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Thermography for the follow-up of skin and soft tissue infections

Termografía para el control evolutivo de las infecciones de piel y partes blandas

Dear Editor,

One of the most typical clinical and biophysical characteristics of skin and soft tissue infections is increased temperature[1]. However, these differences in heat can sometimes be difficult to identify on clinical examination and doubts can arise about how the disease is progressing.

We present the case of a 54-year-old woman, with no history of interest, who went to the Dermatology A&E department with a painful lumbar lesion of several days' duration, accompanied by fever. On examination, an erythematous, hot and indurated plaque was observed in the lumbar region and mid-upper gluteal region, diagnosed as a skin and soft tissue infection or infectious cellulitis. As a portal of entry, an ulcerated, pearly, shiny plaque was seen in the intergluteal fold, consistent with genital lichen sclerosus. A smear was taken from the intergluteal fold, in which Gram-positive mixed flora was found. As treatment, a cycle of amoxicillin-clavulanate 875 mg/125 mg orally every 8 h for 7 days was prescribed, potentiated with topical ozenoxacin and fluticasone in combination to be applied to the intergluteal area, with resolution of the symptoms.

At the time of diagnosis, a thermographic image was taken with a FLIR camera adaptable to an iOS device. In the image, a temperature difference of 2.55 °C was observed between the affected and perilesional skin, with follow-up images taken on days 2 and 7 of therapy revealing a decrease in this temperature difference (2.49 °C and 0.66 °C, respectively) until it was practically equal to healthy skin (Fig. 1). It can be seen that the response to antibiotic therapy was notable from the first day and the changes could be clearly visualised in the thermographic image.

Thermography is a non-invasive imaging technique that makes it possible to establish temperature differences by analysing the infrared radiation emitted by the skin, which correlates with its temperature and facilitates the assessment of subclinical inflammation[2]. Its most widespread medical use has been for fever screening during the COVID-19 pandemic[3]. In the field of infectious diseases, it has been used to screen for tinea unguium, to predict residual neuralgia in herpes zoster or to assess pressure ulcers[4–6].

It is a simple and cheap technique that can be carried out with devices that can be adapted to mobile phones. The results, which are visible on a colour map with zones that correspond to hotter

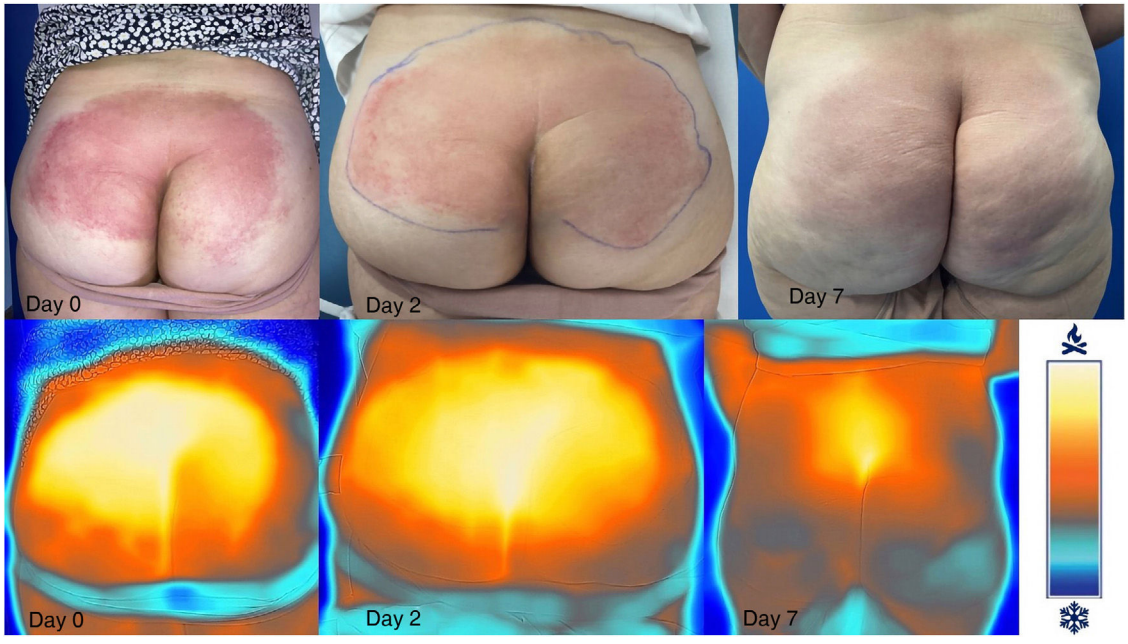

Fig. 1. Clinical and thermographic monitoring of the progression of skin and soft tissue infection on days 0, 2 and 7 of treatment with amoxicillin-clavulanate.

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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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