

## REFERENCE:

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**AB0202** **DYSPHAGIA: POOR PROGNOSIS IN IDIOPATHIC INFLAMMATORY MYOPATHIES**

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**Background:** Dysphagia has been reported to occur in 10% to 73% of these patients and can be present at any time during the disease process (1).

**Objectives:** The primary objective of the study was to evaluate the prevalence of dysphagia in a cohort of patients with idiopathic inflammatory myopathy (MII) and to evaluate factors associated with the presence of dysphagia. The secondary objective was to evaluate the factors associated with severe dysphagia.

**Methods:** Retrospective, observational study, which included patients with a diagnosis of MII according to modified classification criteria of Bohan and Peter (1992-2018). Demographic data, clinical characteristics, laboratory data, autoantibodies, imaging studies, videodeglutition, muscle biopsy and EMG were recorded.

Severe dysphagia was considered: one in which oral feeding was contraindicated and/or which required nasogastric tube feeding (SNG) either by clinical evaluation or by videodeglutition study. The rest of the patients with dysphagia who did not present a contraindication to oral intake during the course of the disease were considered mild/moderate dysphagia.

**Results:** 94/110 patients were included, 76% female, mean age at diagnosis: 48 years (SD  $\pm$  14). Idiopathic dermatomyositis was the most frequent subtype of myopathy (64%). Dysphagia occurred in 53/94 patients (56.4%) and it was presented at the beginning of the disease in 31/94 (32%). Severe dysphagia was found in (22/94) 23%.

When analyzing the clinical features of patients with myopathy and dysphagia, it was found that Idiopathic dermatomyositis was the most frequent MII in these patients (71%). Patients with dysphagia presented: proximal muscle weakness 90%, neck muscles weakness 47%, and respiratory muscle weakness 27%.

Treatment received: 90/94 (97%) oral glucocorticoids, mean dose 48 mg of prednisone (Range 4 -100 mg.), pulses of Intravenous methylprednisolone was indicated in 25 patients (27.5%). The main steroid sparing agents used were: 72% methotrexate, followed by 33% azathioprine.

Significant association was found between dysphagia and weakness of neck muscles, respiratory muscles, of glucocorticoid pulses, gamma globulin and mortality (data not shown). In the Logistic Regression analysis, no variable was independently associated with the presence of dysphagia.

When analyzing the relationship of severe dysphagia and factors associated, a significant association was found with the requirement of mechanical ventilation, hospitalization in an intensive care unit, serious infections, neoplasia and mortality (Table 1). In the multivariate analysis: no associated factors were found independently.

**Table 1.**

Variable	severe dysphagia (present) n=22 (%)	severe dysphagia (absent) n=72 (%)	p	OR	IC
Weak neck muscles	15 (68%)	18 (25%)	<0.001	8.86	2.75-8.86
Weakness of respiratory muscles	10 (45%)	6 (8%)	<0.001	9.5	2.8-32
pulses of corticosteroids	17 (77%)	8 (11%)	<0.001	25	7.5-89.5
intravenous gammaglobulin	10 (45%)	7 (9%)	<0.001	7.73	2.46-24
intensive care unit	8 (36%)	8 (11%)	<0.001	4.57	1.46-14.26
mechanical ventilation	6 (27%)	5 (7%)	<0.001	5.02	1.36-18.55
death	16 (72%)	10 (14%)	<0.001	16.53	5.89-90

neoplasms	6 (27%)	9 (12%)	<0.001	2.62	0.65-9.2
serious infections	11 (50%)	10 (14%)	<0.002	4.95	1.71-14

**Conclusion:** in patients with MII 56% course with dysphagia at some point in the evolution. Of which 23% of cases were characterized as severe dysphagia. Both dysphagia in general and severe dysphagia were associated with parameters of severity, high cost and poor prognosis. However, in the analysis of multiple variables, this relationship could not be demonstrated.

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**AB0203** **INTRAPLALETET ANGIOGENIC AND GROWTH FACTORS IN SYSTEMIC SCLEROSIS**

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**Background:** Systemic sclerosis (SE) is a serious disease of the connective tissue characterized by dysfunction of the microcirculation. Angiogenesis is a complex process regulated by both angiogenic and angiostatic factors. Normally the functions of these factors are under an adequate balance, however, under certain conditions these factors can be induced initiating disorganized angiogenic phenomena.

**Objectives:** For the first time, the levels of intraplatelet growth and angiogenic factors (normalized by platelet number and volumen) are described in patients with systemic sclerosis.

**Methods:** We included 23 patients with systemic sclerosis (ACR/EULAR) and 16 controls. Patients underwent a platelet-apheresis to obtain platelet-rich plasma (PRP) and platelet-poor plasma (PPP). The samples were subjected to a freeze-thaw process to break the cell membranes and release the platelet content. Subsequently the various factors were measured (VEGF- $\alpha$ , PDGF  $\beta\beta$ , HGF, FGF2, G-CSF, MCP-1, IL-1 $\alpha$ , IL-1 $\beta$ , IL-8, IL-6, IL-13). Finally, the results were normalized according to the value of the plaquetocrit (number and volumen of platelets). We compared the levels in platelet-rich plasma and in platelet-poor plasma. A cut-off value p <0.05.

**Results:** The mean age of the patients was 52 years  $\pm$  9.71, with a duration of the disease of 8 years, 74% were patients with limited SE, 70% of the patients had pulmonary involvement. Both patients and controls, the TFG- $\beta$  was 12 times more concentrated in PRP than in PPP (p <0.0001), IL-1b 13 times more (p <0.0001), IL-6 10 times more (p <0.0001), G-CSF 11 times more (p <0.0001), VEGF- $\alpha$  more concentrated (11.6 times) p <0.005. When the PRP of the patients with scleroderma was compared with the PRP of the controls, only differences were found in the VEGF concentrations, being decreased in the patients with scleroderma respect to controls (p <0.0001). Table 1 and 2.

**Conclusion:** The levels of VEGF- $\alpha$  (intraplatelet) are lower in patients with SE vs controls (p <0.0001). The intraplatelet values of the growth and angiogenic factors are higher than the plasma (patients and controls), finally, the plasma levels of these factors are similar in patients compare with controls. In almost all studies until today the measured levels have been carried out in serum, which are not precise since, in serum, several cells and lysate residues can alter the values, therefore, quantification in plasma is important. The present work is the result of the doctoral thesis of the main author.

**Table 1.** Growth factors values (23 patients with SE and 16 controls)

Factor	PPP (pg/mL)	PRP (pg/mL)	p
TFG- $\beta$ 1	21149,7	237185,2	<0.0001
IL-1 $\beta$	7,7	117	<0.0001