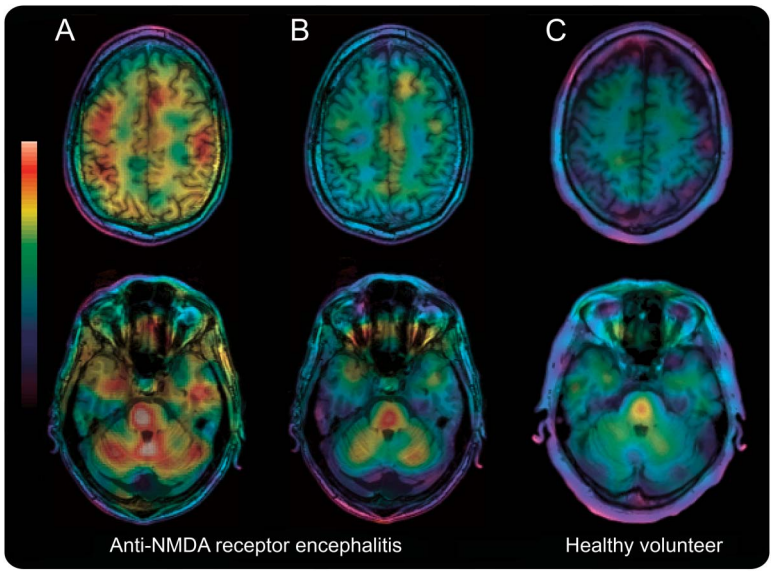
Anti-NMDAR encephalitis

Demonstration of neuroinflammation and the effect of immunotherapy

Figure [^{123}I]CLINDE-SPECT and coregistered MRI in a patient with anti-NMDA receptor encephalitis



At the start of immunotherapy, (A) [^{123}I]CLINDE-binding to TSPO was strongly increased (neocortical distribution volume [V_T] = 6.3 mL/cm^3), but almost normalized (V_T = 4.0 mL/cm^3) after 7 weeks of immunotherapy (B) compared to a healthy volunteer (C) (V_T = 3.0 mL/cm^3).

A 35-year-old man presented with perceptual difficulties and delusions. At presentation, there were orofacial dyskinesias, catatonia, and autonomic instability. Anti–NMDA receptor (NMDAR) antibodies were detected in CSF.[1] Cerebral MRI was unremarkable. At the start of immunotherapy (methylprednisolone and plasmapheresis), [^{123}I]CLINDE-SPECT demonstrated a strongly increased binding to TSPO in cortical and subcortical brain regions similar to the distribution of NMDAR in the brain and different from FDG-PET changes reported in the literature (figure, A). TSPO is present on activated microglia and used as a measure of regional neuroinflammation.[2] After 7 weeks of immunotherapy (figure, B), TSPO binding was close to normal values (figure, C) and the patient was back to work part time as a computer scientist despite mild cognitive problems.

Per Jensen, MD, Daniel Kondziella, MD, PhD, Gerda Thomsen, BS, Agnete Dyssegaard, MS, Claus Svarer, PhD, Lars H. Pinborg, MD, PhD

From Department of Neurology, Rigshospitalet, København, Denmark.

Author contributions: Per Jensen: acquired and analyzed the data. Daniel Kondziella, Gerda Thomsen, and Agnete Dyssegaard: acquired data and revised the manuscript. Claus Svarer: prepared the figure, analyzed data, and revised the manuscript. Lars H. Pinborg: designed the concept, analyzed data, and wrote the clinical summary.

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Correspondence to Dr. Pinborg: lars.pinborg@nru.dk

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