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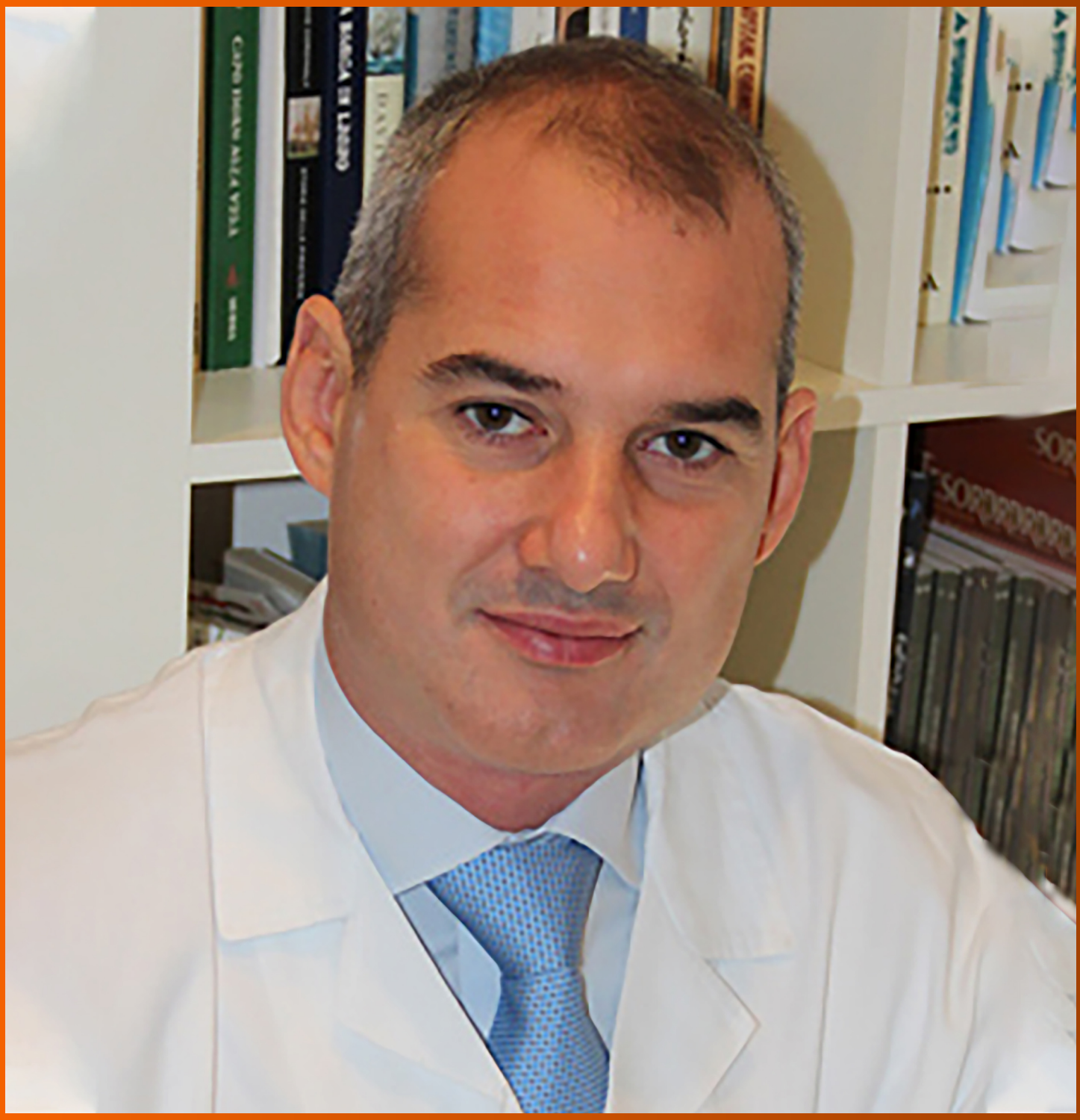

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MINIREVIEWS

Acute acalculous cholecystitis due to infectious causes

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Abstract

Acute acalculous cholecystitis (AAC) is an inflammation of the gallbladder not associated with the presence of gallstones. It usually occurs in critically ill patients but it has also been implicated as a cause of cholecystitis in previously healthy individuals. In this subgroup of patients, infectious causes comprise the primary etiology. We, herein, discuss the pathophysiological mechanisms involved in AAC, focusing on the infectious causes. AAC associated with critical medical conditions is caused by bile stasis and gallbladder ischemia. Several mechanisms are reported to be involved in AAC in patients without underlying critical illness including direct invasion of the gallbladder epithelial cells, gallbladder vasculitis, obstruction of the biliary tree, and sequestration. We emphasize that multiple pathogenic mechanisms may concurrently contribute to the development of AAC in varying degrees. Awareness of the implicated pathogens is essential since it will allow a more focused examination of the histopathological specimens. In conclusion, additional research and a high degree of clinical suspicion are needed to clarify the complex spectrum of mechanisms that are involved in the pathogenesis of AAC.

Key Words: Cholecystitis; Infectious causes; Vasculitis; Sequestration; Epstein-Barr virus

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Core Tip: The most important mechanisms involved in acute acalculous cholecystitis in patients without underlying critical illness are direct invasion of the gallbladder epithelial cells, gallbladder vasculitis, obstruction of the biliary tree, and sequestration. Awareness of the implicated pathogens is essential since it will allow a more focused examination of the histopathological specimens.

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INTRODUCTION

Acute acalculous cholecystitis (AAC) is an inflammation of the gallbladder not associated with the presence of gallstones. AAC accounts for approximately 5%-10% of all cases of acute cholecystitis in adults and 50%-70% of cases in the pediatric population[1]. AAC usually occurs in critically ill patients and is most frequently associated with trauma, surgery, shock, burns, sepsis, total parenteral nutrition (TPN) and mechanical ventilation[2]. Moreover, it is related to high mortality rates (30% in most studies) not only because it is an epiphenomenon of the critical illness, but also because it can progress to gangrene, perforation and empyema more frequently than calculous cholecystitis[2]. AAC has also been implicated as a cause of cholecystitis in previously healthy individuals. In this subgroup of patients, infectious causes comprise the primary etiology[1]. Since its first description by Duncan in 1884, numerous pathogens have been identified as causative agents. We, herein, discuss the pathophysiological procedures involved in AAC, focusing on the infectious causes.

PATHOGENESIS

AAC involves several pathological processes that seem to concurrently play a role in the manifestation of the disease. The pathogenesis of AAC, when it directly originates from infectious agents, falls into two main categories: (1) AAC associated with critical medical conditions; and (2) AAC in patients without underlying critical illness. We focus on the second group of patients and categorize the various pathophysiologic mechanisms identified in several reports in the literature concerning a wide range of pathogens. We briefly report the principal mechanisms that are implicated in the first category, as they have been extensively analyzed in previous articles.

ACUTE ACALCULOUS CHOLECYSTITIS ASSOCIATED WITH CRITICAL MEDICAL CONDITIONS

Bile stasis

Bile stasis has been identified to contribute to the pathogenesis of the disease. Prolonged fasting and TPN have both been linked to the formation of biliary sludge due to the absence of cholecystokinin stimulation of the gallbladder[3]. It is noteworthy to mention that according to a 10-year retrospective review, at least 4 wk of TPN were required for bile sludge formation in 50% of patients. By 6 wk, all patients had developed bile sludge[3]. In the pediatric population, congenital malformations (*e.g.*, multiseptated gallbladder, choledochal cyst) interrupt normal bile flow[1]. Moreover, another factor that is related to the concentration of bile is volume depletion, which is commonly seen in critically ill patients[3]. Bile inspissation elevates intraluminal pressure and according to the principle of Laplace (tension = pressure × radius), the wall tension of the organ is increased. Therefore, arterial, lymphatic and venous flow to the gallbladder wall is impaired[4]. Opioid analgesics may further increase intraluminal pressure, because of the spasm of the sphincter of Oddi[3]. Mechanical ventilation with positive end-expiratory pressure (PEEP) is also implicated

in the induction of bile stasis. PEEP of 7 to 10 cm H_2O increases hepatic venous pressure, which leads to decreased portal perfusion[5]. In addition, bile stasis changes the chemical composition of the bile, directly causing injury to the gallbladder mucosa. For instance, lysophosphatidylcholine, which is found in the bile of patients with acute cholecystitis, has been shown to cause extensive mucosal damage in a dose-dependent manner[6].

Gallbladder ischemia

A second critical mechanism is local ischemia. The gallbladder is quite susceptible to ischemic conditions since the main oxygenated blood supply is from the cystic artery, which is a terminal artery. Various diverse underlying diseases, like trauma, cardiovascular surgeries, septic shock, hypovolemic shock and burns that are identified as etiologic factors of AAC, share tissue hypoxia as the dominant pathophysiologic mechanism. The importance of ischemia in the development of the disease has been proven by Hakala *et al*[7]. They discovered that microangiographic findings of the gallbladder in acute calculous cholecystitis (ACC) differ from those in AAC. Briefly, microangiography revealed a poor and irregular capillary network in AAC, whereas in ACC a dense vessel network and dilated arterioles were demonstrated.

ACALCULOUS CHOLECYSTITIS IN PATIENTS WITHOUT UNDERLYING CRITICAL ILLNESS

Through an extensive review of cases of AAC in patients without underlying critical illness (Table 1), the main mechanisms that were found to be involved are: Direct invasion of the gallbladder epithelial cells, gallbladder vasculitis, and obstruction of the biliary tree. We will also briefly mention one proposed mechanism, that of sequestration. Epstein-Barr virus (EBV) will be discussed separately since it is the most common causative infectious agent of AAC. Lastly, we will present the available data in the medical literature regarding AAC due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. Some of the pathogens mentioned in Table 1 will not be further discussed, as no pathophysiological mechanism was identified.

Direct invasion

A large number of pathogens have been shown to directly invade the gallbladder epithelium. Herein, we will include the cases in which the pathogen was identified by molecular techniques and/or histopathological examination of the gallbladder wall. Evidently, this was feasible only for cases where a cholecystectomy was performed. As on multiple occasions a more conservative approach was chosen, we cannot rule out the possibility that other pathogens may share the same pathophysiologic mechanism. Menendez *et al*[8] demonstrated that *Salmonella enterica* serovar Typhi has a unique tropism for gallbladder epithelial cells. By using microscopy, they were able to find a high concentration of bacteria both in the lumen and in the tissue. The bacterium was rarely seen within the lamina propria and was confined in the epithelial cells. Additionally, electron micrographs showed intracellular *Salmonella* undergoing cell division[8]. The routes by which *Salmonella* was able to access the gallbladder were through the bloodstream, the lymphatic system and by directly ascending from the gastrointestinal tract. Histopathology of the infected gallbladders revealed destruction of the epithelium and massive infiltration of neutrophils along with increased levels of proinflammatory cytokines. It should be emphasized that invasion-deficient bacteria were unable to produce the aforementioned changes, even though they were present in the gallbladder lumen.

Direct invasion was also proved by Mourani *et al*[9]. They were able to detect hepatitis A virus (HAV) antigen in most gallbladder epithelial cells using immunohistochemical staining. A cell-mediated immunologic response was proposed based on the high number of intraepithelial lymphocytes.

A heterogeneous group of infectious causes leads to acute or chronic acalculous cholecystitis in immunocompromised patients: Cytomegalovirus (CMV)[10,11], *Cryptosporidium* spp.[10], *Isospora belli*[12,13], *Sarcocystis* spp.[14], *Cyclospora cayetanensis*[15], *Enterocytozoon bieneusi*[16], *Histoplasma capsulatum*[17], *Mycobacterium tuberculosis*[18]. In a retrospective study by French *et al*[19], it was established that AIDS-related biliary tract disease was most commonly related to CMV and *Cryptosporidium* spp. The most typical histopathological finding was ulceration and epithelial necrosis. CMV inclusion bodies were found in stromal cells in the base of the ulcers, in the mucosa adjacent to

Table 1 Reported cases of acute acalculous cholecystitis due to infectious causes

Ref.	Age (yr)	Sex	Immunocompromised	Pathogen	CCY	Laboratory investigation							Infectious ACC
						Stool culture	Blood culture	Bile culture	Serology	Histopathology[1]	Tissue PCR	IHC	
Garrido-Benedicto *et al*[40], 1994	15	M	-	*Salmonella* spp.	-	+	NP	NP	NP	NP	NP	NP	Possible
Ruiz-Rebollo *et al*[41], 2008	27	M	-	*Salmonella enterica*	-	+	NP	NP	NP	NP	NP	NP	Possible
Khan *et al*[42], 2009	31	M	-	*Salmonella enterica*	-	NP	+	NP	NP	NP	NP	NP	Possible
Rajan *et al*[43], 2014	23	F	-	*Salmonella enterica*	+	NP	+	NP	NP	+	NP	NP	Probable
Lianos *et al*[44], 2019	32	M	-	*Salmonella enterica*	+	+	NP	+	NP	NP	NP	NP	Possible
Mourani *et al*[9], 1994	68	M	-	HAV	+	NP	NP	NP	+	+	NP	+	Proven
Suresh *et al*[45], 2009	2,5	F	-	HAV	-	NP	NP	NP	+	NP	NP	NP	Possible
Prashanth *et al*[46], 2012	12	F	-	HAV	-	NP	NP	NP	+	NP	NP	NP	Possible
Kaya *et al*[47], 2013	31	F	-	HAV	-	NP	NP	NP	+	NP	NP	NP	Possible
Hinnant *et al*[10], 1989	32	M	HIV	CMV, *Cryptosporidium* spp.	+	NP	NP	NP	NP	+	NP	NP	Proven
Hinnant *et al*[10], 1989	44	M	HIV	CMV, *Cryptosporidium* spp.	+	+	NP	NP	NP	+	NP	NP	Proven
Riediger *et al*[11], 2013	60	F	Renal transplantation	CMV	+	NP	NP	NP	+	+	NP	+	Proven
Agholi *et al*[12], 2016	25	F	Corticosteroid therapy	*Cystoisospora belli*	+	+	NP	NP	NP	+	NP	NP	Proven
Agholi *et al*[12], 2016	35	M	HIV	*Cystoisospora belli*	+	+	NP	NP	NP	+	+	NP	Proven
Agholi *et al*[14], 2014	28	F	HIV	*Sarcocystis* spp.	+	+	NP	NP	NP	+	+	NP	Proven
Zar *et al*[15], 2001	35	M	HIV	*Cyclospora cayetanensis*	+	+	NP	NP	NP	+	NP	+	Proven
Knapp *et al*[16], 1996	37	M	HIV	*Enterocytozoon bieneusi*	+	+	NP	+	NP	+	NP	NP	Proven

Shinha *et al*[17], 2015	38	M	HIV	*Histoplasma capsulatum*	+	NP	NP	+	NP	+	NP	NP	Proven
Chen *et al*[18], 2008	36	M	HIV	*Mycobacterium tuberculosis*	+	NP	NP	NP	NP	+	NP	NP	Proven
Takeshita *et al*[20], 2006	64	M	-	HBV	+	NP	NP	NP	+	+	NP	NP	Probable
Unal *et al*[48], 2009	49	F	-	HBV	-	NP	NP	+	NP	NP	NP	NP	Possible
Meier *et al*[22], 2005	NA	NA	-	HCV	+	NP	NP	NP	NP	+	NP	NP	Probable
Wright *et al*[49], 2019	33	M	-	HCV	+	NP	NP	NP	+	+	NP	NP	Probable
Ono *et al*[23], 2018	54	M	Lymphocytic leukemia	Zika virus	+	NP	NP	NP	NP	+	+	NP	Proven[2]
Guarner *et al*[24], 2001	29	F	-	*Leptospira*	+	NP	NP	NP	+	+	NP	+	Proven
Guarner *et al*[24], 2001	60	M	-	*Leptospira*	+	NP	NP	NP	+	+	NP	+	Proven
Walker *et al*[25], 1985	71	F	-	*Rickettsia rickettsii*	+	NP	NP	NP	NP	+	NP	+	Proven
Spernovasilis *et al*[50], 2017	54	M	-	*Rickettsia typhi*	-	NP	NP	NP	+	NP	NP	NP	Possible
Araki *et al*[28], 2017	70	M	Immunosuppressive agents	*Giardia lamblia*	-	+	NP	NP	NP	NP	NP	NP	Proven[3]
Colle *et al*[29], 2002	25	F	-	*Echinococcus granulosus*	+	NP	NP	NP	+	+	NP	NP	Probable
Saha *et al*[51], 2005	7	F	-	*Plasmodium falciparum*	-	NP	NP	NP	+	NP	NP	NP	Possible[4]
Curley *et al*[33], 2011	26	M	-	*Plasmodium vivax*	-	NP	NP	NP	NP	NP	NP	NP	Possible[4]
Harris *et al*[32], 2013	59	M	-	*Plasmodium malariae*	-	NP	NP	NP	NP	NP	NP	NP	Possible[4]
Dinulos *et al*[35], 1994	4	M	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Dinulos *et al*[35], 1994	16	M	-	EBV	+	NP	NP	NP	+	+	NP	-	Probable
Lagona *et al*[52], 2007	4	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Iaria *et al*[30], 2007	18	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible

Prassouli *et al*[53], 2007	13	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Attilakos *et al*[54], 2008	5	M	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Gagneux-Brunon *et al*[55], 2014	18	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Gagneux-Brunon *et al*[55], 2014	20	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Agergaard *et al*[34], 2014	34	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Alkhoury *et al*[56], 2014	15	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Yesilbag *et al*[57], 2017	30	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Ntelis *et al*[36], 2019	15	F	-	EBV	-	NP	NP	NP	+	NP	NP	NP	Possible
Alhassan *et al*[39], 2020	40	F	-	SARS-CoV-2	-	NP	-	NP	NP	NP	NP	NP	Possible
Andriopoulos *et al*[58], 2002	72	M	-	*Brucella melitensis*	+	NP	+	+	+	+	NP	NP	Probable
Hariz *et al*[59], 2019	62	F	-	*Brucella melitensis*	-	NP	NP	NP	+	NP	NP	NP	Possible
Figtree *et al*[60], 2010	38	M	-	*Coxiella burnetii*	+	NP	NP	NP	+	+	+	NP	Proven
Rolain *et al*[61], 2013	71	M	-	*Coxiella burnetii*	+	NP	NP	NP	+	+	NP	-	Probable
Wu *et al*[62], 2003	NA	NA	-	Dengue fever virus	+	NP	NP	NP	+	+	NP	NP	Probable
Marasinghe *et al*[63], 2010	29	F	-	Dengue fever virus	-	NP	NP	NP	+	NP	NP	NP	Possible
Hayakawa *et al*[64], 2011	72	F	-	*Orientia tsutsugamushi*	-	NP	NP	NP	+	NP	NP	NP	Possible
Mahapatra *et al*[65], 2019	57	F	-	*Ehrlichia chaffeensis*	+	NP	NP	NP	+	+	+	-	Proven
Kurtovic *et al*[66], 2004	74	F	CVID	VZV	-	NP	NP	NP	+	NP	NP	NP	Possible
Udayakumar *et al*[67], 2009	35	F	-	*Campylobacter jejuni*	+	+	NP	NP	NP	NP	NP	NP	Possible
West *et al*[68], 1998	55	F	-	*Vibrio cholerae*	+	NP	+	+	NP	+	NP	NP	Probable

Szvalb *et al*[69], 2019	39	F	Acute myelocytic leukemia	*Fusarium* spp.	-	NP	+	+	NP	NP	NP	NP	Possible

[1]Histopathology is considered positive if inflammation is present or if the pathogen is identified.
[2]Zika virus was also identified using electron microscopy.
[3]*Giardia* was identified using bile duct brush cytology and biopsy of the hilar bile duct stricture.
[4]The parasites were detected through peripheral smear.
Proven: Cases in which cholecystectomy was performed, inflammation of the gallbladder was demonstrated, and the causative pathogen of acute acalculous cholecystitis (AAC) was identified using histopathology, polymerase chain reaction (PCR) on tissue specimen or immunohistochemistry (IHC); Probable: Cases in which the causative pathogen of AAC was identified using stool/blood/bile culture or serological methods, cholecystectomy was undertaken and inflammation of the gallbladder was demonstrated, but histopathology, PCR on tissue specimen or IHC were not performed or were negative for pathogens; Possible: Cases in which the causative pathogen of AAC was identified using stool/blood/bile culture or serological methods but cholecystectomy was not performed. CCY: Cholecystectomy; NP: Not performed; NA: Not available; CVID: Common variable immune deficiency; HAV: Hepatitis A virus; CMV: Cytomegalovirus; HIV: Human immunodeficiency virus; HBV: Hepatitis B virus; HCV: Hepatitis C virus; EBV: Epstein-Barr virus; VZV: Varicella zoster virus.

the ulcers and inside the endothelial cells, whereas *Cryptosporidium* spp. was located on the surface of nonulcerated mucosa[10]. It is thought that the presence of ulcers and the profound mucosal pathology that is observed in these patients is associated with secondary bacterial infection[19]. Agholi *et al*[12] and Benator *et al*[13] both reported cases of chronic acalculous cholecystitis associated with *Isospora belli*. Since these patients had positive stool samples for *Isospora belli,* the suspected mechanism is that the parasites migrated retrogradely to the gallbladder from a periampullary focus. All developmental stages of the parasite were seen in the cytoplasm of infected epithelial cells, yet none were identified in the lamina propria. Similarly, *Sarcocystis* spp.[14] and *Cyclospora cayetanensis*[15] also demonstrated the ability to replicate within the gallbladder epithelial cells.

Vasculitis

Vasculitis is a well-established mechanism through which gallbladder injury can occur. When the gallbladder vasculature is involved, local ischemia leads to cell death and gallbladder necrosis. Necrotizing vasculitis is observed in biopsy specimens of the gallbladder in chronic HBV infection. In a case report by Takeshita *et al*[20], a patient with hepatitis B-related polyarteritis nodosa (PAN) was presented. PAN represents one of the most typical extrahepatic complications of HBV. Histopathological examination of the gallbladder and liver revealed necrotizing vasculitis with fibrinoid necrosis in medium-sized vessels. Immunohistochemical analysis showed IgG and complement deposition in the inflamed vasculitis lesions. According to the aforementioned changes and the absence of circulating immune complexes, an antibody dependent cell-mediated cytotoxicity or complement-dependent cytotoxicity are regarded as possible mechanisms[20]. It is noteworthy that previous studies had identified immune complexes as one of the key aspects of hepatitis B-related PAN[21]. HCV-induced AAC has rarely been documented. In a case report by Meier *et al*[22], histologic evaluation was consistent with cryoglobulinemic vasculitis. Nonetheless, the pathogenesis of AAC in chronic HCV infection still remains obscure.

Similar to the other viruses mentioned above, Zika virus was found to cause microangiopathy of the gallbladder vessels in an immunocompromised patient[23]. Ono *et al*[23] used polymerase chain reaction to detect the virus in the gallbladder tissue and in the bile. Furthermore, through electron microscopy, they identified flavivirus-like particles both in intracellular and extracellular compartments of the vessel.

In leptospirosis, AAC has also been linked to the direct localization of these bacteria in the gallbladder that is accompanied by varying degrees of vasculitis[24]. Immunohistochemistry demonstrated areas of antigen staining in the vessel walls and occasionally in the submucosa. In one case, rare intact bacteria were found[24]. *Leptospira* infection seems to cause endothelial damage and leakage of vascular fluid leading to gallbladder wall edema. Interestingly, the mucosal epithelium was unremarkable.

Lastly, *Rickettsia rickettsii* was also found to induce vascular injury and nonocclusive thrombosis leading to AAC[25].

Obstruction

Obstruction of the biliary tract either by intrinsic factors or by extrinsic compression may lead to AAC. This mechanism is most frequently encountered in *Ascaris lumbricoides* infection. Hepatobiliary ascariasis is rare and is the result of high intestinal parasite load in the host[26]. The worms ascend through the ampulla of Vater, reach the bile duct (choledochal ascariasis) and block the cystic duct orifice. Occasionally, they can invade the gallbladder (gallbladder ascariasis)[27]. The anatomical variants of the biliary tree play a determining role in the migration of the worms inside the gallbladder[26]. In both cases, the intraluminal pressure and the gallbladder wall tension are increased causing ischemia.

Araki *et al*[28], presented a very unique case of AAC attributed to *Giardia lamblia*. During the patient's workup, magnetic resonance cholangiopancreatography revealed a stricture of the hilar bile duct and cystic duct obstruction. A transpapillary bile duct brush cytology and biopsy of the stricture were performed, demonstrating active *G. lamblia* trophozoites. Subsequently, they proved that the parasite accessed the gallbladder through the ampulla of Vater by performing a duodenal biopsy, which was positive.

In addition, it is speculated that extrinsic cystic duct obstruction can arise from the formation of hydatid cysts during the course of echinococcal disease[29]. Furthermore, portal lymphadenitis from infectious causes, like EBV, has been proposed as a possible pathophysiological mechanism[5,30].

Sequestration

The phenomenon of sequestration has been proposed as a mechanism to explain the pathogenesis of AAC in the context of malaria infections[31]. During *Plasmodium falciparum* infection, protrusions (knobs) appear on the surface of infected erythrocytes, which cause the infected cells to adhere to each other and to the vessel walls. Once again, this leads to microcirculatory obstruction and ischemia. However, *Plasmodium malariae*[32] and *Plasmodium vivax*[33], which have also been documented to cause AAC, have not been shown to form knobs on the surface of erythrocytes.

EBV

EBV is documented as the most prevalent infectious cause of AAC in many reviews. However, the exact pathophysiological mechanism remains obscure, given that the disease is usually self-limited and a conservative treatment is followed. Direct invasion of the gallbladder epithelial cells is a proposed mechanism, as EBV infects oral epithelial cells[34]. In addition, as we mentioned above, other hepatotropic viruses, and specifically HAV, have been detected inside the gallbladder epithelial cells[9]. It should be mentioned that even when a cholecystectomy was performed, *in situ* hybridization of the tissue did not reveal the virus[35]. Moreover, Ntelis *et al*[36] speculated that vasculitis is the major underlying mechanism, but further investigation is required to support this hypothesis. Finally, compression of the cystic duct by an enlarged celiac lymph node could explain the development of AAC[30].

SARS-COV-2

AAC has been reported in patients withs SARS-CoV-2, and it is usually associated with critical illness, mechanical ventilation and prolonged TPN[37,38]. However, it has

been described a case of a patient with mild coronavirus disease 2019 (COVID-19) who, 14 days later, developed symptoms of AAC[39]. Blood and urine cultures were performed in order to identify other possible etiological pathogens but were negative. Thus, the authors argued that SARS-CoV-2 was the causative agent. The pathogenesis of AAC in the context of COVID-19 infection still remains vastly unknown. It has been well established that SARS-CoV-2 uses the angiotensin-converting enzyme 2 (ACE2) receptor to mediate cellular entry. Direct invasion of the biliary tree and the vascular endothelium of the gallbladder have both been proposed as possible pathophysiological mechanisms, as ACE2 receptor is highly expressed in these structures[38]. It should be noted that in one case where a percutaneous transhepatic gallbladder drainage was performed, SARS-CoV-2 RNA bile sample was negative[37].

CONCLUSION

AAC represents a diverse clinical entity, affecting both healthy and critically ill patients. We must emphasize that multiple pathogenic mechanisms may concurrently contribute to the development of AAC in varying degrees. Awareness of the implicated pathogens is essential since it will allow a more focused examination of the histopathological specimens. In conclusion, additional research and a high degree of clinical suspicion are needed to clarify the complex spectrum of mechanisms that are involved in the pathogenesis of AAC.

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