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Case Report

Fulminant Hepatitis Revealing Complicated Crohn's Disease of Budd Chiari Syndrome: About a Case

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Abstract

The Budd-chiari syndrome (BCS) is a rare vascular disease of multifactorial etiopathogenesis, represents a serious thromboembolic complication of Crohn's disease (CD), and may be the revealing mode. This condition results from obstruction of hepatic venous drainage from the hepatic venules to the terminal part of the inferior vena cava, and can be primary or secondary. Here, we report a case of a young woman with fulminant hepatitis whose etiological diagnosis revealed Crohn's disease with acute budd chiari syndrome and who was successfully treated with anticoagulants associated with biotherapy. The patient had a very good clinical evolution, after three months total recovery from the BCS. Very few cases have been published on this association.

Keywords: Anticoagulants; Budd chiari syndrome; Crohn's disease; Fulminant hepatitis

Introduction

The Budd-Chiari syndrome (BCS) is the set of clinical and biological signs resulting from the obstruction of the blood flow located between the hepatic venules and the junction between the inferior vena cava and the right atrium, recently developed by a group of European experts [1]. The causes of this obstruction can be multiple, mainly represented by myeloproliferative syndromes, hereditary thrombogenic disorders. Such as protein C, S, antithrombin III deficiency, inducing hypercoagulability of the blood, inflammatory diseases, recent oral contraception, mutation of genes coding for factor V coagulation factors Leiden. Acute BCS by simultaneous invasion of the three hepatic veins by massive acute thrombosis appears to be an exceptional phenomenon, encountered only in the event of an exacerbated pro-thrombotic state and produces a picture of fulminant hepatitis can be observed in 2-10% of cases [2]. CD is most often diagnosed in adulthood, sometimes associated with extra digestive manifestations including thromboembolic complications. The diagnosis is mainly based on imaging. The treatment of choice depends on the progressive stage of the thrombosis and the underlying cause. The prognosis is burdened with heavy mortality in the absence of specific treatment in time.

Case Report

A 17-year-old girl presented to the emergency room of our hospital, with impaired consciousness, subicteritis, mild rectal bleeding and fever. In this history notion of COVID infection one month ago treated with antibiotic therapy associated with anticoagulants (lovenox 0.6 IU twice a day) for ten days, and she reported no history of allergy, medications or genetic diseases. Upon physical examination, she was signed patient in poor general condition confused obnubilation hyperthermia , 39°C, blood pressure at 10/4 hepatomegaly, slight edema of the lower limbs keeping the pit, dullness of the flanks, no splenomegaly or collateral venous circulation. Proctologic examination two painless perianal fissures. However, her basic metabolic profile showed severely elevated liver enzymes (Figures 1a,b) and (Table 1).

	Test	Result (normal range)
CBC	Hemoglobin	8.3 (12.0-16,0) g/dL
	WBC count	22.103 (4.0-11.0) k/µ/L
	Platelets	90 (140-450) k/µL
Haemostasis indices	PT	21.6 (10.4-13.4)s
	INR	1.9
	aPTT	33.4 (22.2-32.9)s
LFT	Serum total protein	6.8 (6-8) g/dL
	Albumin	1.3 (3.5-5) g/dL
	SGOT	1496 (15-37) U/L
	SGPT	650,22 (14-63) U/L
	LDH	280 (81-234) U/L
	GGTP	67 (5-40) U/L
	ALP	503 (46-129) U/L
Bilirubin level		123(120)mg/dL
Viral serologies	HBsAg	Negative
Inflammatory proteins	AC anti-HCV	Negative
	HIV	Negative
	C-reactive protein	90,25(0-6) mg/L
	serum ferritin	601(148) mg/ml
	D dimers	79688,81(500) mg/ml

Table 1: Biochemical test results and viral serologies levels.

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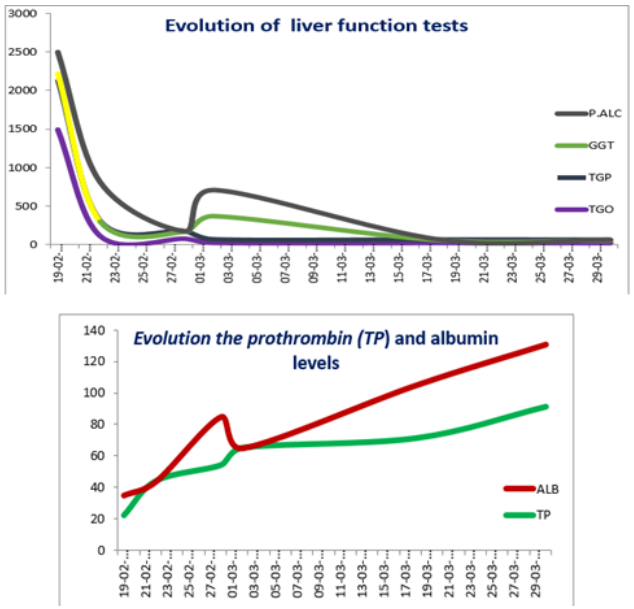

Figures 1a,b: a: Regression of cytolysis with normalization of AST and ALT. **b:** Correction albuminimia and PT correction.

The diagnosis of fulminant hepatitis was retained; An additional assessment was launched, to clarify the etiology of acute hepatitis by viral serologies A, B, C, CMV, HIV, EBV, HSV which came back negative as well as the autoimmunity assessment including anti mitochondrial anti smooth muscle antibodies, antibodies antiLKM Fibroscopy esogastroduodenale (FOGD) was normal. An emergency abdominal CT scan showed complete obstruction of the left suprahepatic veins, and partial obstruction of the right hepatic vein with hepatic perfusion disorder without hepatic dysmorphism.

Moderately abundant ascites, moderately abundant bilateral pleural effusion (Figures 2A-D) compatible with Diagnosis of acute BCS.

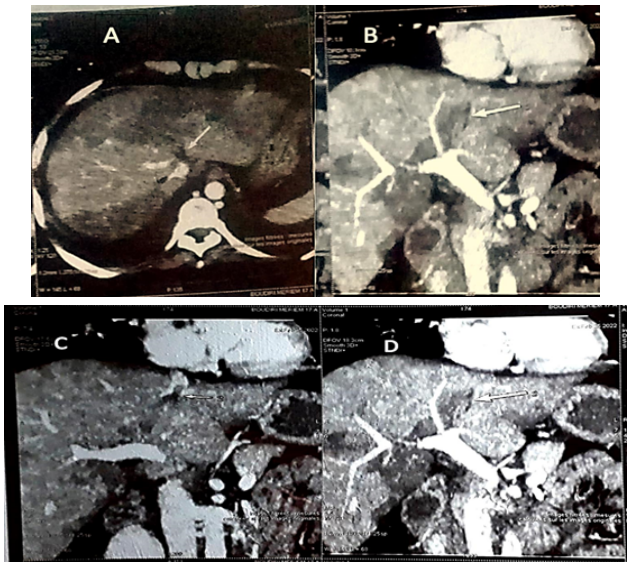

Figure 2: Abdominal CT angiography axial cut shows: Images A and B the arrow points to thrombosis of the left suprahepatic veins, with liver perfusion disorder without hepatic dysmorphism. **Abdominal CT angiography:** Images C and D the arrow points to thrombosis of the left-right suprahepatic veins with hepatic perfusion disorder.

The thrombophilia assessment to find the cause of the BCS carried out was negative, including protein C, protein S, anti-phospholipid antibodies, circulating lupus-type anticoagulant, anti-cardiolipin antibody, the dosage of antithrombin III, mutation of the factor II gene, homocysteinemia, factor V Leiden mutation, Laboratory studies performed to assess Wilson's disease, alpha-1 antitrypsin deficiency, non-alcoholic fatty liver disease and primary biliary cholangitis were also negative.

JAK2 mutation no made was not available in our hospital. Therefore, anticoagulation was instituted at curative doses of 0.6UI twice a day. His liver function and enzymes gradually normalized as well as a remarkable decrease in D-dimers and a clear symptomatic improvement.

For the patient's second digestive problem, an abdominal entero-CT scan performed (Figure 3) showed diffuse colonic intestinal thickening, reactive polyadenopathy with probable specific inflammatory ileitis. We completed the fiscal calprotectin assay, which was positive and high.

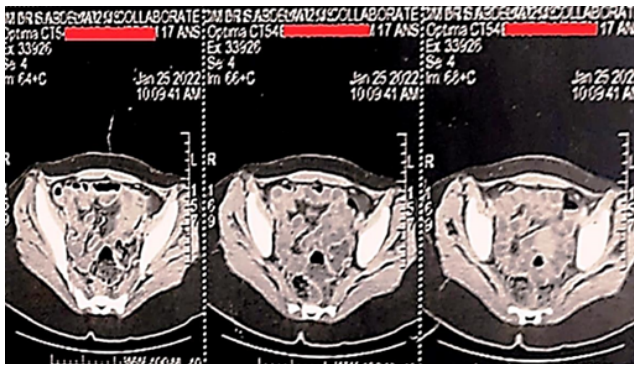

Figure 3: Abdominal scan diffuse colonic thickening with reactive adenopathies.

In addition, to confirm the diagnosis, an ilecoloscopy revealed ulcerations in terminal ileum and caecum with few areas of erythema in the rest of colon, biopsies (Figure 4) from the terminal ileum and caecum and the rest of colon showed (Figure 5) terminal ileitis, swollen colonic mucosa sometimes ulcerated. The chorion is inflammatory with polymorphous inflammatory infiltrate or predominate the lymphocytes encroaching on the muscular mucous membrane. The glands lieberkhuniennes are sometimes ectasic, elsewhere dedifferentiated. Histology findings, a diagnosis of CD was made.

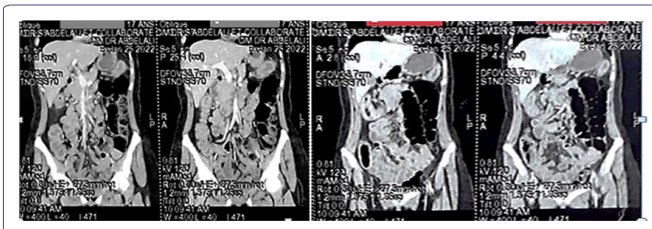

Figure 4: Enhanced CT scan of Abdomen. Coronal cut shows diffuse colonic thickening with reactive adenopathies.

After one month of treatment, a control CT angiography was performed, objectifying (Figure 6) persistence of thrombosis of the left suprahepatic veins and the left accessory vein which is partially regressive with discreet venous reperfusion in the periphery with a clear

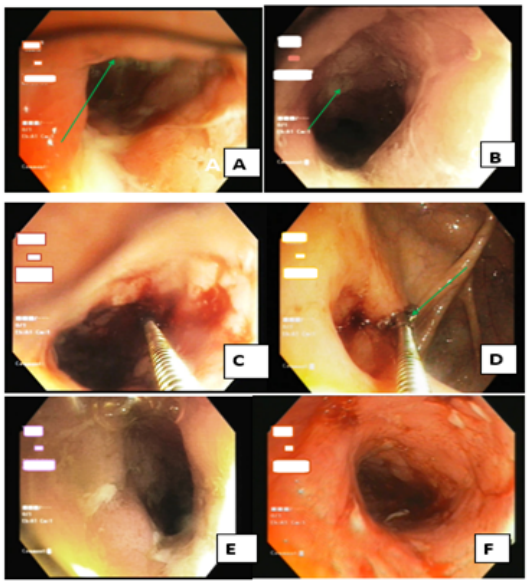

Figure 5: Ileocolonoscopy showing, **A:** Yawning ileocaecal valve with ulcerations. **B:** Ileitis with ulcerations of the terminal ileum. **C:** Caecum biopsy. **D:** Terminal ileum biopsy. **E:** Ulcerations ileum. **F:** Inflammatory and erosive colitis.

regression of 50% of hepatic perfusion disorders, partial thrombosis of the right hepatic vein, complete regression of pleural effusion and ascites, persistent colitis at the right colic level, transverse with mesenteric hyperaemia.

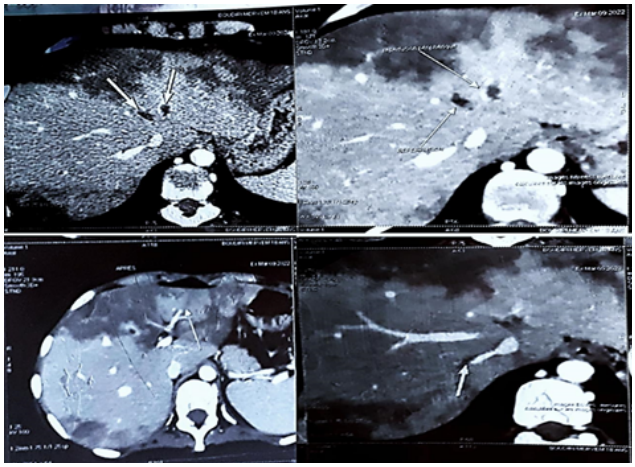

Figure 6: A: Abdominal CT angiography absence of opacification of the 2 supra-hepatic veins, associated with a congestive aspect of the hepatic parenchyma. **B:** CT angiography absence of liver dysmorphism.

The patient was put on biotherapy remicade 5mg/kg (S0,S2) and continuation of anticoagulation a per os relay, to prevent the recurrence of thrombosis, by antivitamins K (Scintron), was administered at doses adapted according to the Progressive TP and INR to reach the curative dose and allowed total repermeabilization of the supra-hepatic veins (Figure 7), and disappearance of ascites, pleural effusion. The two-biotherapy courses took place without incident. However, at the third treatment, she presented during the remicade infusion with major intolerance due to dyspnoea, edema, rapid arterial hypotension, cyanosis but rapidly resolving under injectable corticosteroids. Thus, a decision was made to change biotherapy and switch to Adalumumab inj.160mg in attack dose followed by one week after 80mg and 40mg every two-weeks. A biological control assessment was carried out one month later showing a normalization of the CRP and the VS negotiations of D. dimers. A control radiology by abdominal ultrasound performed 6 months later. There were no abnormal results. The patient had a good colonic evolution and did not have another recurrence of thrombosis.

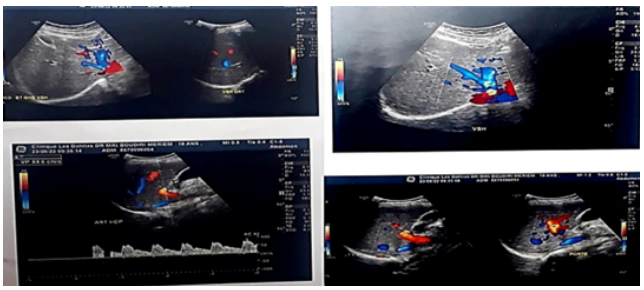

Figure 7: Abdominal Doppler repermeabilization of the hepatic veins.

Discussion

BCS occurs mainly in young women, can occur in three acute, subacute, or chronic modes, and results from an obstruction of the hepatic venous flow sitting between the right atrium and the terminal veinlets of the hepatic vein. Clinical and biological manifestations are variable and range from lack of symptoms of incidental discovery of fulminant liver failure [3,4]. This syndrome represents a very rare thromboembolic complication of CD. The diagnosis of CD usually precedes or occurs at the same time as the diagnosis of BCS. This syndrome is often observed during a flare-up of CD and correlated to the extent and location of the disease [5,6]. In our observation, this is a female case, the CD was diagnosed at the same time as the BDS, the development mode was acute, and there was no portal hypertension syndrome on the physical exam, endoscopic exam (FOGD) and abdominal angioscanner.

BCS takes the acute aspect when there is likely a simultaneous obstruction of the three-suprahepatic veins in the absence of underlying liver disease, it is then characterized by an absence of hepatic dysmorphia, Very frequent acute liver failure that can be severe, sometimes fulminant or sub fulminant associated with hepatic encephalopathy. It is a rare situation representing less than 1% of severe acute hepatitis that engages the vital prognosis. The presence of hepatomegaly, unusual during severe acute hepatitis, as well as an aspartate aminotransferase/alanine aminotransferase ratio greater than 1, could lead to BCS [7]. In our patient, the clinical examination showed ascites, jaundice and hepatic encephalopathy, hepatomegaly, and transaminases greater than five times the normal associated liver failure with a PT less than 10%. In acute forms hepatocellular insufficiency may be severe, rarely fulminant or sub fulminant with elevated transaminases, greater than 5 times normal, prothrombin level 50%, protein-rich ascites and renal insufficiency there is no relationship between the progressive form (fulminant, acute, subacute or chronic) and the age of the venous or hepatic lesions [8]. Less than 10% of acute forms correspond to recent lesions. The most common cause of BCS is myeloproliferative syndrome, but may also be secondary to coagulation disorders: antiphospholipid syndrome, protein deficits C, S, antithrombin deficiency, Leiden factor V mutation and hyperhomocysteinemia. Rarer causes such as paroxysmal nocturnal hemoglobinuria and genetic abnormalities of the factor II gene should be investigated. Finally, in 10 to 30% of cases, no favoring cause is retained.

To diagnose a BCS, several imaging tools are available including color Doppler Echo, Injected Abdominal Scanner and Magnetic Resonance Imaging (MRI). The echo-Doppler has a sensitivity of about 90% its performance depends on the morphotype and the experience of the operator. In case of suspicion of acute thrombosis, the injected scanner must be performed in first intention [9]. It makes it possible to make the diagnosis of thrombosis, to appreciate the extension of thrombosis, especially the 3 hepatic sus veins, detect signs of intestinal ischemia, and look for a local cause. MRI offers similar advantages to the scanner and is a possible alternative, especially in renal failure.

Our attitude to confirm the diagnosis was the realization of abdominal angiography, which specified the location of the thrombosis, its extension and local complications. Before any BCS, a complete etiological assessment must be carried out in search of an innate/acquired pro-coagulant state, a myeloproliferative disease or a systemic or inflammatory pro-coagulant pathology. In our patient, etiological research revealed crohn's disease, the diagnosis of complicated crohn's disease of acute budd chiari syndrome was established. This association is exceptional. Data from the literature are lacking at present two cases have been reported [10,11].

Crohn's disease (CD) is an inflammatory intestinal disease (IBD). It causes a systemic inflammatory condition that can affect the joints, eyes, liver, skin and hematological system. In the acute phase, elevated biological parameters such as the sedimentation rate of white blood cells and reactive C protein can also be observed. The disease can induce a prothrombotic state resulting in occlusion of arteries and veins. people with CD have a triple risk of developing venous thromboembolic disease four times higher. Thrombosis has been reported in the liver veins, portal veins and inferior vena cava (IVC). One Hepatic venous obstruction is the underlying cause of BCS.

The pathogenesis of this entity remains unclear, the causal condition being CD, which is a prothrombotic condition [12,13]. The mechanism leading to increased thrombotic risk in CD is complex and unclear. The existence of a hypercoagulability state related to the interaction between the mediating cytokines of chronic intestinal inflammation especially at the time of an acute outbreak and the coagulation cascade is the main hypothesis. However, 30 to 40% of thromboses occur in the quiescent phase of the disease [14,15]. Acquired risk factors such as smoking, obesity, diabetes, cancer dyslipidemia, high blood pressure, recent surgery, oral contraception, prolonged immobilization, corticosteroids have a significant thrombogenic contribution. Currently, the incidence of CD is significantly increasing around the world, including in Northern countries with a predilection for young adults [16] as in our case. The diagnosis of CD is usually made using endoscopic results with histological evidence or imaging studies in a patient with suggestive clinical symptoms. Ileo-colonoscopy is usually the first examination of choice with basic blood tests. Different types of blood and fecal markers, including antisaccharomyces, p-ANCA, CRP, fecal calprotectin, are used to help support the diagnosis [17]. Ileo-colonoscopy in our patient had shown ulcerations at the last ideal loop with an inflammatory colic mucosa strewn with some superficial ulcerations. The biopsy results were characteristic of a specific inflammatory CD. The patient was in moderate to severe flare-up with acute vascular complication. The attack treatment must be started immediately, given the critical presentation of the disease, it was decided to start biotherapy with anti-TNF alpha. Infliximab and adalimumab show good clinical response, a better degree of remission and healing of the mucosa than placebo, with no increased side effects [18]. In our presentation, given the facts, the goal of a remission was achieved. Regarding the anticoagulant treatment of venous thrombosis during CD is not consensual; which is certain, must be started early at curative doses to prevent the extension of thrombosis with the occurrence of another thromboembolic event and severe congestion of the liver with irreversible hepatocellular insufficiency leading to necrosis and fibrosis and finally cirrhosis. Anticoagulation is based on heparin therapy with low molecular weight heparin (LMWH) (Lovenox®) subcutaneous injection and relai by vitamin K (Sintrom) antagonists; INR should be maintained between 2 and 3. Recent recommendations encourage maintenance of LMWH or antivitamin K for 3 to 6 months, with a minimum duration of 3 months [19-22]. The duration of treatment depends on the etiology and can be prolonged for life in some cases. In our case, the patient was on lovenox for a month and then relayed by Sintrom. Heparin-prophylaxis with PMHB may also be considered for severe flare-up or risk factors such as bed rest or prolonged hospitalization, thrombocytosis greater than 750,000 elements/mm3 or personal or family history of thromboembolism. The ultrasound-Doppler, liver vein control had not identified an obstacle on the VSH. Our data as well as those of the literature show that the anticoagulant treatment is generally effective, allowing a repermeabilization of the hepatic overlying veins especially in case of acute thrombosis. People with CD after a first episode of venous thromboembolic events are at increased risk of developing another episode after stopping anticoagulant therapy. Our patient was kept on Sintrom for 9 months and no other episodes of thrombosis occurred. It seems that young age and female sex most likely related to hormonal status are factors of good prognosis in this association, while the extent of the lesions and their importance as well as the severity of the outbreak of crohn's disease are not necessarily correlated with the occurrence of BCS.

Conclusion

Acute budd chiari syndrome is an exceptional complication of Crohn's disease in young adults and may represent the revealing mode. It is imperative to evoke the diagnosis of SBC during any acute liver disease. Urgent evaluation and early contributes to the good therapeutic management of the budd chiari syndrome even before the search for the causal affection and conditions the vital prognosis. Secondly, it is very important to identify the etiology to undertake a safe and certain therapeutic strategy for the disease responsible for SDB in order to avoid recurrences of thrombosis or new complications that can be fatal.

Abbreviations

BCS: Budd-Chiari syndrome; CD: Crohn's disease; CT: computed tomography; COVID: Coronavirus Disease; CMV: Cytomegalovirus; AST: Aspartate aminotransferases; ALT: Alanine aminotransferases; PT: Prothrombin time; CRP: C- reactif protein; HIV: Human immunodeficiency virus; EBV: Epstein-Barr virus; HSV: Herpes simplex virus; LKM: Anti-liver and kidney microsome; JAK2: Janus kinase2; FOGD: Fibroscopy esogastroduodenale; MRI: Magnetic Resonance Imaging.

Declarations

Ethics statements

Ethical approval and consent to participate in this study was done in accordance with the Declaration of Helsinki and approved by the

Research and Ethics Committee of Algeria; the reference number is not applicable. This study does not contain personal information or information that could lead to the identification of the patient.

Consent to publication

We obtained informed written consent from the patient and her parents for participation in the study.

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None.

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All authors certify that they meet the current ICMJE criteria for authorship.

Declaration of Competing Interests

The authors declare that they have no known competing financial interests or personal relationships that could influence the work presented in this document.

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Sample Credit author statement

Amal Belkhatir: Project administration, Funding acquisition, Supervision, visualisation, writing-review editing.

Mehdi Hassain: Data curation, Resources, Formal analysis.

Habib Kies: Investigation, Validation, Writing-Original Draft.

The author confirms sole responsibility for the following: Study conception and design, data collection, analysis and interpretation of results, and manuscript preparation.

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