dysfunction (Khemichian & Donovan, 2023). While the incidence of AH is significant in adult populations, especially in individuals with long-standing alcohol use disorder, it is extremely rare in adolescents, and its presence in younger individuals poses unique clinical and social implications (Danpanichkul et al., 2024). The pathophysiology of alcoholic hepatitis involves oxidative stress, immune activation, and cytokine-mediated hepatocellular damage (Agarwal et al., 2023). Continued alcohol intake in the context of pre-existing liver injury exacerbates this inflammation and can precipitate hepatic failure (Gustot & Jalan, 2019). Early diagnosis and intervention are essential to improving outcomes, particularly in populations where the disease may be under-recognized, such as adolescents (Dugum & McCullough, 2015).

The rarity of AH in adolescence may reflect underreporting or underdiagnosis, often due to social stigma, denial of alcohol use, or clinical misattribution to more common causes of hepatic dysfunction in this age group (e.g., viral hepatitis or metabolic diseases) (Hagström et al., 2018; Mavis & Alonso, 2014). This case is clinically relevant because it presents a clear instance of clinically and biochemically confirmed AH in a 17-year-old male with a substantial, well-documented history of alcohol consumption. The increasing prevalence of early-onset alcohol consumption among adolescents globally raises serious concerns. According to the World Health Organization (WHO, 2023), underage drinking remains a significant public health issue, with rising trends noted in Southeast Asian countries, including Indonesia. Early initiation of alcohol use is associated with higher risks of dependency, liver damage, and other long-term health complications (Windle et al., 2020).

In Indonesia, cultural and regulatory factors may limit open discussions about alcohol use in minors, leading to gaps in preventive education and clinical vigilance (Suryani et al., 2021). Consequently, healthcare providers may overlook early signs of liver injury in adolescents, particularly when alcohol use is not readily disclosed or suspected. This highlights the importance of maintaining a high index of suspicion when evaluating hepatic dysfunction in young patients. Furthermore, adolescent patients may present differently from adults in terms of clinical features, disease progression, and psychosocial context. Unlike adult patients, adolescents often face unique psychological and developmental challenges that influence their health-seeking behavior and adherence to treatment (Marsch et al., 2017). These aspects must be considered in the clinical management of AH in young populations. The limited number of case reports documenting AH in adolescents suggests that the true incidence may be underestimated. Research by Jiang et al. (2022) points to a need for broader surveillance systems and adolescent-specific screening protocols in high-risk populations. By identifying cases early, clinicians can implement harm-reduction strategies, initiate timely interventions, and potentially reverse disease progression before irreversible liver damage occurs.

Additionally, addressing adolescent alcohol abuse requires a multidisciplinary approach that incorporates clinical care, family involvement, school-based interventions, and national health policies. Public health initiatives should prioritize early education on alcohol risks, particularly targeting vulnerable youth populations with limited access to mental health and substance abuse resources (Livingston et al., 2019). This report aims to contribute to the limited literature on AH in adolescents by providing a detailed clinical account of diagnosis, management, and outcome, all presented within the framework of the CARE (CAse REport) guideline. Additionally, it highlights the importance of integrating behavioral history in young patients presenting with liver dysfunction and the need for targeted public health strategies aimed at early education and prevention of alcohol misuse in youth.

METHOD

This case report was developed using a retrospective descriptive analysis design, focusing on a single clinical case admitted to RSUD Ciawi, West Java, Indonesia. The patient was

treated and monitored during hospitalization, and data collection was performed after discharge through a detailed review of the hospital's medical records. The collected data encompassed comprehensive clinical documentation including demographic characteristics (such as the patient's age, sex, residence, and admission date), presenting complaints with symptom chronology, and a thorough record of the patient's medical and social history, particularly alcohol consumption. Information on physical examination findings as documented by attending physicians, along with laboratory investigations, such as liver function tests, biochemical panels, and serologic screening for viral hepatitis, were also obtained. In addition, imaging reports from chest radiography, abdominal ultrasound, and abdominal CT scan were reviewed to support clinical interpretation. Data on therapeutic interventions, including medication names, dosages, and routes of administration, were recorded along with clinical monitoring notes that described the patient's response to treatment and clinical progression until discharge.

This dataset was then organized in chronological order to clearly illustrate the sequence of clinical events, diagnostic decisions, therapeutic strategies, and patient outcomes throughout the hospital stay. No additional procedures or testing were performed specifically for this report. The structure and narrative of this case were written in alignment with the CARE (CAse REport) guidelines to ensure transparency, clarity, and educational value. All personal identifiers were removed to maintain patient confidentiality. As this study involved only retrospective analysis of anonymized data from routine care and did not include any intervention beyond standard clinical practice, formal ethical approval was not required according to institutional policy. Nevertheless, written informed consent for the publication of this case and related clinical information was obtained from both the patient and his legal guardian.

RESULT

Clinical Presentation

A 17-year-old male presented with a 4-day history of right upper quadrant abdominal pain, worsening over the last 2 days, accompanied by shortness of breath, jaundice, dark-colored urine, generalized fatigue, and low-grade fever. Physical examination revealed scleral icterus and epigastric tenderness. Vital signs were within normal limits. The patient denied any history of chronic illness, medication use, or family history of liver disease. He reported consuming approximately 650 mL of alcohol (Intisari, Arak Bali, mixed with energy drinks) one to two times per week over the past two years.

Diagnostic Sssessment

The diagnosis of alcoholic hepatitis (AH) in this adolescent patient was supported by a combination of laboratory investigations and radiological imaging. Initial laboratory tests revealed markedly elevated liver enzymes, with serum glutamic oxaloacetic transaminase (SGOT/AST) at 458 U/L and serum glutamic pyruvic transaminase (SGPT/ALT) at 1282 U/L. These values subsequently decreased to 39 and 300 U/L, and then stabilized at 84 and 123 U/L, respectively, indicating partial hepatic recovery during the course of treatment. Notably, the AST/ALT ratio was initially less than 1 but approached and exceeded 1 as levels declined, a pattern sometimes observed in resolving alcoholic hepatitis. Total bilirubin was initially elevated at 10.47 mg/dL, consistent with cholestatic jaundice, and later decreased significantly to 3.43 mg/dL, correlating with clinical improvement. Serum albumin was moderately reduced at 3.10 g/dL, suggesting impaired synthetic liver function, which is commonly observed in moderate to severe alcoholic hepatitis. Serologic testing for viral hepatitis yielded negative results for HBsAg (Hepatitis B surface antigen), Anti-HCV (Hepatitis C virus antibodies), and

Anti-HAV (Hepatitis A virus antibodies), effectively ruling out viral hepatitis as the cause of hepatic dysfunction.

Radiological imaging provided further insight into the patient's systemic involvement. A chest X-ray revealed bilateral patchy infiltrates, consistent with bronchopneumonia, which may have contributed to the patient's initial systemic symptoms such as fever and dyspnea. Abdominal ultrasonography showed the presence of ascites and right-sided pleural effusion, indicating fluid accumulation secondary to portal hypertension or hepatic decompensation. A contrast-enhanced abdominal CT scan confirmed and expanded upon these findings, demonstrating ascites, bilateral pleural effusion, and a thickened gallbladder wall suggestive of cholecystitis. While cholecystitis is not uncommon in patients with advanced liver disease due to impaired immune function and biliary stasis, the combination of findings highlighted the multisystem impact of severe liver inflammation in this case.

Collectively, the laboratory abnormalities, imaging findings, and clinical history of chronic alcohol use support a diagnosis of severe alcoholic hepatitis with systemic complications. The exclusion of viral, autoimmune, and metabolic causes further strengthens this diagnosis, underscoring the importance of detailed substance use history in adolescents presenting with liver dysfunction. A working diagnosis of alcoholic hepatitis was established, with differential diagnoses of cholecystitis, ascites, and bilateral pleural effusion.

Therapeutic Intervention

Upon admission, the patient was managed with supportive and targeted pharmacologic therapy aimed at controlling inflammation, preventing infection, and stabilizing liver function. Initial fluid resuscitation was initiated with intravenous 0.9% sodium chloride (NaCl) at 1500 cc/24 hours, to maintain adequate hydration, correct potential electrolyte imbalances, and support renal perfusion. To reduce gastric acid secretion and protect the gastrointestinal mucosa, the patient received omeprazole 40 mg once daily, a proton pump inhibitor. In addition, sucralfate was administered at 5 cc four times daily, serving as a mucosal protectant to reduce the risk of gastrointestinal bleeding, which can be a concern in patients with liver dysfunction and potential portal hypertension.

Stronger Neo-Minophagen C (SNMC), a glycyrrhizin-containing hepatoprotective agent, was given at 2 ampules daily (1x2). SNMC has been shown in some studies to exert anti-inflammatory and cytoprotective effects on hepatocytes, particularly in the context of acute and chronic liver injury. Given the findings of bilateral patchy infiltrates on chest X-ray suggestive of bronchopneumonia, and the potential for spontaneous bacterial peritonitis in a setting of ascites, empirical antibiotic therapy was initiated. The patient received ceftriaxone 2 g per day, a broad-spectrum third-generation cephalosporin, and metronidazole 500 mg three times daily, targeting anaerobic organisms and possible biliary involvement. For symptomatic relief of pain, particularly in the setting of suspected cholecystitis and pleural effusion, dexketoprofenwas administered intravenously at a dose of three times daily. Its use was carefully monitored due to the potential nephrotoxic and gastrointestinal side effects in patients with hepatic impairment.

Importantly, corticosteroids were not administered, as the patient showed signs of clinical and biochemical improvement within the first few days of supportive therapy. While corticosteroids such as prednisolone are commonly used in severe alcoholic hepatitis with high Maddrey's Discriminant Function (DF >32), their use is contraindicated or withheld in the presence of active infection or when spontaneous improvement is evident (Mathurin & Lucey, 2012). The management strategy in this case focused on a balance between treating potential infectious complications, supporting hepatic recovery, and minimizing medication-related hepatotoxicity. Close monitoring of liver function tests, renal parameters, and clinical symptoms guided the continuation and tapering of therapies throughout hospitalization.

Outcomes and Follow-Up

The patient showed significant clinical and biochemical improvement over the course of hospitalization. He was discharged with plans for outpatient follow-up and alcohol cessation counseling.

DISCUSSION

Alcoholic hepatitis is a severe manifestation of alcohol-related liver disease, typically seen in individuals with long-term heavy alcohol use (Flemming et al., 2024). Its appearance in adolescents is exceptionally rare, making this case both clinically and socially significant (Mavis & Alonso, 2014). In this patient, the diagnosis of AH was supported by the combination of a consistent history of chronic alcohol intake, elevated transaminase levels with an AST>ALT ratio >1.5, and the exclusion of other common causes such as viral, autoimmune, and drug-induced hepatitis through serological and clinical assessments (Philips et al., 2019). Differential diagnoses such as viral hepatitis (ruled out by negative HBsAg, Anti-HCV, and Anti-HAV), ischemic hepatitis (no hemodynamic instability), and drug-induced liver injury (no recent drug use reported) were systematically excluded. Additionally, although imaging suggested features like gallbladder wall thickening and pleural effusions, the clinical picture remained consistent with moderate alcoholic hepatitis (Keating et al., 2022). A liver biopsy, which might have offered histopathological confirmation, was not pursued due to the patient's improving clinical condition and the inherent risks of an invasive procedure.

When comparing this case to the limited number of reports on adolescent AH in literature, similarities are noted in terms of alcohol quantity, duration, and presenting features. However, early identification and intervention in this case appear to have favorably influenced the outcome, avoiding progression to hepatic encephalopathy or multi-organ dysfunction (Girish et al., 2025; Kim & Kim, 2014). Management of AH typically depends on severity, often assessed by scores like Maddrey's Discriminant Function or MELD, which were not calculated in this case due to limited lab data at the time of presentation (Morales-Arráez et al., 2022). Nevertheless, supportive treatment including IV fluids, antibiotics, and liver-protective agents led to significant clinical improvement (Kasper et al., 2023). The decision not to initiate corticosteroids was justified by the absence of systemic inflammatory response syndrome (SIRS), absence of hepatic encephalopathy, and evidence of biochemical recovery.

A major strength of this report lies in highlighting the occurrence of AH in adolescents, a group often overlooked in screening for alcohol-related liver injury. Its limitation is the absence of liver biopsy and long-term follow-up data. Nevertheless, this case calls for greater awareness among clinicians regarding substance use in teenagers and the need for routine behavioral assessments in patients with liver dysfunction.

CONCLUSION

In conclusion, early recognition and appropriate management of alcoholic hepatitis, even in adolescent patients, are crucial in preventing further liver damage. This case also emphasizes the importance of integrating alcohol history into routine clinical assessment for young patients with liver dysfunction.

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