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LUNG DAMAGE IN PATIENTS WITH MICROSCOPIC POLYANGIITIS

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Objectives: We evaluated the frequency of clinical and CT features of lung damage in patients with microscopic polyangiitis (MPA).

Methods: We enrolled 97 patients with MPA, that was diagnosed according to CHCC2012. The median age at disease onset was 50.7±16.6 (M±SEM) years, the median duration of follow-up was 47.6±47.5 (M±SEM) months. 64 (66.0%) patients were ANCA-MPO-positive, 24 patients (24.7%) were ANCA-PR-3-positive, and 9 (9.3%) patients had undifferentiated ANCA. Diffuse alveolar haemorrhage (DAH) was diagnosed by the presence of dyspnea, hemoptysis, anaemia and pulmonary infiltrates on chest CT.

Results: Lung damage was diagnosed in 77 (79.4%) patients. 43 (55.8%) patients had pulmonary damage at the disease onset, while 34 (44.2%) patients developed signs of lung involvement within 8.0±4.1 (M±SEM) months. At baseline, the median pulmonary BVAS was 4.2.⁶ The interstitial changes occurred in more than half of cases at the onset of the disease. The most frequent CT-patterns included pulmonary infiltrates (n=49) and ground-glass opacity (n=39) (table 1). DAH developed in 30 (30.9%) patients, among them 15 (15.5%) had DAH at the onset of the disease.

The pulmonary fibrosis was the most common CT-pattern at the end of follow-up (52 patients). Notably, interstitial damages at the onset of disease were associated with the development of fibrotic changes (OR=4.7, 95% CI 1.7–12.9) and bronchiectasis (OR=9.8, 95% CI 1.2–78.3) at the end of follow-up. The median of pulmonary VDI was 1 (0;4) at the end of the follow up.

PR-3-positive group had higher occurrence of consolidations at the end of the follow-up as compared to patients with anti-MPO-antibodies (53.8% versus 16.0%, p=0.023).

Abnormality	N (%) of cases	
	At baseline (n=77)	At the end of follow-up (n=47)
Interstitial damage	68 (70.1%)	18 (18.6%)
Infiltrates	49 (50.5%)	0 (0%)
Ground-glass opacity	39 (40.2%)	16 (16.5%)
Consolidation	5 (5.2%)	8 (8.2%)
Bronchial wall thickening	10 (10.3%)	18 (18.6%)
Bronchiectasis	4 (4.1%)	18 (18.6%)
Bronchiolitis	3 (3.1%)	5 (5.2%)
Pleural lesions	11 (11.3%)	5 (5.2%)
Pleural effusion	12 (12.4%)	0 (0%)
Pleural thickening	3 (3.1%)	5 (5.2%)
Pulmonary fibrosis	14 (14.1%)	52 (53.6%)
Honeycombing	0 (0.0%)	4 (4.1%)
Atelectasis	3 (3.1%)	8 (8.2%)
Emphysema	0 (0.0%)	13 (13.4%)
Pulmonary hypertension	0 (0.0%)	7 (7.2%)

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Conclusions: In patients with MPA, the CT signs of interstitial damage were usually reversible. However, they predicted a higher incidence of lung fibrosis and bronchiectasis at the end of follow-up. DAH occurred in one third of patients with MPA.

Disclosure of Interest: None declared

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THE MYRIAD OF NEPHRITIS IN LEVAMISOLE-ADULTERATED COCAINE VASCULOPATHY

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Background: Up to 88% of cocaine is tainted with levamisole, an anthelmintic withdrawn from the market due to toxicity. Since 2010 levamisole-adulterated

cocaine vasculopathy (LACIV) patients, characterised by retiform purpura, ear necrosis, multisystemic compromise and positivity for multiple autoantibodies, have been reported. Renal involvement is the most serious and heterogeneous manifestation.

Objectives: To describe the renal involvement of patients with LACIV.

Methods: We describe the renal manifestations of 30 patients with LACIV evaluated from December 2010 to May 2017.

Results: All patients were mestizo, median age of 31 (IQR 27–38), male:female ratio 5:1, time from symptoms to diagnosis 12 months (IQR 6–24). Nephritis found in 57%, creatinine elevation in 40%, median 1.85 mg/dl (IQR 1.2–4.0), 70% had proteinuria, median 3184 mg/day (IQR 552–5100), 58% in nephrotic-range; 88% had hematuria and 41% pyuria and cilindruria. Biopsy was performed in 7 patients (41%), showing immune complex mediated extracapillary glomerulonephritis (29%), membranous glomerulonephritis (29%), pauci-immune proliferative glomerulonephritis (14%), focal and segmental glomerulosclerosis (14%) and C3 mediated extracapillary glomerulonephritis (14%) (image). Three patients (10%) developed end-stage kidney disease.

Image: **A. Membranous glomerulonephritis:** HE: glomerular thickened capillary walls, mild mesangial cellularity increase. **B. GMS:** capillary wall thickening and spikes (arrow). **C. DIF:** strong and diffuse IgG deposits on capillary walls. **D. Immune complex mediated Extracapillary Focal Necrotizing Glomerulonephritis:** HE: endocapillary proliferation, epithelial crescent (arrow). **E. GMS:** capillary wall rupture next to the crescent (arrow). **F. DIF:** Mesangial strong and diffuse IgG deposits. **G. C3 mediated extracapillary glomerulonephritis:** HE: glomerular capillary narrowing, epithelial crescent (arrow). **H. GMS:** normal capillary networks, epithelial crescent (arrow). **I. DIF:** C3 strong and diffuse positivity. **J. Pauci-immune proliferative glomerulonephritis:** PAS: epithelial crescents (arrows and asterisk). **K. Trichrome:** epithelial crescent (arrow). **L. DIF for C3:** weak deposits on capillary tuft. Non-immune complexes. **M. Focal and segmental glomerulosclerosis:** HE: segmental sclerosing lesions (arrow). **N. PAS:** segmental sclerosing lesions (asterisks). **O. DIF for IgM:** nonspecific positivity on a sclerotic segment.

Conclusions: Although skin manifestations are the most characteristic and prevalent features in LIVEN, renal involvement is frequent, clinically and histologically heterogeneous, and potentially serious. The great heterogeneity on the histopathological findings suggests the participation of different physio-pathological mechanisms, establishing renal biopsy as necessary to identify the type of nephropathy and thus, optimal guidance of therapy.

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