Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

The anatomical pathology findings of the skin biopsy showed an epidermis with orthokeratin and no significant abnormalities. The superficial dermis showed signs of acute vascular damage, with a mild peripheral leukocyte infiltration, leukocytoclasis, blood extravasation, deposition of fibrinoid material, isolated microthrombi and prominent endothelium. No accompanying eosinophils were identified. It was associated with mild oedema of the papillary dermis, with no other findings, and without involvement of the deep dermis (Fig. 1A and B).

Thus, the initial diagnostic suspicion of allergic vasculitis was confirmed, and the lesions resolved with rest without the need for anti-inflammatory or steroid treatment.

The main skin reaction reported so far after administration of the Moderna vaccine was a local delayed skin reaction approximately 7 days after the first dose, in 94% of cases, with this percentage decreasing with the second dose, as well as the extent of the skin lesion. No cases of anaphylaxis or life-threatening cases were reported.[2,3]

Although most post-vaccination reactions are mild and limited to the site of inoculation, cases of vasculitis have been reported; in particular, allergic vasculitis following influenza vaccination in elderly patients[4] and following pneumococcus, chickenpox and hepatitis A vaccination in an immunosuppressed patient.[5]

Regarding the vaccines developed against the coronavirus, no cases of vasculitis have yet been described after administration, but hypersensitivity with cutaneous vasculitis developed after the above-mentioned vaccines reinforces the role of vaccination against SARS-CoV-2 as a trigger for vasculitis.

The case described was reported to pharmacovigilance because of its temporal relationship with the SARS-CoV-2 vaccination, as it occurred 11 days after the inoculation of the second dose of Moderna.

SARS-CoV-2 vaccines can potentially precipitate cutaneous vasculitis, although well-designed and methodologically sound trials are needed to draw a definitive conclusion.

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Oropharyngeal persistence of SARS-CoV-2: Influence of viral load☆

Persistencia orofaríngea de SARS-CoV-2: influencia de la carga viral

Dear Editor:

Several clinical factors have been associated with oropharyngeal persistence of SARS-CoV-2.[1] Our study included the viral load determination by quantitative PCR (Exact Diagnostics SARS-CoV-2 Standard; Bio-Rad, Texas, USA) of 33 COVID-19 patients with persistent PCR in nasopharyngeal samples > 4 weeks and 33 controls, adjusted for age and sex, who tested negative before. Values are expressed as Log10 mean viral load (N and R genes) and cycle threshold (Ct) values. The Clinical Research Ethics Committee (CREC) of Cantabria approved the study. Student's *t*-test or Mann–Whitney *U* were used to compare quantitative variables, and chi-square or Fisher's test for qualitative variables. All analyses were performed using SPSS 23.0 software (Chicago, IL, USA). A p value < 0.05 was considered statistically significant.

The viral load of COVID-19 patients with mild disease (outpatients) and persistent SARS-CoV-2 was significantly higher than that of their controls in copies/ml (Log10: 7.04 ± 1.81 copies/ml vs. 5.15 ± 2.14 copies/ml; p = 0.018) and Ct of the N gene (25.7 ± 5.6 versus 31.3 ± 6.7 in controls; p = 0.02). Their clinical profile showed no peculiarities (Table 1). Hospitalized patients with persistent SARS-CoV-2 (49 ± 20 days) had the same viral load as their controls (Log10: 6.21 ± 2.06 copies/ml vs. 5.98 ± 1.97 copies/ml; p = 0.73 and Ct values of the N and R genes) and there were no differences in terms of their clinical characteristics (Table 1).

Viral shedding in respiratory samples varies from 2 to 3 weeks after the onset of symptoms, but it has been reported up to 83 days later.[2] Various clinical factors are related to this fact, including male gender, age over 65, the use of invasive mechanical ventilation, the presence of immunodeficiency or diabetes.[2] Some studies find that persistence is more common in seriously ill hospitalised patients with high comorbidity[3]; however, others associate it with asymptomatic cases.[2] The use of corticosteroids and lopinavir/ritonavir also seems to be associated.[4,5] Our study has the limitations of observational studies and we do not know the clinical translation of the persistence of SARS-CoV-2; however, we consider it impor-

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Table 1
COVID-19 patients.

Outpatients	Persistent SARS-CoV-2, N = 15	Controls, N = 15	p
Age (years)	57 (20)	56 (20)	0.85
Sex, n (%)	2 (13%)	2 (13)	0.64
Days until negative PCR	45 (9)	18 (5)	< 0.001
Smoking, n (%)	1 (7)	1 (7)	0.05
ACE or ARB, n (%)	2 (13)	1 (7)	0.55
Comorbidities, n (%)			
Hypertension	3 (20)	1 (7)	0.35
Dyslipidemia	3 (20)	3 (20)	0.60
Diabetes mellitus	0 (0)	1 (7)	0.46
Asthma	0 (0)	0 (0)	–
Atrial fibrillation	1 (7)	1 (7)	0.72
Neoplasm	1 (7)	0 (0)	0.53
COPD	0 (0)	0 (0)	–
Symptomatic, n (%)	11 (73)	7 (47)	0.18
Duration of symptoms, (days)	15 (8)	12 (9)	0.48
Chest x-ray, n (%)	3 (20)	1 (7)	0.42
Pulmonary infiltrates, n (%)	2/3 (66)	0/1 (0)	0.36
Viral load			
Log10 (copies/ml)	7.0 (1.8)	5.1 (2.1)	0.018
N gene Ct	25.7 (5.6)	31.3 (6.7)	0.020
R gene Ct	25.1 (7.0)	29.8 (7.9)	0.12
Hospitalized	Persistent SARS-CoV-2, N = 18	Controls, N = 18	P
Age (years)	71 (16)	72 (13)	0.78
Sex, n (%)	7 (40)	7 (40)	0.63
Days until negative PCR	49 (20)	16 (9)	< 0.001
Smoking, n (%)	0 (0)	4 (22)	0.10
ACEI or ARBs, n (%)	8 (44)	4 (22)	0.14
Comorbidities, n (%)			
Hypertension	11 (61)	7 (39)	0.15
Dyslipidemia	7 (39)	10 (56)	0.25
Diabetes mellitus	2 (11)	2 (11)	0.64
Asthma	1 (5.6)	0 (0)	0.50
Atrial fibrillation	0 (0)	2 (11)	0.24
Neoplasm	0 (0)	1 (5)	0.50
COPD	1 (5.6)	3 (17)	0.30
Duration of symptoms (days)	14 (7)	16 (10)	0.49
Chest x-ray n (%)	18 (100)	18 (100)	–
Pulmonary infiltrates n (%)	12 (67)	17 (94)	0.10
Viral load			
Log10 (copies/ml)	6.21 (2.06)	5.98 (1.97)	0.73
N gene Ct	28.0 (6.6)	28.9 (6.1)	0.67
R gene Ct	27.3 (7.1)	26.8 (5.6)	0.82

Mean (SD) or n (%).
ARBs: angiotensin receptor blockers; COPD: chronic obstructive pulmonary disease; ACEI: ACE inhibitors.

tant to continue analysing the problem of prolonged shedding and fluctuations of the virus.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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