Table 1.

		ΔIGF-1 (ng/mL)		
		<15 (n=52)	≥15 (n=29)	Univariate analyses (p-value)*
Pubertal at rII	n (%)	4 (7.7%)	2 (6.9%)	-
Age at rII (years)	Mean (SD) 95%CI	6.8 (4.0) 5.7;7.9	10.3 (3.3) 9.0;11.5	<0.001**
Basal IGF-1 (ng/mL)	Mean (SD) 95%CI	39.0 (36.9) 28.8;49.3	65.7 (33.8) 52.9;78.6	0.005
Height SDS at rII	n Mean (SD) 95%CI	43 -4.2 (1.9) -4.7;-3.6	25 -3.2 (0.9) -3.6;-2.8	0.029

*Significance level of 0.05; **Significant in the multivariate analysis. CI: confidence interval; SD(S): standard deviation (score)

Conclusions: In patients diagnosed with SPIGFD, IGFGT was performed in >40.0%. However, there is geographic heterogeneity. ΔIGF-1 (ng/mL) ≤15 was reported in >60.0% of naïve patients with SPIGFD; ΔIGF-1 (ng/mL) increased with age at rhIGF-1 initiation. All naïve patients with SPIGFD with LS had ΔIGF-1 (ng/mL) <15.

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Growth and final height of adolescents with systemic juvenile idiopathic arthritis in the transitional age: a monocentric case series

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Introduction: Systemic Juvenile Idiopathic Arthritis (sJIA) is an autoinflammatory disease, characterized by the association of arthritis with fever, often accompanied by rash, generalized lymphadenopathy, hepatosplenomegaly, and serositis. The diagnosis requires adequate exclusion of infectious, autoimmune, autoinflammatory, and oncologic diseases. These patients need to be treated with glucocorticoids plus biologic drugs, anti-IL-1 or anti-IL-6 monoclonal antibodies. The glucocorticoids dose sparing is useful to permit a better growth and pubertal development.

However, chronic inflammation, chronic stress, malnutrition are additional factors that contribute to growth and pubertal impairment.

Materials and Methods: We evaluated a case series of 14 patients (7 M; 7 F; age at the diagnosis: 8-14 years), with an evolution of the disease monocyclic (4/12); polycyclic (7/12); persistent (1/12). All the patients reached the final height.

Results: All the patients were treated with glucocorticoids for a variable period, plus biological drugs (tocilizumab: 21%; anakinra: 7%; canakinumab: 72%). All the patients reached the final height, and their stature was in the target height. Puberty was reached in the physiological time in 12/14 patients (86%); 2/14 males showed a pubertal delay, secondary to prolonged glucocorticoid treatment, maintained during the pubertal age. However, they completed puberty, after the glucocorticoid discontinuation. None of the females showed oligomenorrhea or amenorrhea.

Conclusions: Adolescents with sJIA have a growth delay and/or impairment secondary to chronic inflammation, glucocorticoids, malnutrition, chronic stress. Long-term treatment with glucocorticoids compromises growth velocity and pubertal development.

Most of our patients, started biological drugs in an early window of treatment opportunity, with a significant improvement of growth prognosis. Growth and pubertal delay were associated with a worsen response to treatment and a late start of biological drugs. We highlight the role of the early introduction of anti-IL-1 or anti-IL-6 treatment, as glucocorticoids dose sparing and the winning strategy to reach target height.