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Results: We analyzed 19,531 patients with ischemic stroke pre-
and endovascular therapy(EVT), adjusted for age, sex, comorbidities, and
pre-admission care needs; and in work-flow, stroke severity(National
Institutes of Health Stroke Scale/NIHSS), and in-hospital outcomes.
Results: We analyzed 19,531 patients with ischemic stroke pre-
pandemic versus 2,255 during the pandemic. Hospitalizations/pre-
sentations dropped(weekly adjusted-incidence-rate-ratio[aIRR]:0.48,
95%CI:0.46-0.50), as did population-level incidence of thromboly-
sis(aIRR:0.49,0.44-0.56) or EVT(aIRR:0.59,0.49-0.69). However,
proportions of presenting patients receiving thrombolysis/EVT did
not decline (thrombolysis:11.7% pre-pandemic vs 13.1% during-
pandemic, aOR:1.02,0.75-1.38). For out-of-hospital strokes, onset-
to-door times were prolonged(adjusted-coefficient:37.0-minutes,
95%CI:16.5-57.5), and EVT recipients experienced greater door-
to-reperfusion delays(adjusted-coefficient:18.7-minutes,1.45-36.0).
NIHSS scores and in-hospital mortality did not differ. Conclusions:
The first COVID-19 wave was associated with a halving of presenta-
tions and acute therapy utilization for ischemic stroke at a population
level, and greater pre-/in-hospital treatment delays. Our data can
inform public health messaging and stroke care in future pandemic
waves.

GP.2
Changes in Leptin, CCL16 and sTNF-RII as a distinctive
plasma immune profile in patients with fast progressing ALS
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Background: Amyotrophic lateral sclerosis (ALS) is highly
heterogeneous with survival rate ranging from months to decades.
Approximately 10-20% of patients develop a rapidly progressive
disease and may die within the first year. Therefore, there is an
increasing need for an early detection of unique molecular signatures
associated with more aggressive forms of disease as it may
help identify therapeutic targets. Methods: To identify a unique
molecular signature in fast progressing patients, we recruited 45
sporadic ALS (sALS) patients and 35 age-matched healthy controls
and measured 62 immune markers in plasma using cytokines array.
Results: We found that leptin was significantly downregulated in
plasma of sALS patients and more importantly in fast progressing
disease. Immune markers CCL16 and sTNF-RII were significantly
increased in rapidly progressing disease. We also found that leptin
was significantly downregulated in plasma of SOD1G93A mice
across disease stage. This was caused by an increased proportions
of phospho-AMPK in mice adipocytes and in adipocytes exposed to
fast SALS patients’ plasma. Conclusions: We propose that the
combination of decreased plasma leptin levels and up-regulation in
CCL16/sTNF-RII may be used as a prognostic biomarker to
identify fast progressing ALS patients. This unique immune/meta-
bolic profile may cause dysfunction in metabolic homeostasis.