Seema Sutay [2025]. International Journal of Medical Justice, IJMJ,
Volume 3, Issue 2: July-December 2025 [E-ISSN: 2583-7958]
International ISSN [CIEPS]: 3006-208X[Print] 3006-2098[Online]
Content list Available at ijmj.net

International Journal

of Medical Justice

Journal Homepage: https://www.ijmj.net

Case Report:

Case Report: Autopsy Findings in Fatal Budd-Chiari Syndrome – A Rare Cause of Sudden Death

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Article History:
Date of Submission: Sunday September 12, 2025.
Date of Start of Review Process: Sunday September 12, 2025.
Date of Receipt of Reviewers Report: Saturday December 6, 2025.
Date of Revision: Saturday December 13, 2025.
Date of Acceptance: Saturday December 13, 2025.
Date of Publication: Wednesday December 24, 2025.
Digital Object Identifier **[DOI]: 10.5281/zenodo.17993577**

Available Online: Monday December 15, 2025

Website Archive: https://www.ijmj.net/archive/2025/2/IJMJ-2025-336.pdf

Citation: Sutay S, Bhushan A, Narang A, Saini PA, Sabri I. Autopsy findings in fatal Budd-Chiari syndrome: a rare cause of sudden death. Int J Med Justice. 2025 Dec 24;3(2):101-108. doi: 10.5281/zenodo.17993577

Indexing: INDEX COPERNICUS INTERNATIONAL, OpenAIRE, INTERNATIONAL Scientific Indexing, LetPub, ASCI

Academic Editor: Dr Richa Gupta

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Abstract: Budd-Chiari Syndrome (BCS) is a rare but life-threatening disorder characterized by the obstruction of hepatic venous outflow, which leads to hepatic congestion, portal hypertension, and potential progression to cirrhosis (Valla, 2009). Predisposing factors include hypercoagulable states, myeloproliferative disorders, and infections (Janssen, Garcia-Pagan, Elias, Mentha, & Valla, 2003). We present an autopsy case of a 24-year-old male, a known case of BCS complicated by Hepatitis C virus (HCV) infection and chronic liver disease, for whom liver transplantation was planned. The patient succumbed during medical management. The medico-legal autopsy revealed gynecomastia, micronodular liver cirrhosis, massive ascites, splenomegaly, esophageal varices, and bilateral pleural effusions. Histopathological examination confirmed cirrhotic architectural disruption. The immediate cause of death was determined to be hepatic failure secondary to BCS. This case underscores the aggressive natural history of BCS, particularly when compounded by concurrent HCV infection, and highlights the critical medico-legal role of autopsy in confirming the diagnosis and elucidating the pathophysiological sequence in rare, fatal presentations.

Keywords: Budd-Chiari Syndrome, Hepatic Vein Thrombosis, Liver Cirrhosis, Portal Hypertension, Sudden Death, Forensic Autopsy

Introduction: Budd-Chiari Syndrome (BCS) is an uncommon but potentially fatal condition defined by the obstruction of hepatic venous outflow. This obstruction can occur at the level of the hepatic veins or the supra- or infra-hepatic inferior vena cava (Kumar, Abbas, & Aster, 2021). The resulting hepatic venous congestion initiates a cascade of events, including increased sinusoidal pressure, hepatocyte necrosis, and, in chronic cases, fibrosis and cirrhosis, ultimately leading to portal hypertension (Menon, Shah, & Kamath, 2004). The global incidence is estimated at approximately 1 in 100,000 individuals (Michael, 2019). Clinical presentation can range from an acute abdomen with painful hepatomegaly and ascites to more insidious symptoms of chronic liver disease and portal hypertension.

The etiology is often multifactorial, involving primary thrombotic events (e.g., in myeloproliferative neoplasms or inherited thrombophilias) or secondary compression/invasion (e.g., by tumors or abscesses) (Darwish Murad et al., 2009). Due to its nonspecific presentation and high mortality if untreated, prompt diagnosis is essential. However, BCS

remains a rare and often overlooked cause of sudden or unexpected death in forensic practice. This report details the comprehensive autopsy findings in a young male with a known diagnosis of BCS, highlighting the terminal complications and affirming the value of postmortem examination in correlating clinical history with pathological sequelae.

Case History: A 24-year-old male was brought to the mortuary for a medico-legal autopsy following death during inpatient treatment. Clinical records indicated a history of Budd-Chiari Syndrome, chronic liver disease, and Hepatitis C positivity. A liver transplantation had been under consideration as definitive management prior to his demise.

Autopsy Findings

1. External Examination: The body was of average build. Rigor mortis was present universally. Notable findings included bilateral gynecomastia and pallor of the lower palpebral conjunctivae. Multiple, dark, dry, adherent cutaneous patches with black discoloration were present over the buttocks, suprapubic region, and upper thighs, consistent with prolonged recumbency. No evidence of traumatic injury or decomposition was observed.

2. Internal Examination:

- **Peritoneal Cavity:** Approximately 2 liters of straw-colored serous ascitic fluid was present.
- **Hepatobiliary System:** The liver was grossly shrunken (approx. 1000 g), with a pale, diffusely granular, and firm consistency, indicative of established cirrhosis. The portal vein was thickened and sclerotic.
- **Spleen:** Marked splenomegaly was noted, with the organ weighing 1000 g, and appearing pale and firm.
- **Gastrointestinal Tract:** The stomach contained approximately 400 ml of blood. The mucosa was pale, with congestion noted at the gastroesophageal junction, suggestive of varices.
- **Genitourinary System:** Both kidneys were pale (Right: 250 g; Left: 200 g).
- **Thoracic Cavity:** Bilateral pleural effusions (500 ml each, straw-colored) were present. The lungs were pale, and sectioning yielded reddish, frothy fluid consistent with pulmonary edema.
- **Central Nervous System:** The brain was congested, edematous, and weighed 1220 g.

3. Histopathological Examination:

- **Liver:** The architecture was disrupted by regenerative nodules bordered by fibrous septae. The septae exhibited bile duct proliferation, mononuclear inflammatory infiltrates, and focal hemorrhage, confirming micronodular cirrhosis.
- **Stomach:** Submucosal vascular congestion was prominent, with autolytic changes in the antrum.
- **Lungs:** Alveolar spaces contained eosinophilic, pink fluid (pulmonary edema) with mild septal vascular congestion.
- **Spleen:** Dilated sinusoids engorged with red blood cells and atrophy of the white pulp were observed, consistent with chronic passive congestion.

Cause of Death
Based on the clinical history and concordant autopsy findings, the cause of death was certified as: Hepatic failure secondary to Budd-Chiari Syndrome due to hepatic venous obstruction.

Discussion: This autopsy provides a definitive clinicopathological correlation in a fatal case of Budd-Chiari Syndrome. The findings—cirrhosis, massive ascites, splenomegaly, and pleural effusions—are classic sequelae of chronic hepatic venous outflow obstruction and subsequent portal hypertension (Valla, 2009). The presence of blood in the stomach, coupled with congested gastroesophageal mucosa, strongly suggests terminal hemorrhage from esophageal varices, a lethal complication of portal hypertension.

BCS pathogenesis involves an imbalance between hepatic arterial/portal inflow and restricted venous outflow, leading to increased intra-sinusoidal pressure (Menon et al., 2004). While our patient had a confirmed diagnosis, the autopsy could not retrospectively identify the specific underlying prothrombotic state, a common limitation in such cases. The co-existing Hepatitis C infection likely acted as a synergistic factor, accelerating fibrogenesis and worsening the prognosis, creating a challenging clinical scenario where transplant became the only viable option (Kozielewicz, Smukalska, & Dybowska, 2008).

From a forensic perspective, this case is instructive. BCS is a rare but documented cause of sudden and unexpected death, which may initially present as an unexplained demise (Shrestha et al., 1996). A meticulous autopsy, as performed here, is paramount to establish the diagnosis, especially in undiagnosed cases. It distinguishes natural disease progression from potential therapeutic misadventure or

neglect, which is crucial for medico-legal clarity. The findings of cirrhosis and splenomegaly, while nonspecific, when combined with a detailed clinical history, allow for a definitive conclusion. This underscores the indispensable role of forensic pathology in elucidating rare causes of death, contributing to mortality statistics, and informing clinical understanding of disease end-stages (Harrison et al., 2022; Okhuda, Kage, & Shrestha, 1998).

Conclusion

This report details the comprehensive postmortem findings in a young male with terminal Budd-Chiari Syndrome. It illustrates the severe complications of portal hypertension and hepatic failure that characterize the end-stage of this condition. The case reinforces that BCS, though rare, must be considered in the differential diagnosis of sudden death, particularly in individuals with signs of chronic liver disease. Forensic autopsy remains the gold standard for confirming the diagnosis, excluding other causes, and providing a definitive clinicopathological correlation, thereby fulfilling both medical and legal obligations.

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Figure 1. Gross appearance of liver

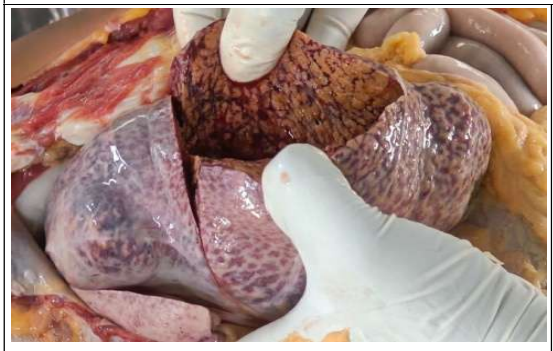

Figure 1A: Liver showing congested surface and altered morphology

Figure 1B: Liver after formalin fixation showing nodular surface consistent with cirrhosis

Gross examination of the liver showing congestion and surface irregularity. After formalin fixation, the liver demonstrates a nodular appearance suggestive of cirrhosis.

Figure 2. Gross appearance of gastroesophageal junction

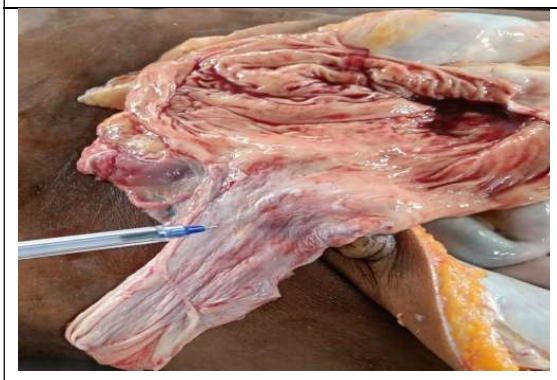

Figure 2A: Congested gastroesophageal junction

Figure 2B: Splenomegaly

Gross examination showing congestion at the gastroesophageal junction along with associated splenomegaly, indicating portal hypertension

Figure 3. Histopathology of liver (H&E stain)

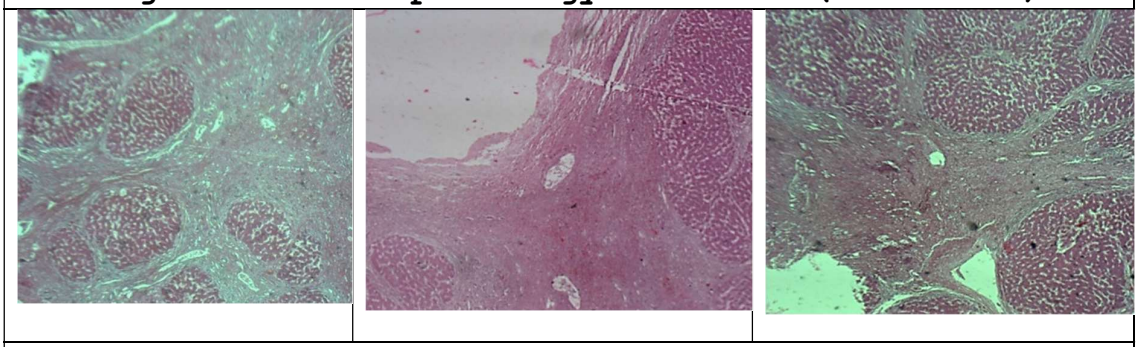

Figure 3: Liver histology showing disrupted hepatic architecture with regenerative nodules separated by fibrous septa

Histopathological examination of the liver reveals disrupted architecture with formation of regenerative nodules separated by fibrous septa. The septa show bile duct proliferation, mononuclear cell infiltration, and focal hemorrhage—findings consistent with liver cirrhosis.

Figure 4. Histopathology of gastroesophageal junction (H&E stain)

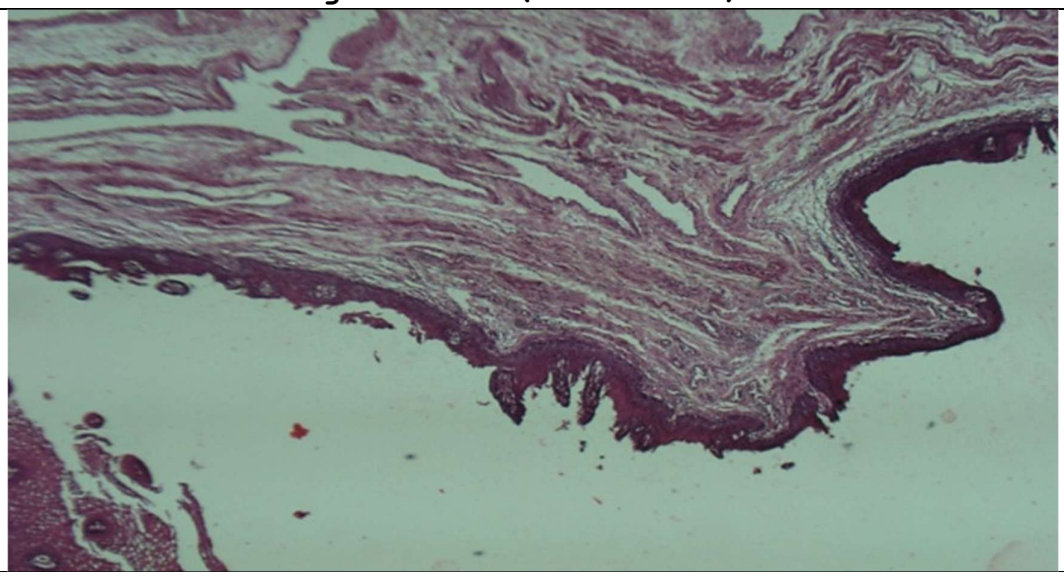

Figure 4: Esophageal submucosa showing dilated and tortuous blood vessels

Histopathological section of the gastroesophageal junction showing dilated and tortuous blood vessels in the esophageal submucosa, consistent with esophageal varices.