Case Report

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Splenic Infarct In An Immunocompetent Patient With CMV Infection. A Case-Report And A Review Of The Literature

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1. Abstract

Infection with Cytomegalovirus (CMV) is very common among the adult population, with a prevalence of 50-90%, varying according with age, country and socioeconomic status. Although its presentation in immunocompetent adults is usually mild and a self-limited flu-like syndrome is often the rule, the immunocompromised patients are quite vulnerable, as this herpes virus can significantly increase the morbidity and mortality among them. CMV infection has been linked with thromboembolic events not only in this subgroup of patients, but also in otherwise healthy adults.

2. Keywords

CMV infecton, immunocompetence, splenic infarct

3. Case Presentation

We present the case of an immunocompetent adult woman who was diagnosed with acute CMV infection and splenic infarcts.

A 56 years old woman presented in the Emergency Department of our hospital with a 2 weeks history of fever without any other symptom. During this period, she reported 3-4 fever waves per day, with a maximum temperature of 39,4C. She had received oral Clarithromycin for 5 days, without any improvement. She had no other medical comorbidities, she was not on a regular medication and she had not travelled abroad the preceding months.

The initial assessment in the ED revealed a normal blood pressure, an axillary temperature of 37,2C, a pulse rate of 105bpm and an Oxygen saturation of 99% (FiO2 21%). Lung and heart auscultation was normal, the examination of the abdomen did not reveal any remarkable finding, there were neither a palpable lymph node, nor a skin rash and the patient had no neurological signs.

The initial laboratory workup demonstrated a normal White Blood Cell count, but elevated Liver Function Tests (SGOT 210U/L, SGPT 249U/L, GGT 239U/L, and ALP 187U/L), an LDH of 783U/L and a CRP of 69,73mg/dl (with an upper normal limit of 5mg/dl).

An abdomen ultrasound was performed, which revealed an increased size of both liver (16,5 cm) and spleen (13 cm). The patient was subsequently transferred to the medical department for further evaluation.

The serologic tests demonstrated positive antibodies of IgM and IgG class for EBV, HSV and CMV, with a high avidity of the IgG class. A PCR test of a blood sample for these viruses was positive for CMV, with a viral load of $3x10^3$ copies/ml. The serologic tests for HIV, hepatitis B and C were negative.

The patient continued to be febrile, so a CT scan of the thorax and the abdomen, with the use of intravenous contrast medium was conducted. It revealed multiple wedge shaped hypodense areas of the spleen, primarily associated with infarcts. It also demonstrated splenomegaly (13,5x9 cm) and hepatomegaly (19 cm). The transthoracic ultrasound showed a mild pericardial effusion (<5mm), without any other major pathologic finding. The administration of an antiviral agent was considered, but since our patient was immunocompetent, it was ruled out as an option.

A further laboratory evaluation revealed increased levels of IgM anticardiolipin (105MPL/ml with an upper limit normal of 18MPL/ml) and β2GPI antibodies (133GPL/ml with an upper limit normal of 18GPL/ml). The patient was also homozygote for the MTHFR 677 mutation, with normal levels of blood homocysteine and a heterozygote for the FII 20210 mutation. As far as SLE is concerned, the anti-ds-DNA and ENA screening were also negative

Gradually the fever subsided and the patient was discharged. At a 2 week follow-up the abdomen ultrasound revealed a smaller size of the spleen (10 cm) and the viral load of CMV was diminished as well.

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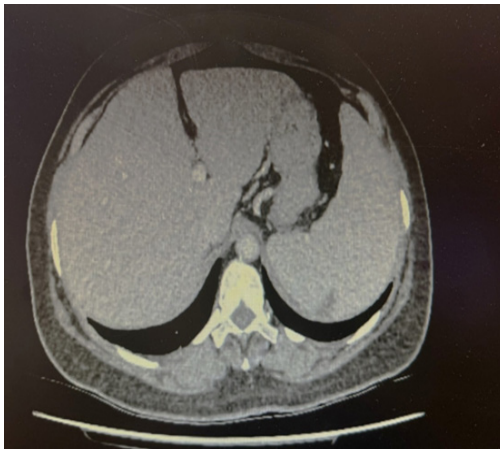
Table 1: Laboratory results on admission, at discharge (1 week after admission) and 2 weeks later

	Admission	Discharge	2 weeks later	Normal range
WBCs(K/ml)	7, 48	6,23	5,90	5,2-12,4
Hgb(g/dl)	12,6	10,1	11	12-18
HCT%	37,8	31,2	35,6	37-52
PLTs(K/ml)	252	225	338	130-400
PMNs%	48	27,1	31,8	40-74
LYMPH%	45	53,2	53,8	19-48
SGOT(U/L)	**210**	**64**	**32**	**5-34**
SGPT(U/L)	**249**	**123**	**28**	**0-55**
ALP(U/L)	**187**	**173**	**100**	**40-150**
γGT(U/L)	**239**	**267**	**119**	**9-36**
LDH(U/L)	**783**	**465**	**375**	**125-220**
Cr(mg/dl)	0,75	0,67	0,87	0,57-1,11
Ur(mg/dl)	19	20	26	15-43
K(mmol/L)	4,1	3,9	5	3,5-5,1
Na(mmol/L)	141	140	140	136-145
Bilirubin(mg/dl)	0,40	0,30	0,40	0,20-1,20
CRP(mg/dl)	**69,73**	**30,65**	**5,74**	**0,00-5,00**
C M V IgM(AU/ml)	**138**	**74,4**	**51,5**	**Positive>22**
CMV IgG(IU/ml)	**90,20**	**119**	**178**	**Positive>14**
CMV viral load	**3 x 1 0 ³ copies/ml**			
HSV 1,2 IgM index	**2,89**			**Positive>1,1**
HSV 1,2 IgG	<0,50			**Positive>1,1**
VZV IgM index	**1,28**			**Positive>1,0**
VZV IgG mIU/ml	**2138**			**Positive>150**

Figure 1: CT scan of the abdomen. The arrow points the splenic infarct.



4. Discussion

Thromboembolic events are considered a relatively rare manifestation in the course of CMV infection, with only a few small series of patients and case reports in the medical literature [1]. In a 2011 meta- analysis, the incidence of thrombosis in hospitalized patients with CMV infection was 6,4% [2]. Later in 2018, a systematic review tried to elucidate the main characteristics of patients with this complication [1]. Thrombosis was reported mainly in portal, mesenteric and splenic veins and the majority of patients complained of abdominal pain. In search of possible risk factors, the use of oral contraceptives and the presence of antiphospholipid antibodies (APA) were the most common acquired predisposing factors, whereas the factor II G20210 and the factor V G1691A were the most common inherited among the patients in the review.

Although the pathophysiology of thrombosis remains unclear, CMV is considered a vascular pathogen. The main theories describe the shift of balance between pro- and anticoagulation, through the activation of endothelial cells, the subsequent adhesion of platelets and leukocytes and the increased production of TGF-β, PDGF and pro-inflammatory cytokines [3]. The CMV associated hepatitis also contributes to the inflammatory changes of the vasculature [1]. All these events create the proper conditions for thrombus formation. The transient increase in APA production, observed in some patients with CMV infection also contributes to the thrombogenic manifestations, through the activation of smooth muscle cells. In a 2017 case report, Kelkar et al. proposed the formation of anti- β2GPI antibodies against TIFI, a CMV derived peptide, resembling the phospholipid binding site of β2GPI [5]. The coagulation cascade is consequently activated on the surface of endothelial cells, where these antibodies bind with the endogenous β2GPI. This theory was based on a study by Gharavi et al. who showed that mice injected with TIMI, produce aPL, an antibody which increases thrombus formation, in a way similar to the patients with SLE [6].

Despite the potential danger, the overall morbidity and mortality of splanchnic vein thrombosis remains low and the majority of patients fully recovers after the administration of low molecular weight heparin or vitamin K antagonists. However the duration of anticoagulation treatment is not fully determined. Some physicians treated the patients until the APAs disappeared, whereas others stopped the medications 2 months after APAs disappearance [1]. Splenic rupture, a potentially life threatening condition has been rarely described during the course of CMV infection, as it is mainly a complication of primary EBV infection or malaria [7].

5. Conclusion

Although CMV infection is very common among the adult population, the occurrence of infarcts in this setting remains relatively low. A number of theories and experimental models have

tried to explain the main pathophysiological mechanisms through which thrombosis develops. Nevertheless, further research in this field is warranted, in order to fully elucidate why some patients develop this rare complication, whereas other do not.

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