areas and others to colder areas, are easy and intuitive to interpret[7]. Focusing on the subject at hand, innovative data have recently been published on the potential use of this technique to establish the differential diagnosis between cellulitis and pseudocellulitis, yielding a sensitivity of up to 95.2%[8]. However, to our knowledge, this is the first case in which it has been used for the clinical follow-up of this condition, to determine the response to treatment. More extensive studies are required.

In conclusion, thermography is an imaging technique that is simple to perform during a consultation and is easy and intuitive to interpret, with potential application in the field of infectious diseases for the diagnosis and follow-up of skin and soft tissue infections by detecting subtle changes in temperature.

Ethical considerations

The patients gave their informed consent for the publication of the images from this study.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Cerebral venous sinus thrombosis and portal vein thrombosis associated with acute cytomegalovirus infection in an immunocompetent patient

Trombosis venosa cerebral y portal asociadas a infección aguda por citomegalovirus en paciente inmunocompetente

In recent years, evidence has emerged that viral infections may play a role in atherosclerosis and thrombosis. Herpes viruses have been shown to cause atherosclerosis in experimental models and have been detected in atherosclerotic lesions in humans. However, the presence of cerebral venous thrombosis (CVT) associated with acute cytomegalovirus (CMV) infection in immunocompetent patients has rarely been described. We present the case of a 28-year-old woman admitted with CVT in the context of acute CMV infection, after having two generalised tonic-clonic seizures. Her history of interest included the use of oral contraceptives. During the initial examination, she was conscious, with amnesia for the episode, mixed aphasia and laterocervical adenopathies, with no other data. She had fever spikes during the first days of admission. Brain CT and CT angiography of cerebral arteries showed complete occlusion of the left transverse and sigmoid sinuses and posterior temporal infarction with haemorrhagic transformation (Fig. 1, left), with no signs of arterial vasculitis. Intravenous heparin and levetiracetam were started. A complete analytical study was requested with autoimmunity, serologies (herpes virus, hepatitis, HIV and *Treponema pallidum*), chest X-ray, echocardiogram and CT of the chest, abdomen and pelvis. Cerebrospinal fluid (CSF) showed 5 leukocytes/μl, 225 erythrocytes/μl, glucose 70 mg/dl and protein 77.7 mg/dl. Gram staining, which did not show microorganisms, and culture for bacteria, which was negative, were performed. The electrocardiogram showed sinus rhythm. The electroencephalogram showed slow left temporal focal activity with bilateral diffusion. In the blood tests, the high atypical lymphocyte count (LUC/LYC) (12.4%) was striking, without anaemia or thrombocytopaenia, with an erythrocyte sedimentation rate of 61 mm in the first hour, C-reactive protein of 3.8 mg/dl, and in biochemical tests, elevation of transaminases and alkaline phosphatase (GOT: 207 IU/l, GPT: 211 IU/l, GGT: 166 IU/l, alkaline phosphatase: 335 IU/l), positive IgM serology for CMV with negative IgG. After testing positive for CMV, treatment with intravenous ganciclovir was started for 14 days at a dose of 5 mg/kg/12 h, and five days after the start of treatment, quantitative plasma PCR was performed for CMV DNA, which was positive (>700 copies/ml). The CT of the chest, abdomen and pelvis showed a non-occlusive portal thrombus in its left branch and a hypodense lesion in the right branch, probably related to thrombosis at that level (Fig. 1, right). The patient responded favourably, her fever disappeared and a PCR for CMV was negative. She was discharged and prescribed acenocoumarol and levetiracetam.

At three months, the hypercoagulability study (prothrombin time, activated partial thromboplastin time [ratio], lupus anticoagulant, IgG and IgM anticardiolipin antibodies, protein C, protein

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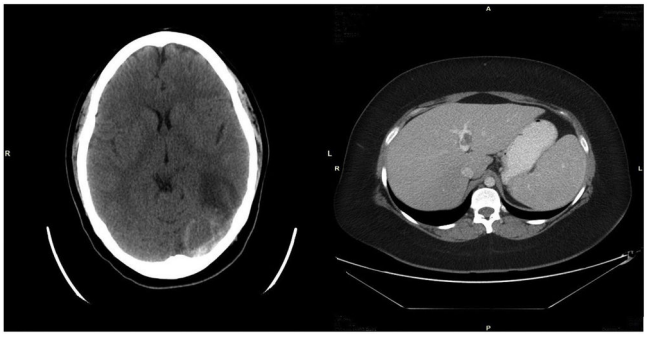

Figure 1. Left. Brain CT shows hyperdensity of the left transverse sinus associated with posterior temporal infarction with haemorrhagic transformation.
Right. Abdominal CT reveals a non-occlusive thrombus in the portal vein.

S, antithrombin), performed after replacing acenocoumarol with low molecular weight heparin, was normal and CT showed permeable sinuses, so anticoagulation was suspended. It was not possible to perform further serological tests due to loss to follow-up.

CMV infection in immunocompetent patients is usually asymptomatic. In 10% of cases, it gives rise to a mononucleosis-like syndrome. It rarely causes a serious infection, the most affected organs being the gastrointestinal tract and the central nervous system. Less frequently, haematological disorders, or thrombosis of the venous or arterial vascular system appear, with hepatic, pulmonary, cardiac or ocular involvement.[1] The occurrence of thrombotic events associated with CMV infection has been described mainly in immunocompromised patients (HIV infection, transplant recipients). In recent years, thrombotic phenomena have been described in immunocompetent patients, generally with other associated risk factors (inherited or acquired thrombophilia, use of contraceptives or hormonal treatments, tobacco use).[2–4] The pathophysiology of thrombotic events associated with CMV infection is unknown. It has been suggested that infection of endothelial cells might increase the expression of adhesion molecules and tissue factors on their surface and trigger platelet adhesion and aggregation on the vascular wall. Meanwhile, an immune response would be produced through humorally-transmitted cytokines and/or inflammatory cells that would contribute to vascular damage. The infection could also activate factor X and thrombin formation, increase circulating levels of von Willebrand factor and factor VIII, and trigger the transient appearance of antiphospholipid antibodies.[5–7]

It has also been suggested that the infection might inhibit apoptosis of smooth muscle cells, causing their proliferation and migration, a factor that has been related to the development of atherosclerosis and restenosis. In this light, CMV infection is described as a risk factor for vascular thrombosis in patients with solid organ transplants (liver, heart, kidney) and coronary restenosis after stent placement.[8] Anticoagulation for three months seems appropriate in CVT associated with acute CMV infection, but thrombophilia screening should be performed in patients with a personal or family history of thrombosis, and in those without a predisposing factor to identify the need for long-term anticoagulation when faced with an irreversible risk factor.[9] Deconinck et al.[10] raise the possibility of considering antiviral treatment in immunocompetent patients with severe vascular thromboembolism, vascular thromboembolism with a poor response to anticoagulation or severe organic involvement, or patients admitted to intensive care units.

In the reviewed literature, this is the first reported case of CVT and portal vein thrombosis secondary to acute CMV infection in an immunocompetent patient. The purpose of this case is to highlight the importance of diagnosing CMV infection in immunocompetent patients with thrombosis in unusual locations. This would raise the possibility of antiviral treatment and anticoagulation time.

Ethical considerations

The informed consent of the patient was obtained.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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