REUMAIMAGING

Reumatologi e Radiologi a confronto:
l'importanza del Decision Making
dalla diagnosi al follow up

«Giovane donna affetta da LES con iniziali disturbi cognitivi»

Prof. Marcello Govoni
UOL di Reumatologia - Dipartimento di Scienze Mediche - Università degli Studi di Ferrara

MR F 43 anni

SINTESI CLINICO-ANAMNESTICA

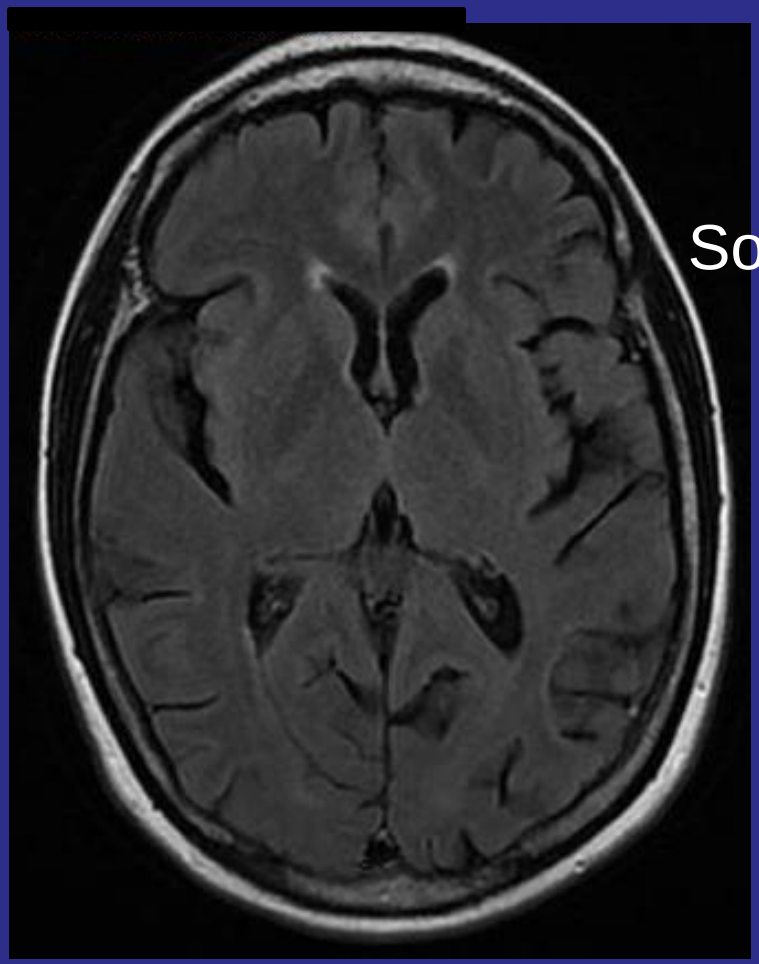
Paziente nota alla nostra U.O. per un quadro di LES in overlap con Sindrome di Sjogren esordito nel 2002 con febbre, poliartralgie, astenia, leucopenia, linfopenia, linfoadenopatie, artrite. ANA + ad alto titolo, anti-SSA, anti-RNP e anti-Sm +, anti DNA e LAC + (unico riscontro);
Pervietà FO

RM encefalo negativa; terapia con steroidi, antiaggreganti e Idrossiclorochina

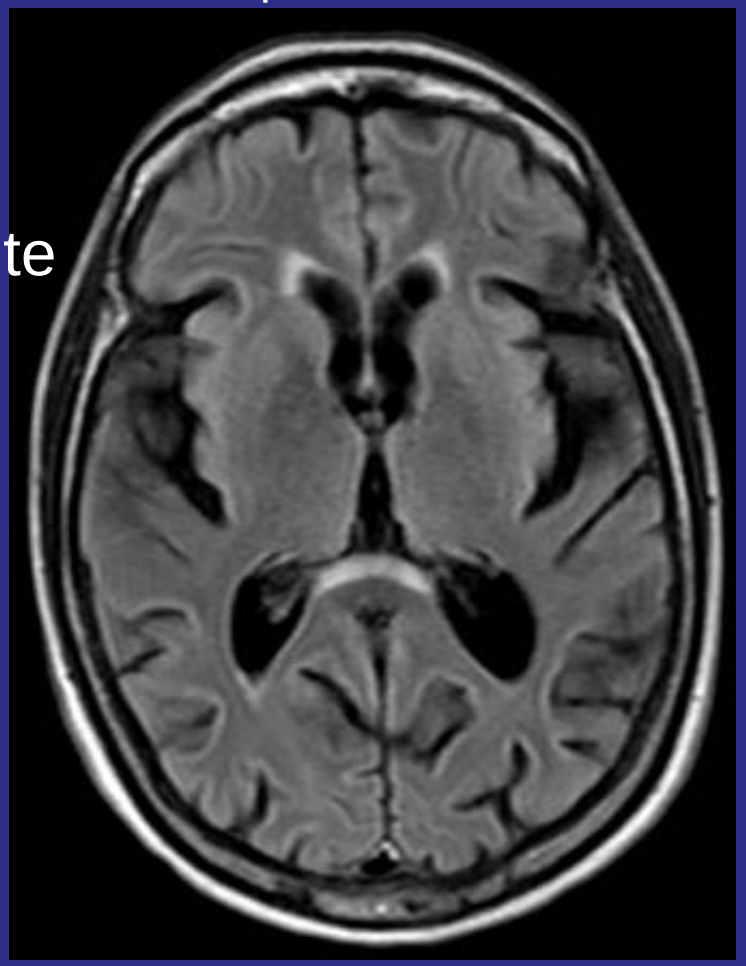
Nel 2007 comparsa di **deficit attentivo-mensici, afasia nominum, disturbi del tono dell'umore, stati di disorientamento spazio-temporale** (**RM encefalo negativa**)
Inizia AZA
Nel 2008 RM encefalo : 3 lesioni T2-iperintense in sede frontale e parietale dx (non più riscontrate a successivi controlli RM).

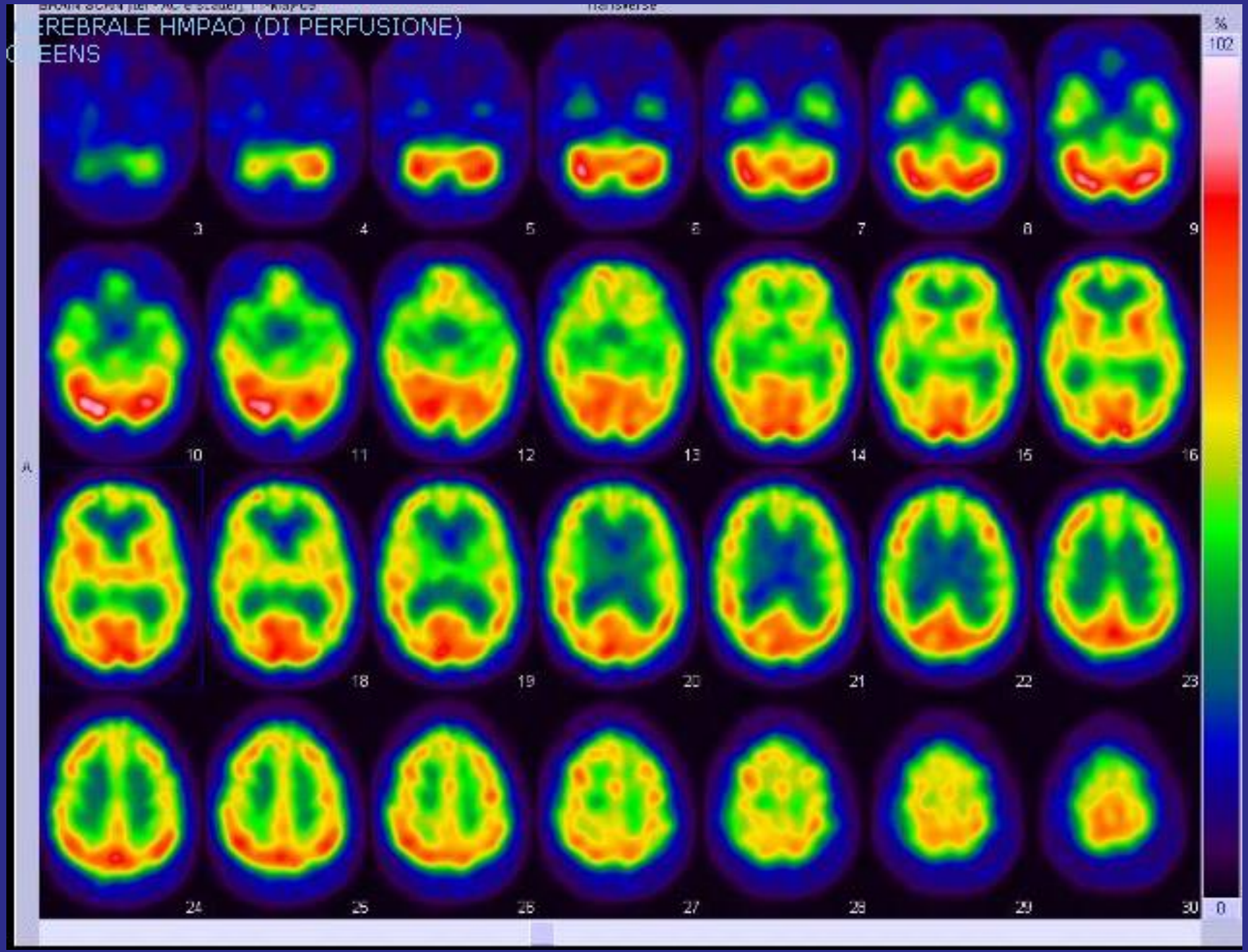
Somministrata **Ciclofosfamide e.v.** (dose cumulativa 3750 mg) + **AZA**, sospesa per quadro pancitopenico.
2011 terapia con basso dosaggio steroideo e **Micofenolato**
Marzo 2013 per **episodio ipomaniacale, disturbo bipolare** (RM encefalo invariata) EEG nn
Ultimamente comparsa di urge-incontinence e peggioramento dei deficit mnesici e del fenomeno di Raynaud con comparsa di lesioni simil-vasculitiche a livello periungueale.

Novembre 2008 → Aprile 2016

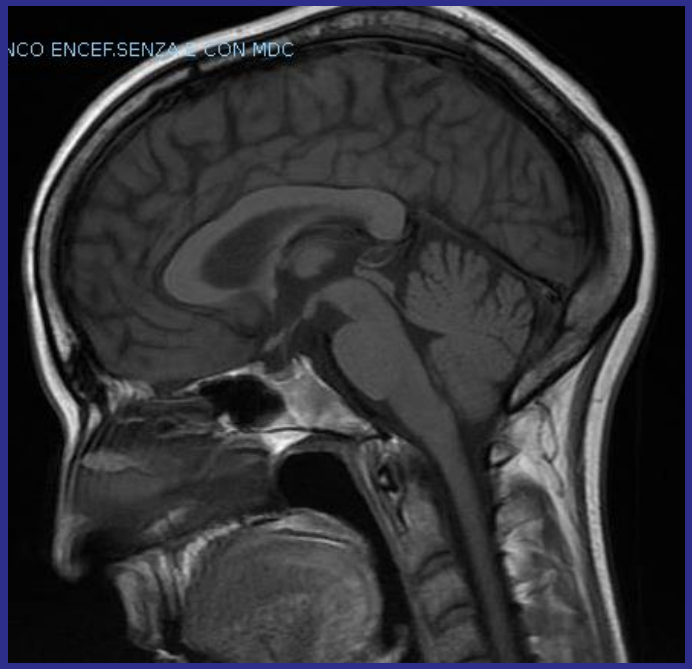


RM
Sostanzialmente
nella norma

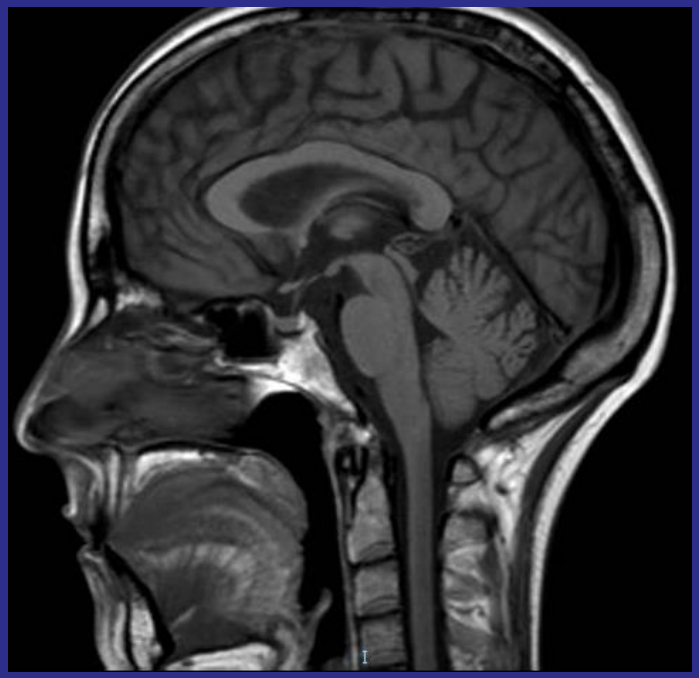



EREBRALE HMPAO (DI PERFUSIONE)
EENS
%
102
0
3
4
5
6
7
8
9
10
11
12
13
14
15
16
18
19
20
21
22
23
24
25
26
27
28
29
30

Novembre 2008

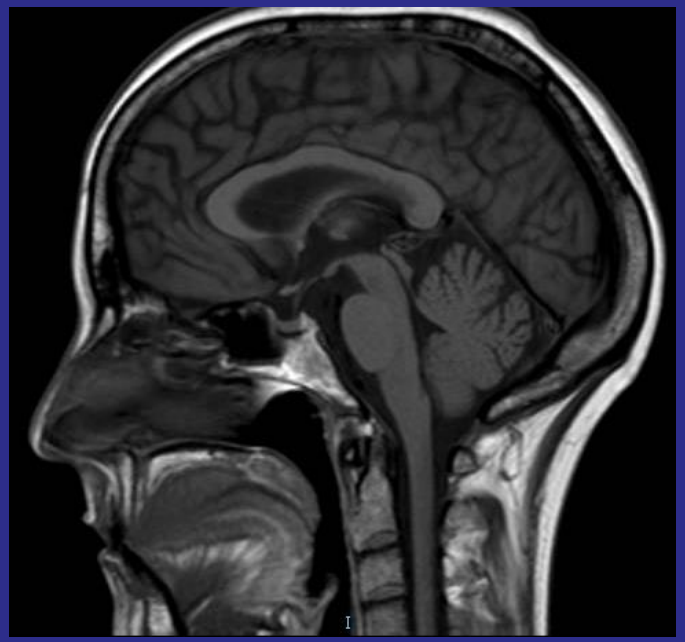


Dicembre 2010

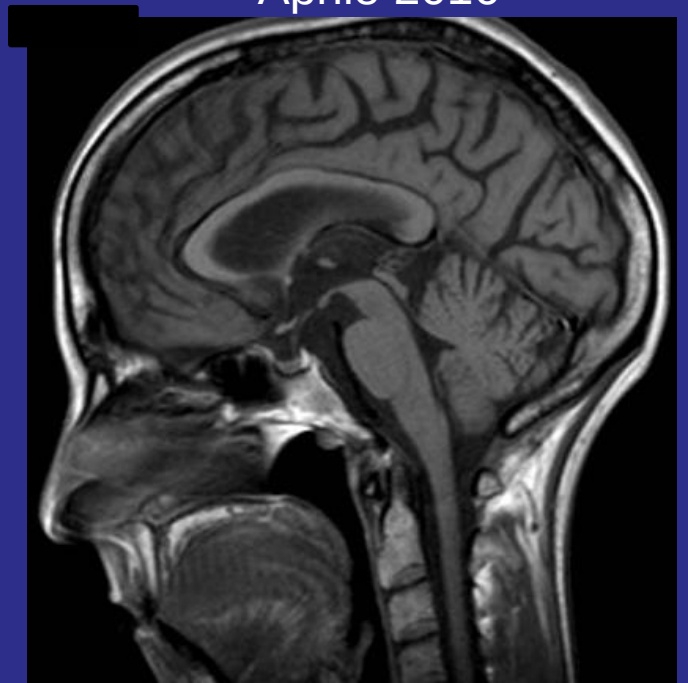


Cosa è cambiato ?

Marzo 2013



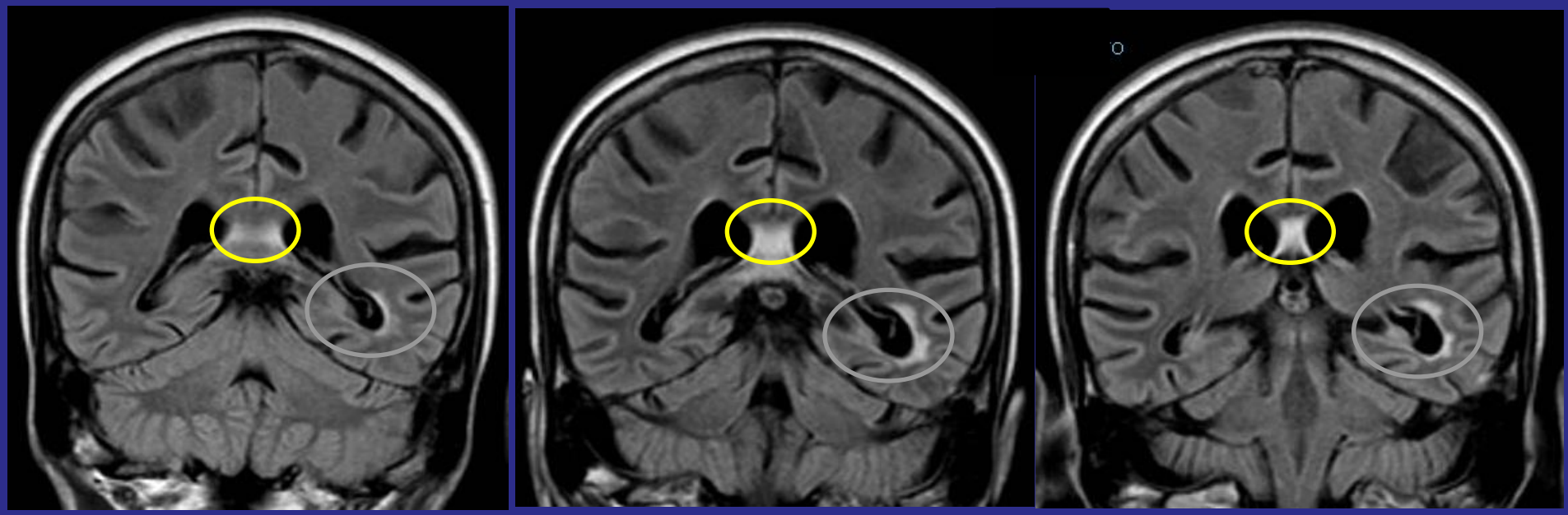
Aprile 2016



Marzo 2013

Febbraio 2015

Aprile 2016



Referto RM ENCEFALO CON E SENZA MDC:

L'esame, eseguito prima e dopo somministrazione di mdc con tecnica Spin Echo, Turbo Spin Echo, Gradient Echo, FLAIR e DWI ed acquisizioni multiplanari, è stato confrontato con precedente del 20.03.13. Al controllo attuale si osserva :

- **incremento volumetrico del sistema ventricolare su base atrofica**, più pronunciato in sede sovra-tentoriale, con consensuale e diffuso maggior ampliamento degli spazi liquorali periencefalici.
- maggiormente estesa **l'anomala iperintensità del segnale proveniente dalla sostanza bianca periventricolare** limitrofa al trigono del ventricolo laterale sx
- Alcune nuove minute areole iperintense di alterato segnale, prive di correlato in diffusione, sono ora riconoscibili in corrispondenza della sostanza bianca sottocorticale in regione frontale anteriore dx e parietale sn.
- **Globale assottigliamento del corpo calloso**.

La somministrazione di mdc non evidenzia potenziamenti contrastografici intracranici con franco carattere patologico. Sostanzialmente invariati i restanti reperti.

CNS lesions detectable by MRI

Acute lesions

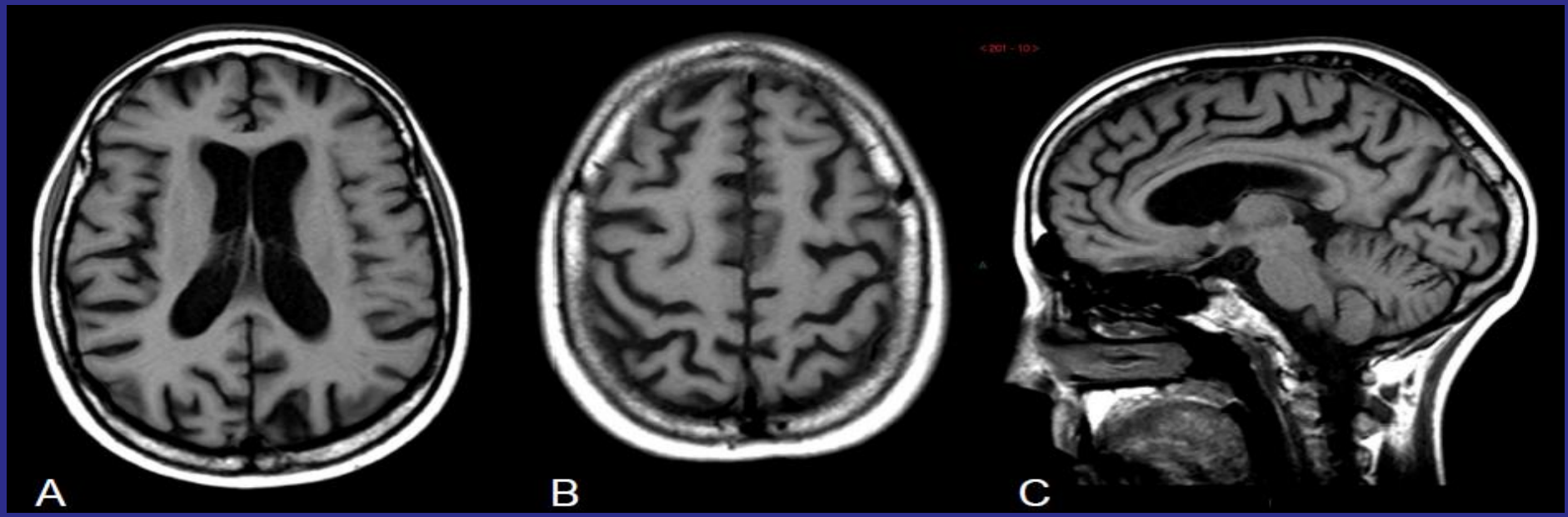
- Large infarcts
- Major hemorrhagic events
- Subarachnoid Haemorrhages
- Inflammatory-type lesions
- Myelopathies
- Posterior Reversible Encephalopathy Syndrome (PRES)

Chronic lesions

- White Matter Hyperintensities
- Grey Matter Hyper-Intensities
- Lacunes
- Brain Atrophy
- Cerebral Microbleeds

Brain Atrophy is defined as a generalized enlargement of peripheral CSF spaces (**diffuse atrophy**). The **focal one** consists of gyrus atrophy or widening of sulci and is usually associated with WMHIs or GMHIs, reflecting chronic microangiopathy.

The best way to evaluate brain atrophy is by **volumetric 3D-T1** or **FLAIR images**, because of the good contrast between CSF signal (hypointense) and brain parenchyma.



Brain atrophy

General population

Usually considered as part of normal ageing (at a mean velocity of 0,1-0,3% / year) with pathologic accelerations (myelin damage, loss of axon integrity, wallerian degeneration, loss of extracellular spaces). Rarely noticed < 50 yrs.

NPSLE

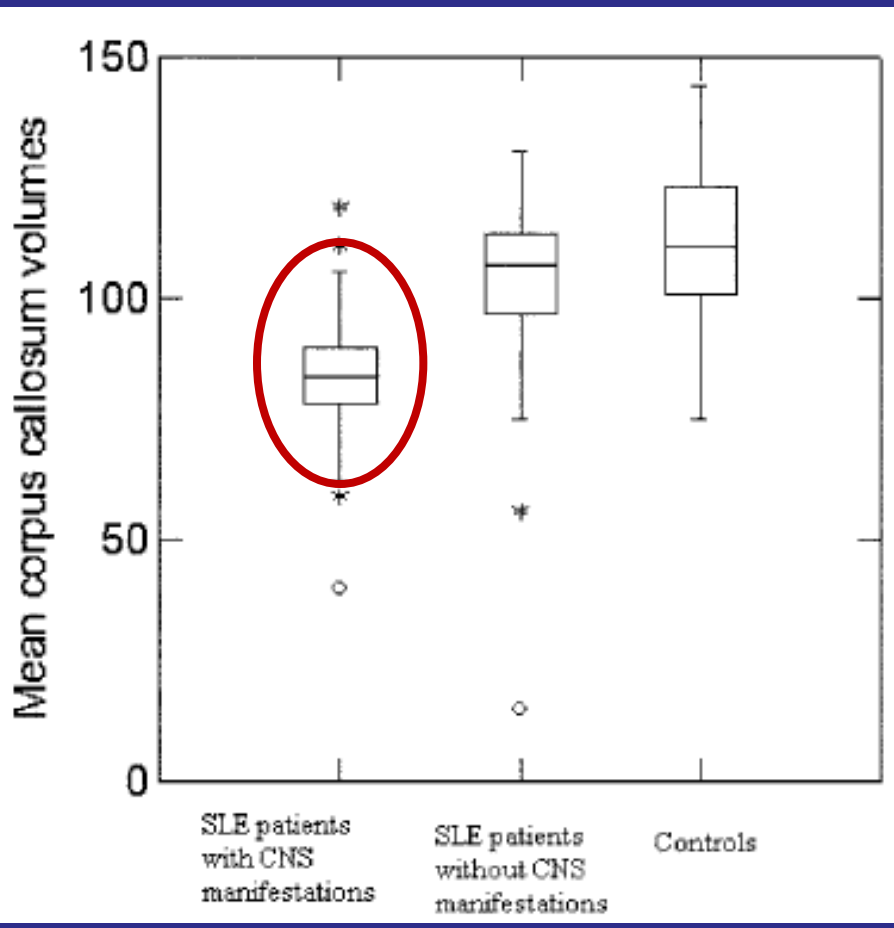
Its prevalence varies widely (different imaging modalities and different patient selection criteria), but systematically differs from general population and often occurs earlier (6-18,5% of the subjects at a mean age of 42,5 years

Atrophy correlates with small vessels disease, number of WMHIs, lacunes, microbleedings, longer disease duration, cognitive disfunction, presence of aPL antibodies and CVD. Association with steroids is controversial.

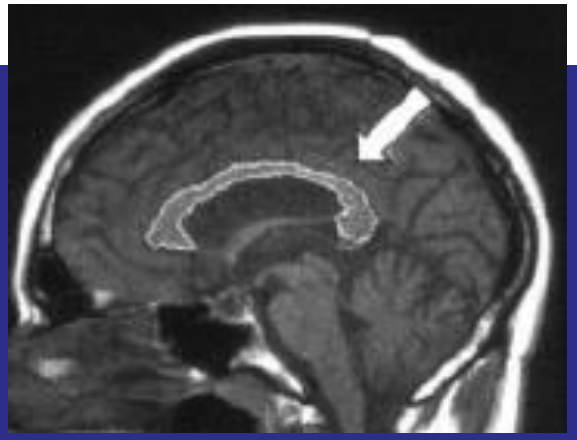
Cerebral and Corpus Callosum Atrophy in Systemic Lupus Erythematosus

ARTHRITIS & RHEUMATISM
Vol. 52, No. 9, September 2005, pp 2783–2789

Simone Appenzeller, Jane Maryam Rondina, Li Min Li, Lilian T. L. Costallat, and Fernando Cendes



21.7 % of patients



In patients with SLE, a reduction in cerebral and corpus callosum volumes is associated with :

- **disease duration**
- **history of CNS involvement**
- **cognitive impairment**

The Journal of Rheumatology

J Rheumatol 2008;35:2348–54

Brain Magnetic Resonance Imaging in Newly Diagnosed Systemic Lupus Erythematosus

MICHELLE PETRI, MOHAMMAD NAQIBUDDIN, KATHRYN A. CARSON, DANIEL J. WALLACE, MICHAEL H. WEISMAN, STEPHEN L. HOLLIDAY, MARGARET SAMPEDRO, SHALINI NARAYANA, PETER T. FOX, CRYSTAL FRANKLIN, PATRICIA A. PADILLA, and ROBIN L. BREY

Objective : to determine the prevalence of cerebral atrophy and focal lesions in a cohort of patients with newly diagnosed SLE (97 pts **< 9 months** from diagnosis underwent brain MRI)

Cerebral atrophy	**18 %**
Focal lesions	**8 %**

Mean age **38 yrs**

These findings suggest that the brain may be affected extremely early in the course of SLE, even before the clinical diagnosis of SLE is made

Take home message

- **Don’t miss early CNS damage in SLE**
- **Screen patients with new diagnosis of SLE**
  - Baseline neuroimaging ?
  - Check for cognitive or subtle neurological impairment
- **Monitor patients during follow up**
  - Repeat MRI (when ? How many times ?)
  - Consider periodic neurologic evaluation and neuropsychological testing

References

- Sarbu N, Alobeidi F, Toledano P et al. Autoimmun Rev. 2015; 14: 153-9
- Appenzeller S, Bonilha L, Rio PA et al. Neuroimage. 2007; 34: 694-701
- Jung RE, Segall JM, Grazioplene RG et al. PLoS One. 2010; 5 : e9302
- Appenzeller S, Rondina JM, Li LM et al. Arthritis Rheum. 2005; 52: 2783-9
- Lauvsnes MB, Beyer MK, Kvaløy JT et al. Arthritis Rheumatol. 2014; 66 : 3387-94