Neuro-inflammation :

Bilan d'encéphalites

APPORTS DE LA TEP AU 18F-FDG

Chawki GHAYOR (Interne DES-MN 3eme semestre)

HCL
HOSPICES CIVILS
DE LYON

Présentation

Généralités

- Définition : encéphalopathie rapidement progressive, liée à une inflammation cérébrale.

- Rare : 5-10 cas/100 000 hab/an selon les études (pays développés).

- Mauvais pronostic : décès ou séquelles dans 7-12%.

Etiologies

- **Infectieuses** (40-52%) : principalement virale

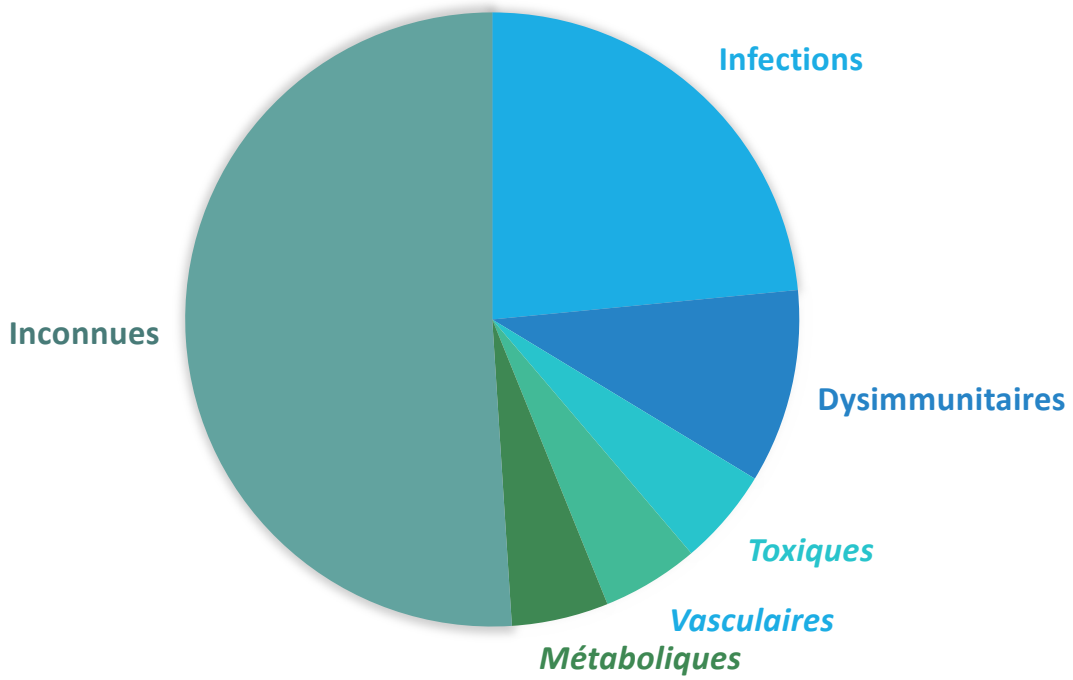
  cf HSV, VZV, *L. monocytogenes* et *M. tuberculosis*

  (>>> … En France)

- **Dysimmunitaires** (ou auto-immune *AE*) (21%)

  : inclus les causes paranéoplasiques

- Autres : toxiques, vasculaires, métaboliques…
- Inconnues : environ la moitié des cas



Encéphalites dysimmunitaires

Classement selon la cible :

- Antigène **intra-cellulaire** : anti-HU..
- **Récepteur synaptique** : anti-NMDAR >>> GABAB, AMPA >...
- **Canaux ioniques** et autres protéines de surface : VGKC (*LGI1, CASPR2)…*

	Syndrome	Diagnostic assay	Frequency of cancer	Main type of cancer
Antibodies against intracellular antigens				
Hu (ANNA1)[8*]	Limbic encephalitis	Western blot	>95%	Small-cell lung carcinoma
Ma2[9]	Limbic encephalitis[†]	Western blot	>95%	Testicular seminoma
GAD[10]	Limbic encephalitis[‡]	Radioimmunoassay	25%[§]	Thymoma, small-cell lung carcinoma
Antibodies against synaptic receptors				
NMDA receptor[11]	Anti-NMDA receptor encephalitis	Cell-based assay	Varies with age and sex	Ovarian teratoma[¶]
AMPA receptor[12]	Limbic encephalitis	Cell-based assay	65%	Thymoma, small-cell lung carcinoma
$GABA_B$ receptor[13]	Limbic encephalitis	Cell-based assay	50%	Small-cell lung carcinoma
$GABA_A$ receptor[14]	Encephalitis	Cell-based assay	<5%	Thymoma
mGluR5[15]	Encephalitis	Cell-based assay	70%	Hodgkin's lymphoma
Dopamine 2 receptor[16]	Basal ganglia encephalitis	Cell-based assay	0%	..
Antibodies against ion channels and other cell-surface proteins				
LGI1[17]	Limbic encephalitis	Cell-based assay	5–10%	Thymoma
CASPR2[18]	Morvan's syndrome[‖] or limbic encephalitis	Cell-based assay	20–50%	Thymoma[**]
DPPX[19]	Encephalitis[††]	Cell-based assay	<10%	Lymphoma
MOG[20‡‡]	Acute disseminated encephalomyelitis	Cell-based assay	0%	..
Aquaporin 4[21‡‡]	Encephalitis	Cell-based assay	0%	..
GQ1b[22]	Bickerstaff's brainstem encephalitis	ELISA	0%	..

Encéphalites dysimmunitaires

Médiation selon la cible

Antigènes intra-cellulaires :

- Médiation LTc
- Sujets âgés
- Cancer associé (CBPC)
- Evolution péjorative

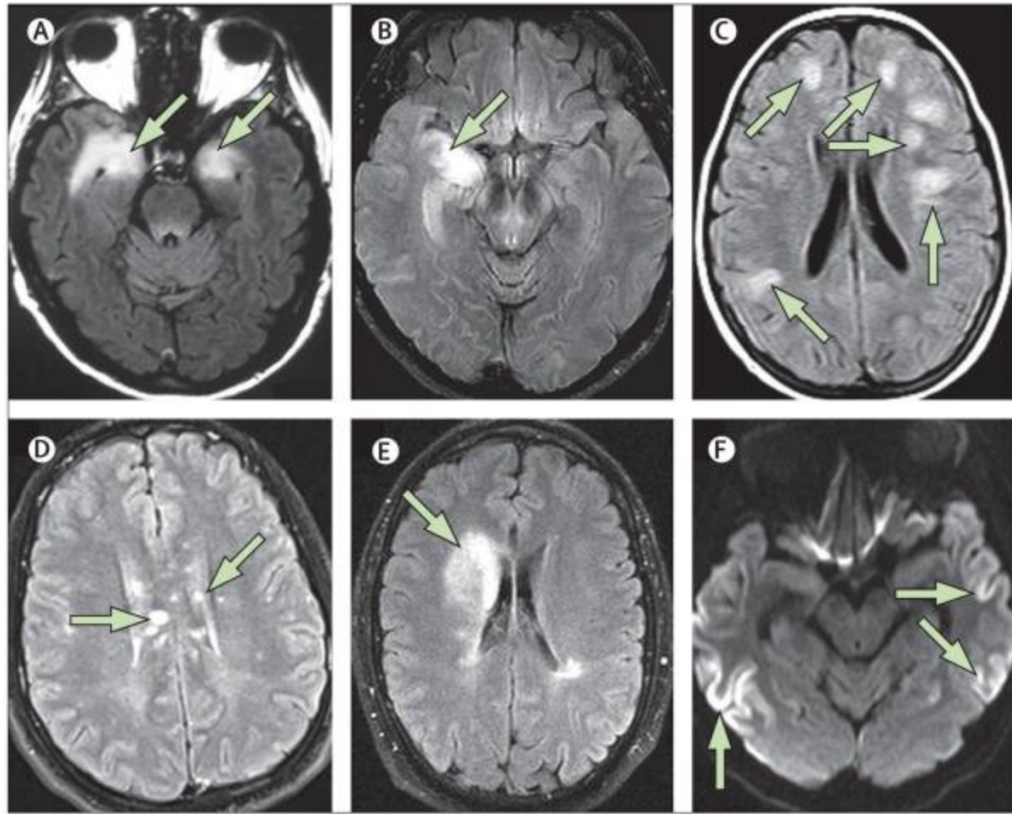
Antigènes membranaires :

- Médiation humorale
- Tout âge
- Cancer non systématique
- Guérison sans séquelle

Diagnostic

Démarche diagnostique

- PL : recherche agent infectieux ou Ac
- IRM : imagerie de 1er intention ; signes peu spécifiques et inconstants
  - HyperT2 et hyperFLAIR temporaux et frontaux
  - Prise de contraste en Gado
- EEG : foyers épileptiques ; ondes lentes



Démarche diagnostique : AE

Diagnostic difficile pour les AE :

- Clinique peu spécifique
- PL : résultats tardifs ; séronégativité fréquente
- IRM : Se seulement 25-50%
- EEG : non spécifique

➔ Immunothérapie probabiliste en urgence

Encéphalites dysimmunitaires

Critères diagnostiques : AE « **possible** »

1. Début subaigu
2. ≥ 1 anomalie (para-clinique) :
   - ➢ SNF, épilepsie, PL ou IRM
3. Exclusion des diagnostics différentiels

Diagnostic criteria for possible autoimmune encephalitis

Diagnosis can be made when all three of the following criteria have been met:

1. Subacute onset (rapid progression of less than 3 months) of working memory deficits (short-term memory loss), altered mental status*, or psychiatric symptoms
2. At least one of the following:
   - New focal CNS findings
   - Seizures not explained by a previously known seizure disorder
   - CSF pleocytosis (white blood cell count of more than five cells per mm^3)
   - MRI features suggestive of encephalitis†
3. Reasonable exclusion of alternative causes (appendix)

A clinical approach to diagnosis of autoimmune encephalitis

Prof. Francesc Graus, MD, Maarten J Titulaer, MD, Ramani Balu, MD, Susanne Benseler, MD, Prof. Christian G Bien, MD, Tania Cellucci, MD, Irene Cortese, MD, Prof. Russell C Dale, MD, Jeffrey M Gelfand, MD, Michael Geschwind, MD, Carol A Glaser, MD, Prof. Jerome Honnorat, MD, Romana Höftberger, MD, Takahiro Iizuka, MD, Sarosh R Irani, MD, Eric Lancaster, MD, Frank Leypoldt, MD, Harald Prüss, MD, Alexander Rae-Grant, MD, Prof. Markus Reindl, PhD, Prof. Myrna R Rosenfeld, MD, Kevin Rostásy, MD, Albert Saiz, MD, Arun Venkatesan, MD, Prof. Angela Vincent, FRS, Prof. Klaus-Peter Wandinger, Patrick Waters, PhD, and **Prof. Josep Dalmau, MD**

Encéphalites dysimmunitaires

Critères diagnostiques : AE « **probable séronégative** »

1. Début subaigu
2. Exclusion des syndromes cliniques auto-immuns
3. ≥ 2 anomalies (para-clinique) + Ac négatifs :
   - ➢ PL, IRM ou biopsie cérébrale
4. Exclusion des diagnostics différentiels

Panel 7

Criteria for autoantibody-negative but probable autoimmune encephalitis

Diagnosis can be made when all four of the following criteria have been met:

1. Rapid progression (less than 3 months) of working memory deficits (short-term memory loss), altered mental status, or psychiatric symptoms
2. Exclusion of well defined syndromes of autoimmune encephalitis (eg, typical limbic encephalitis, Bickerstaff's brainstem encephalitis, acute disseminated encephalomyelitis)
3. Absence of well characterised autoantibodies in serum and CSF, and at least two of the following criteria:
   - MRI abnormalities suggestive of autoimmune encephalitis*
   - CSF pleocytosis, CSF-specific oligoclonal bands or elevated CSF IgG index, or both*
   - Brain biopsy showing inflammatory infiltrates and excluding other disorders (eg, tumour)
4. Reasonable exclusion of alternative causes

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Encéphalites dysimmunitaires

Critères diagnostiques : AE « **définitive** »

1. Début subaigu
2. Anomalies FLAIR MTL bilatérales
3. ≥ 1 anomalies (para-clinique) parmi :
   - ➢ PL ou EEG
4. Exclusion des diagnostics différentiels

Diagnostic criteria for definite autoimmune limbic encephalitis

Diagnosis can be made when all four* of the following criteria have been met:

1. Subacute onset (rapid progression of less than 3 months) of working memory deficits, seizures, or psychiatric symptoms suggesting involvement of the limbic system
2. Bilateral brain abnormalities on T2-weighted fluid-attenuated inversion recovery MRI highly restricted to the medial temporal lobes†
3. At least one of the following:
   - CSF pleocytosis (white blood cell count of more than five cells per mm^3)
   - EEG with epileptic or slow-wave activity involving the temporal lobes
4. Reasonable exclusion of alternative causes (appendix)

A clinical approach to diagnosis of autoimmune encephalitis

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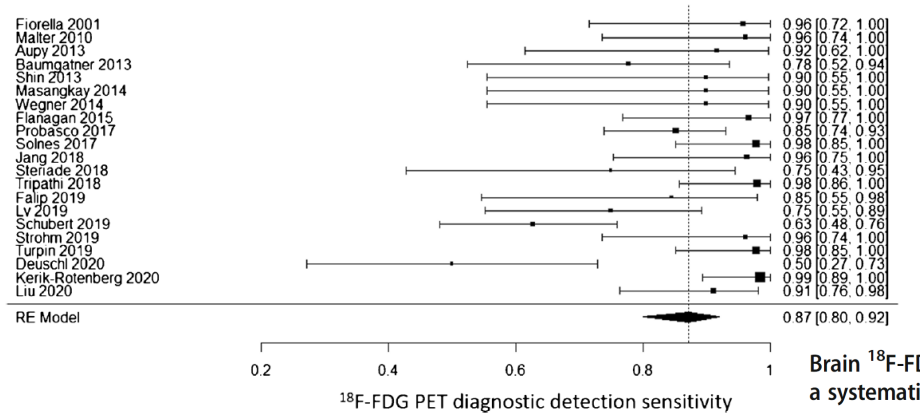
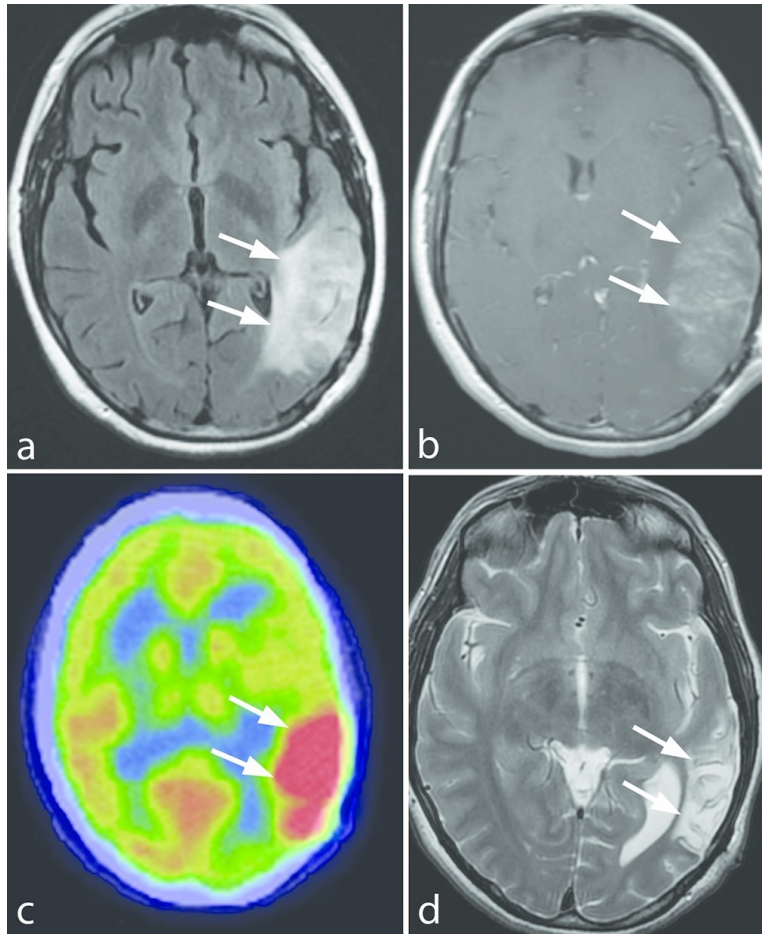
AE : place de la TEP

- Actuellement : recherche de primitif uniquement.
- Performances > IRM : Se 87% (80–92%) versus 56% (46–66%)



Brain ^{18}F-FDG PET for the diagnosis of autoimmune encephalitis: a systematic review and a meta-analysis

Manon Bordonne[1] · Mohammad B. Chawki[1] · Matthieu Doyen[1,2] · Aurelie Kas[3] · Eric Guedj[4] · Louise Tyvaert[5] · Antoine Verger[1,2]



En pédiatrie

- Critères cliniques spécifiques
- Performances TEP > IRM et PL
- Pronostic selon précocité du traitement

Table 2 The proposed classification criteria by AE International Working Group [26]

Categorical features of AE	Possible AE	Probable antibody-negative AE	Definite antibody-positive AE
Acute or subacute onset: Onset of neurologic and/or psychiatric symptoms over ≤ 3 mo in a previously healthy child	Yes	Yes	Yes
Clinical evidence of neurologic dysfunction: Altered mental status/level of consciousness or EEG with slowing or epileptiform activity (focal or generalized) Focal neurologic deficits Cognitive difficulties Acute developmental regression Movement disorder (except tics) Psychiatric symptoms Seizures not explained by a previously known seizure disorder or other condition	≥ 2 features present	≥ 2 features present	≥ 2 features present
Paraclinical evidence of neuroinflammation: CSF inflammatory changes (leukocytosis > 5 cells/mm^3 and/or oligoclonal banding) MRI features of encephalitis Brain biopsy showing inflammatory infiltrates and excluding other disorders	Not available	≥ 1 features present	≥ 1 features present
AE serology: Presence in serum and/or CSF of well-characterized autoantibodies associated with AE	Not available	No	Yes
Exclusion of other etiologies: Reasonable exclusion of alternative causes, including other causes of CNS inflammation	Yes	Yes	Yes

Se	93%
Sp	84%
VPP	89%
VPN	91%

Usefulness of brain FDG PET/CT imaging in pediatric patients with suspected autoimmune encephalitis from a prospective study

Yafu Yin[1] · Jing Wu[2] · Shuqi Wu[1] · Suyun Chen[1] · Weiwei Cheng[1] · Ling Li[2] · Hui Wang[1]

En pédiatrie

- Critères cliniques spécifiques
- Performances TEP > IRM et PL
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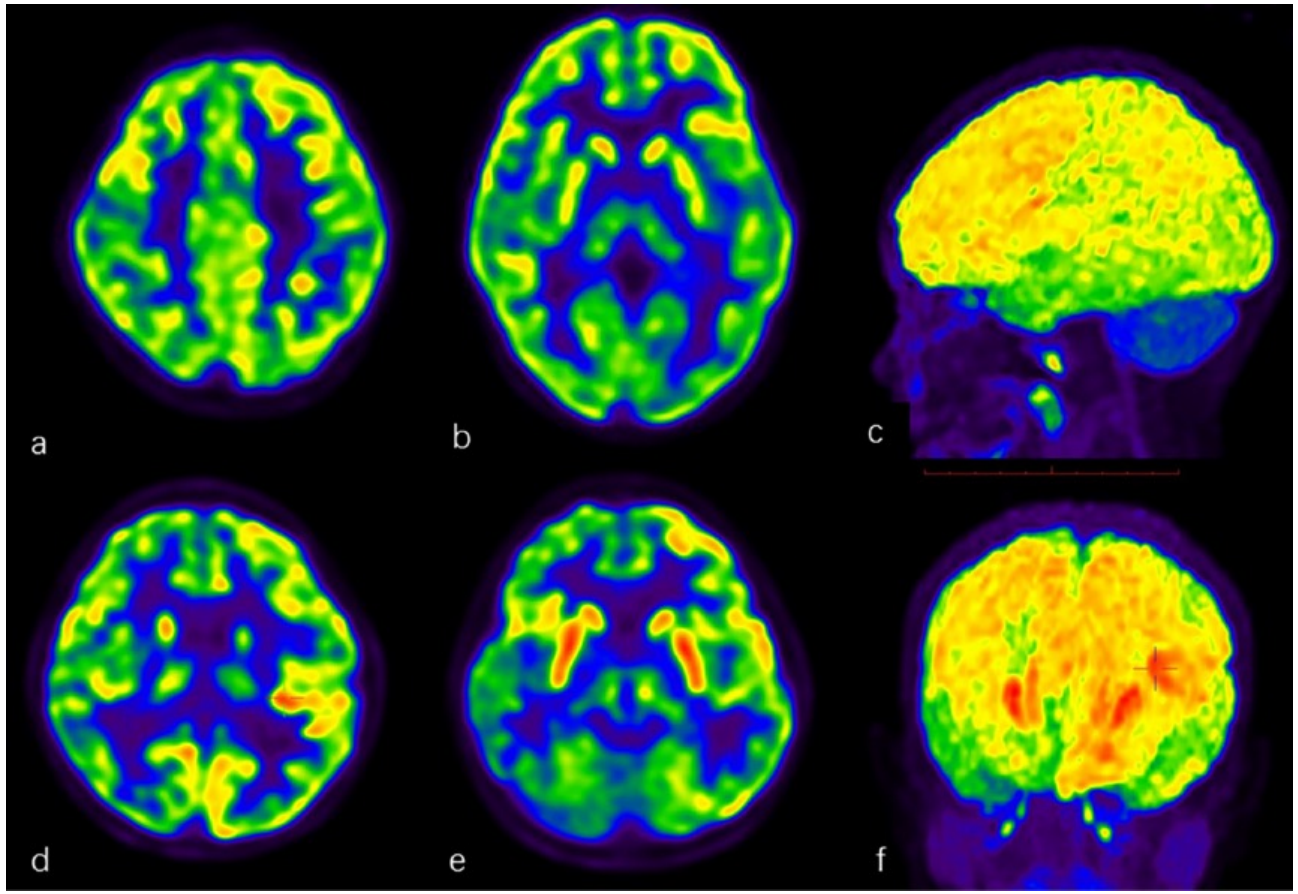

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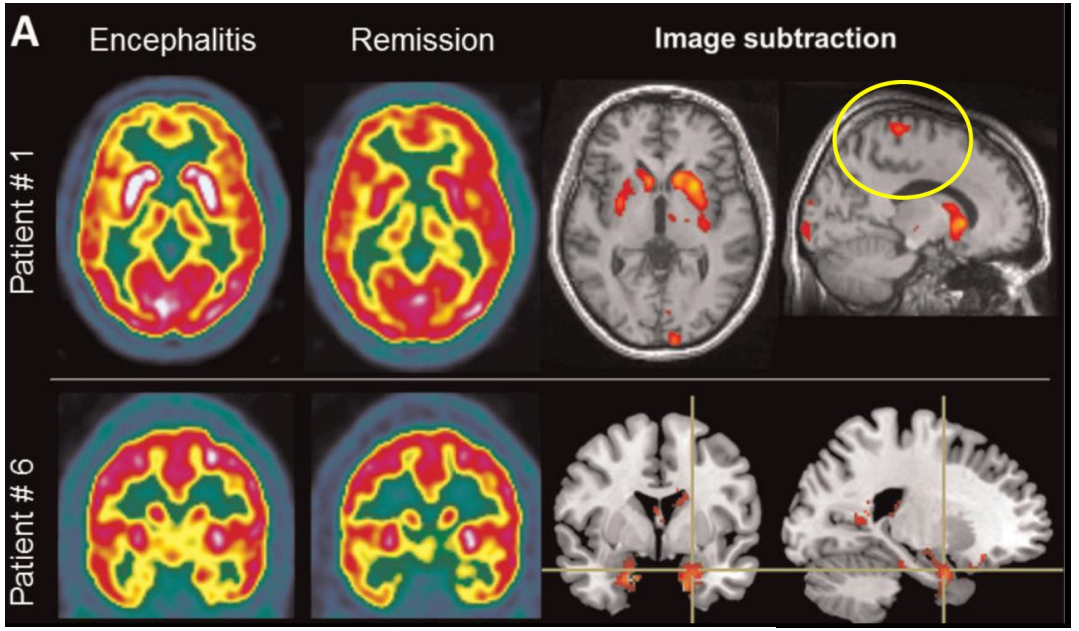
Analyse en TEP

Anomalies métaboliques classiques :

- Hypermétabolisme mésiotemporal : le plus fréquent
- +/- Hypermétabolisme : striatal et/ou thalamique
- Cortex : hypo- ou hyper- selon la clinique et le délai de réalisation (inflammation initial vs déficit secondaire).

Analyse semi-quantitative > visuelle seule.



Semi-quantitative FDG-PET Analysis Increases the Sensitivity Compared With Visual Analysis in the Diagnosis of Autoimmune Encephalitis

Rui-Juan Lv[1], Jian Pan[2], Guifei Zhou[2], Qun Wang[1], Xiao-Qiu Shao[1], Xiao-Bin Zhao[3*] and Jiangang Liu[2,4*]

Intérêt du ratio Cortex/Striatum

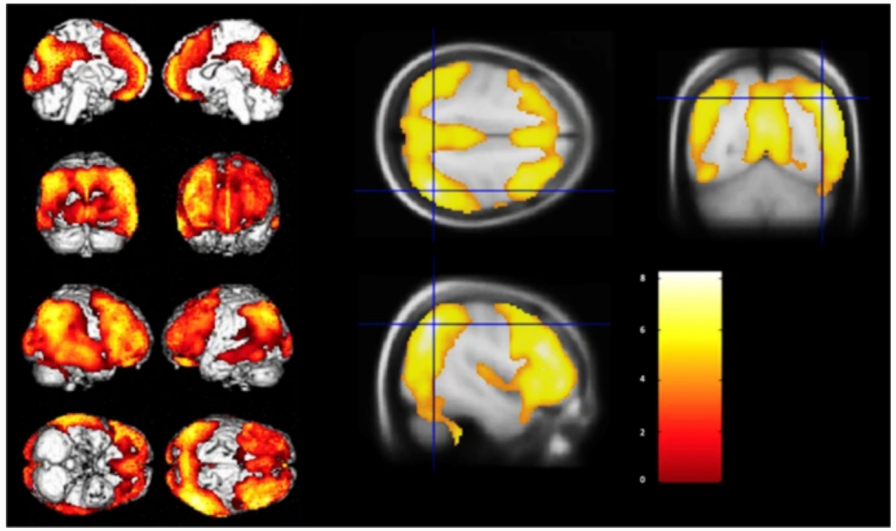
- Atteinte métabolique globale = difficulté de normalisation.

- Seuil < 1.23 permet :

Se	71%
Sp versus MCI	82%
Sp versus contrôles sains	98%

- *Potentiel pronostique et théranostique.*

Fig. 1



Voxel-based analysis of brain [^{18}F]-FDG PET/CT following parametrization to the striatum. Widespread decrease of cortex/striatum metabolic ratio in patients with AE, in comparison to healthy subjects (p-voxel < 0.05, FWE corrected)

Decrease in the cortex/striatum metabolic ratio on [^{18}F]-FDG PET: a biomarker of autoimmune encephalitis

Nicolas De Leiris, Berangère Ruel, Jean Vervandier, José Boucraut, Stephan Grimaldi, Tatiana Horowitz, Jean Pelletier, Frederique Fluchere, Jacques-Yves Campion, Alzheimer's Disease Neuroimaging Initiative, Elsa Kaphan & Eric Guedj

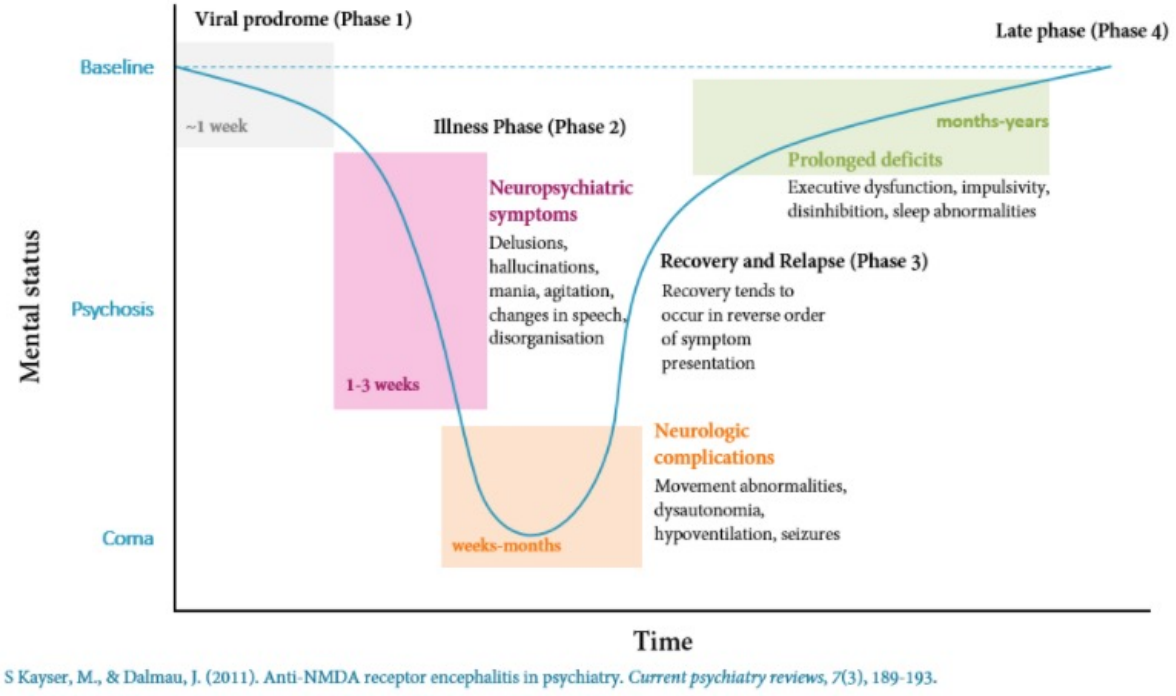
Sémiologie anticorps-spécifique

Encéphalite à anti-NMDAR

Femmes à tout âge
Etiologie : **tératomes ovariens (98%)**>>> …
Récepteur NMDA : Ag de surface cellulaire

Clinique :

- Prodromes (70%) pseudo-viraux
- Phase psychotique : *mnésiques, hallucinations…*
- Phase mutique : *troubles phasiques*
- Phase d'état : *clonies **orofaciales**, dysautonomie, épilepsie, hypoventilation, tr de conscience*



S Kayser, M., & Dalmau, J. (2011). Anti-NMDA receptor encephalitis in psychiatry. *Current psychiatry reviews*, *7*(3), 189-193.

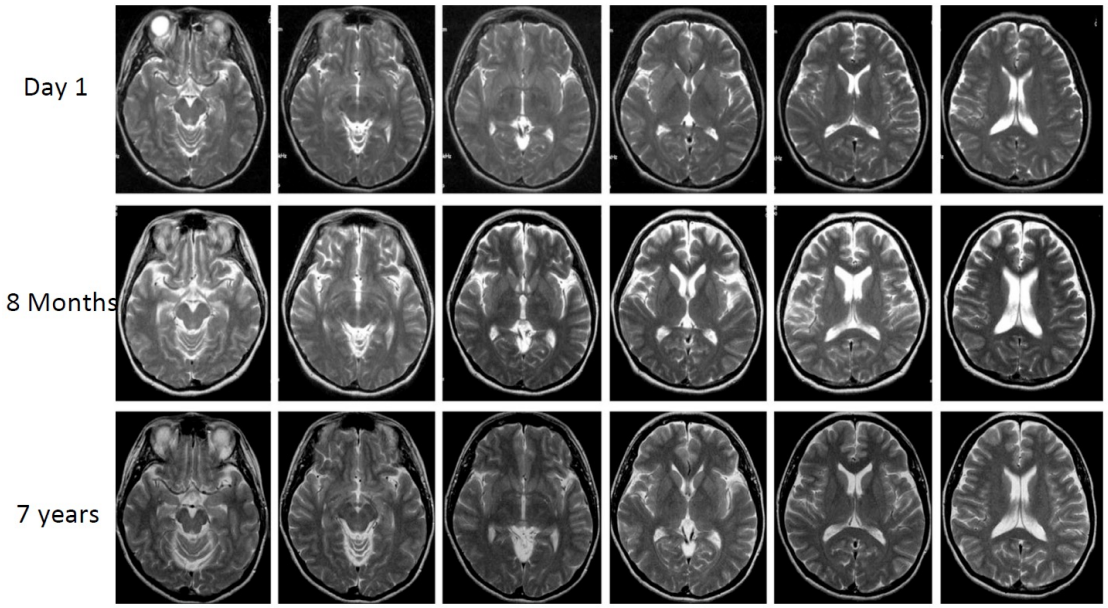
Phase of illness in anti-NMDA receptor encephalitis

Encéphalite à anti-NMDAR

Paraclinique :

- IRM : 50% des cas normale ; anomalies peu spécifiques
- PL : bonne Se mais résultats > 10 jours
- EEG : toujours pathologique ; peu spécifique

Encephalitis with NMDAR-Ab. Reversible cerebral atrophy
Patient treated after 6 months of the disease.
Improvement more than 1 year after the onset.



Iizuka et al, J Neurol 2010

Decreased occipital lobe metabolism by FDG-PET/CT

An anti–NMDA receptor encephalitis biomarker

John C. Probasco, MD
Lilja Solnes, MD
Abhinav Nalluri, BS
Jesse Cohen, MD
Krystyna M. Jones, MD
Elcin Zan, MD
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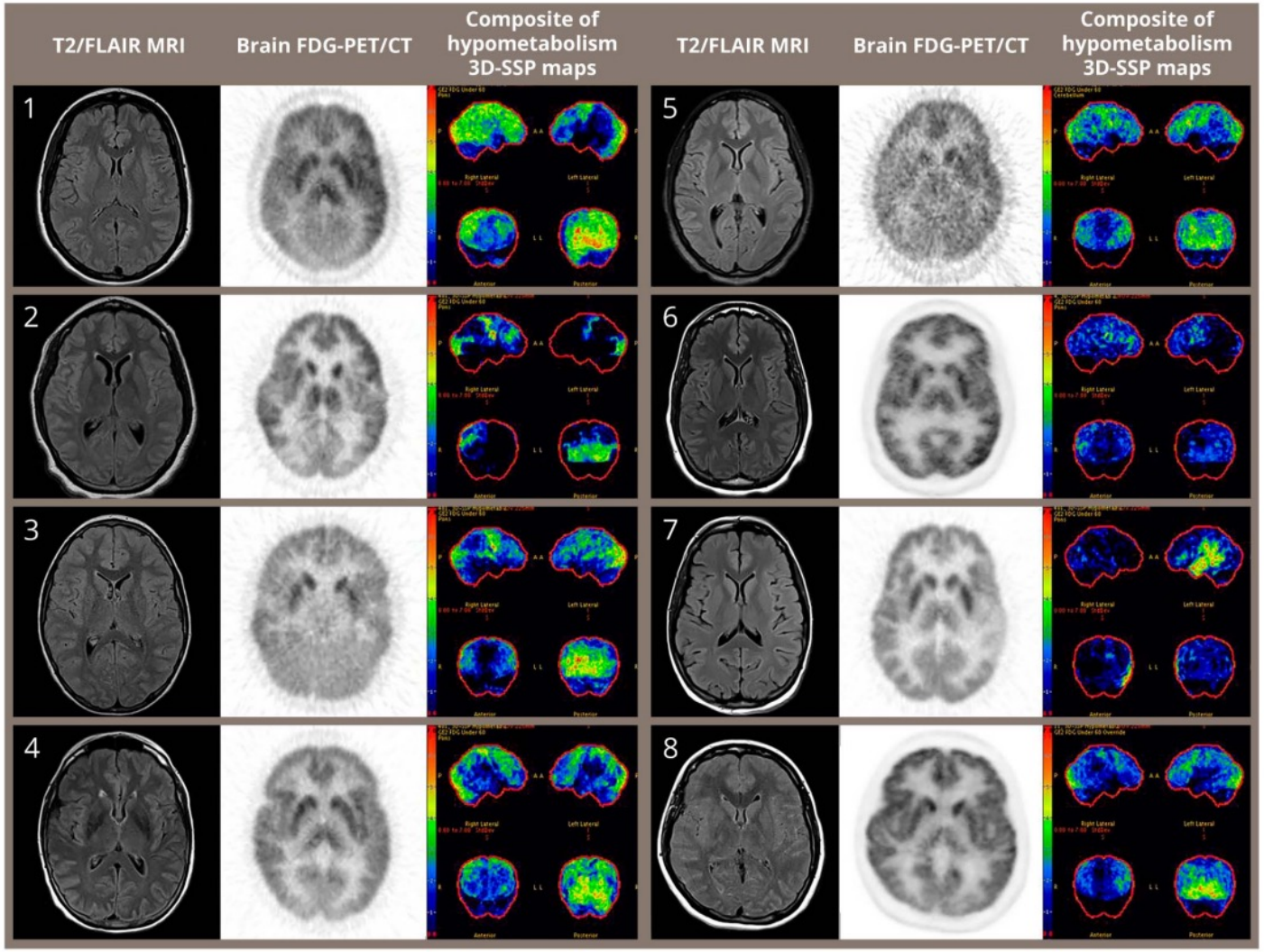

Initial brain fluorodeoxyglucose (FDG)-PET/CT of 8 patients with anti-NMDA receptor, studied within 12 weeks of symptoms. Composite of hypometabolism

TEP : anti-NMDAR

Gradient antéropostérieur :

- Hypométabolisme occipital
  - Médial > latéral
  - Profondeur selon sévérité
  - Dès phase prodromale
  - Disparait avec guérison
- Hypermétabolisme fronto-temporal

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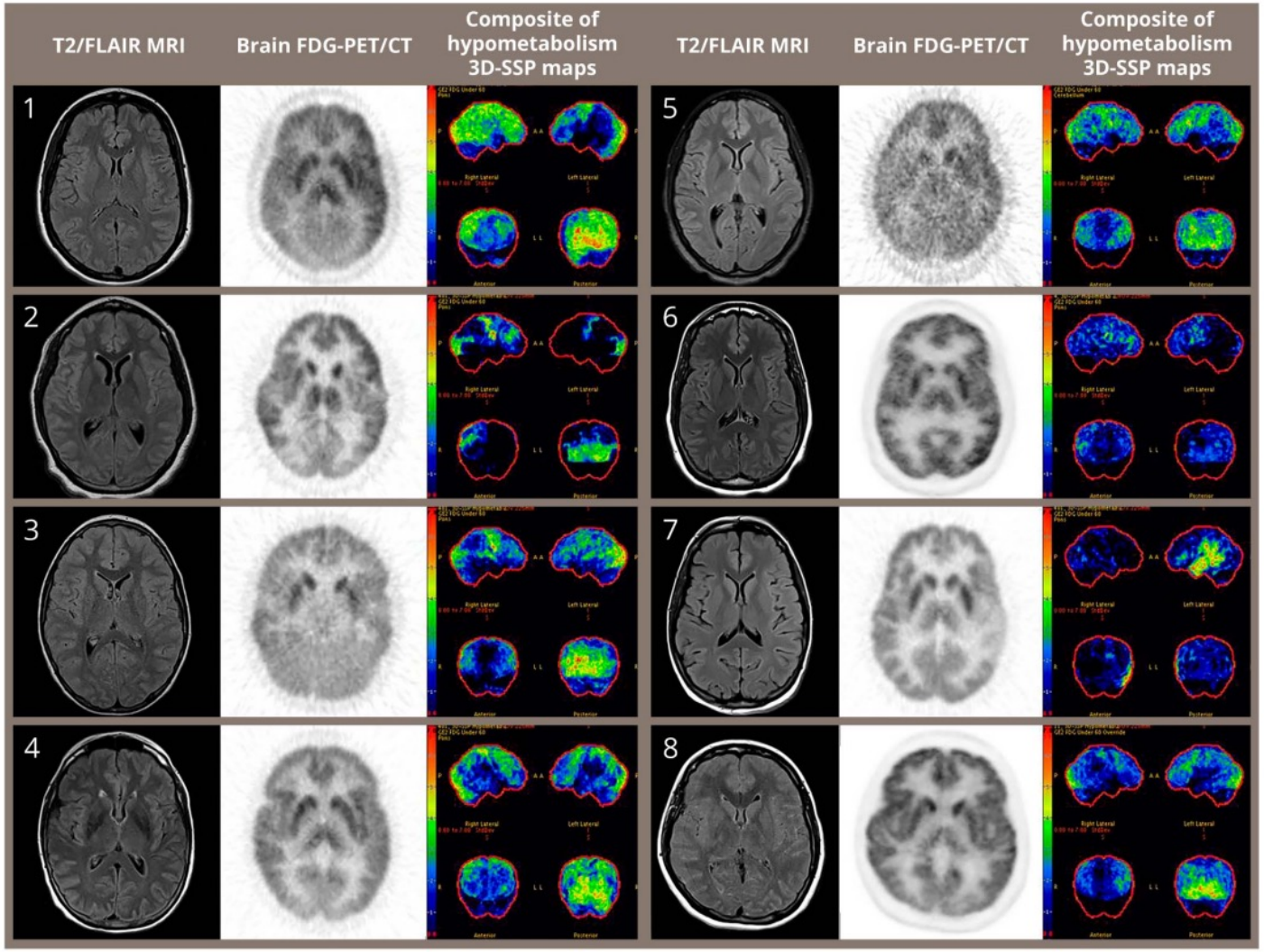


Initial brain fluorodeoxyglucose (FDG)-PET/CT of 8 patients with anti-NMDA receptor, studied within 12 weeks of symptoms. Composite of hypometabolism

Pattern : anti-NMDAR

Gradient antéropostérieur :

- Hypométabolisme occipital
- Hypermétabolisme fronto-temporal

ET/OU

Pattern aspécifique *:*

- *Hypermétabolisme des NGC*
- *Hypométabolisme cortical diffuse*

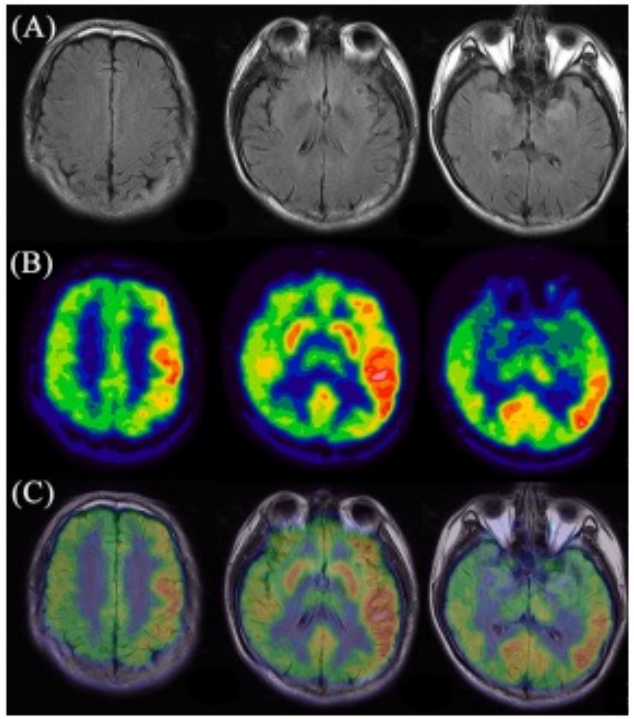
Distinct cerebral ^{18}F-FDG PET metabolic patterns in anti-N-methyl-D-aspartate receptor encephalitis patients with different trigger factors

Jingjie Ge*, Bo Deng*, Yihui Guan, Weiqi Bao, Ping Wu, Xiangjun Chen and Chuantao Zuo

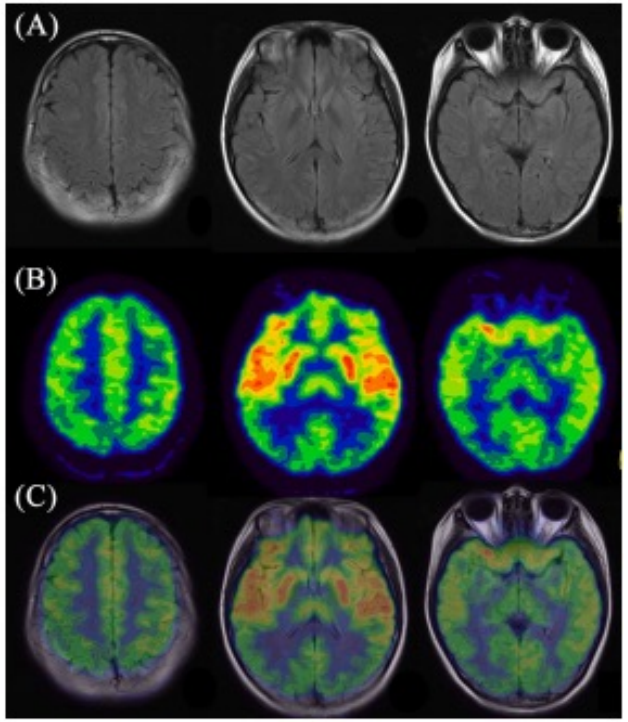
Encéphalite à anti-NMDAR

Pattern selon l'étiologie :

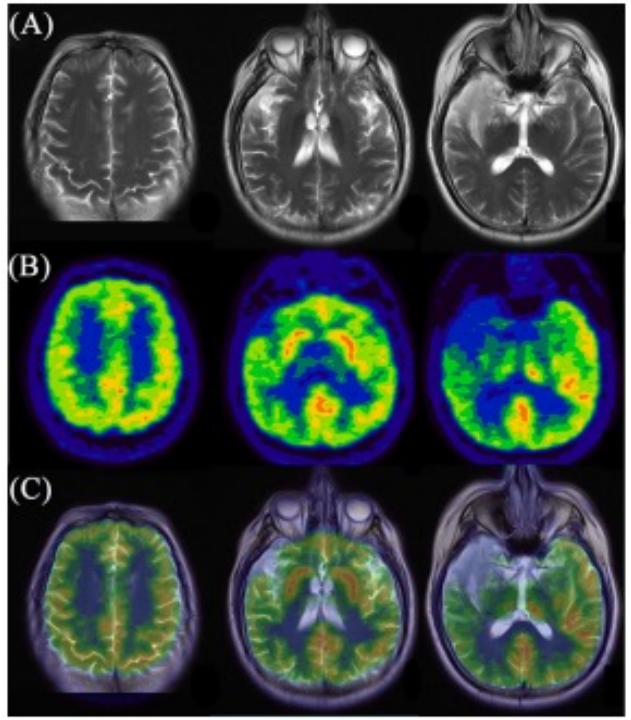
Cryptogénique



Paranéoplasique



Virale



Encéphalite à anti-VGKC

Anti-LGI1

AE récidivante rarement paranéoplasique
Début subaigu
H ≈ 40 ans

Clinique :
- Déficit cognitif progressif
- Crises convulsives
- **Crises dystoniques facio-brachiales**
- Symptômes neuropsychiatriques

Anti-CASPR2

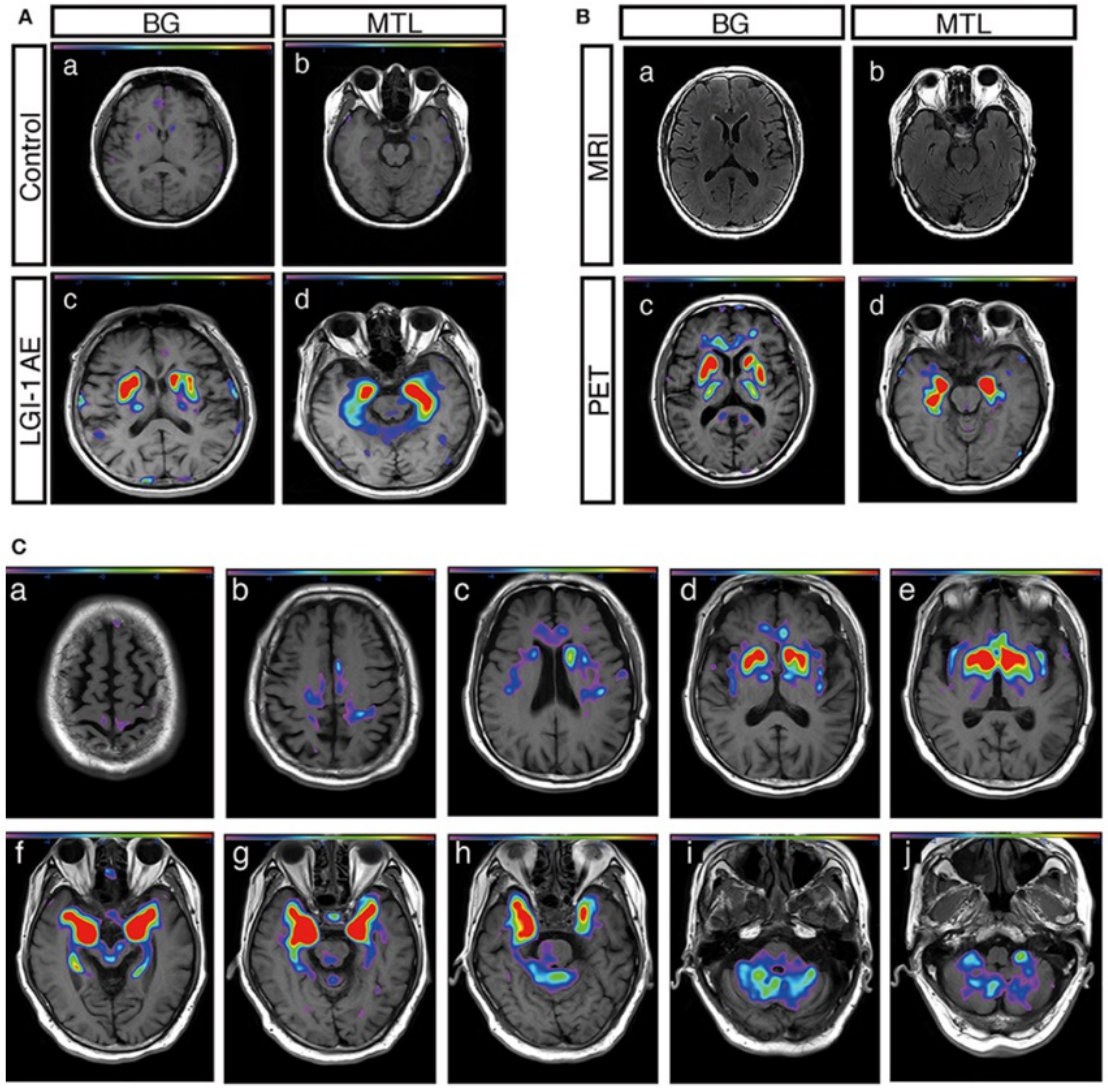
Hommes âgés

Clinique hétérogène :
- *Neuromyotonie*
- *Troubles du sommeil*
- *Troubles de la marche*

Absence de pattern TEP spécifique

The Clinical Value of ^{18}F-FDG-PET in Autoimmune Encephalitis Associated With LGI1 Antibody

Xiao Liu[1†], Wei Shan[1,2,3†], Xiaobin Zhao[3,4], Jiechuan Ren[1,3], Guoping Ren[1,3], Chao Chen[1,3], Weixiong Shi[1,3], Ruijuan Lv[1,3], Zhimei Li[1,3], Yaou Liu[3,5], Lin Ai[3,4*] and Qun Wang[1,2,3*]



Encéphalite à anti-LGI1

Hypermétabolisme temporal interne et striatal

- Peu spécifique (*NDMAR, CASPR2*)

Hypermétabolisme striatal *sans MTL* : spécifique des CDFB+

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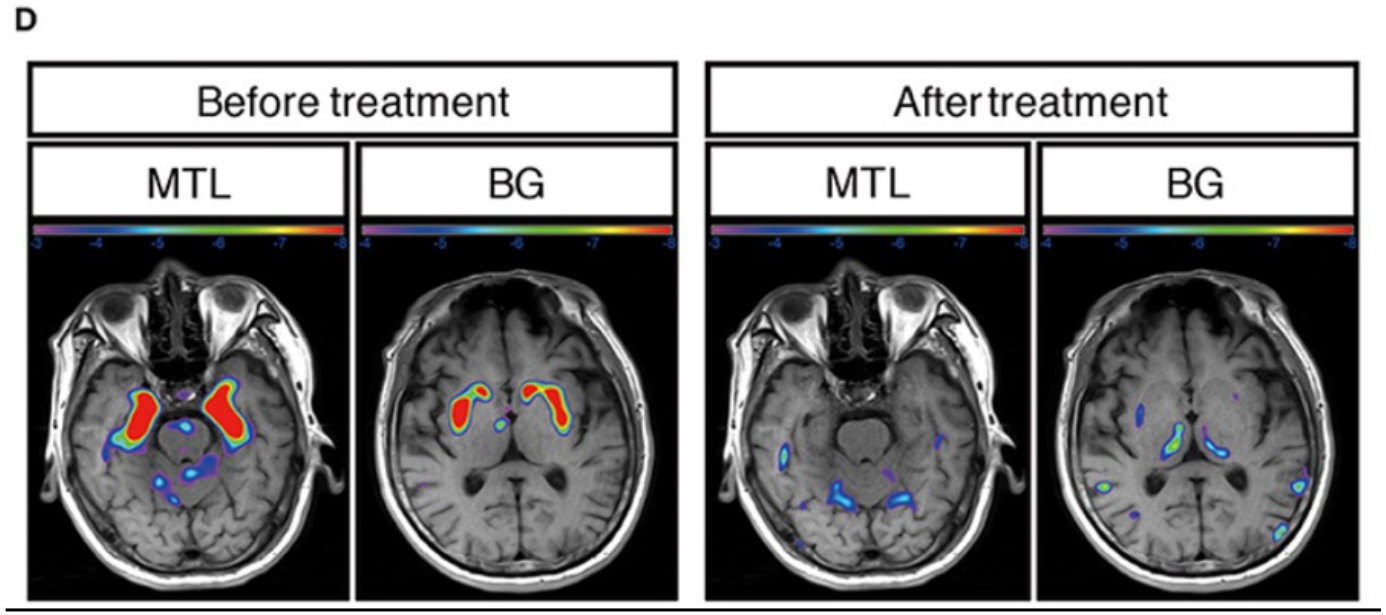


Encéphalite à anti-LGI1

Hypermétabolisme MTL + NGC

ou

Hypermétabolisme striatal isolé

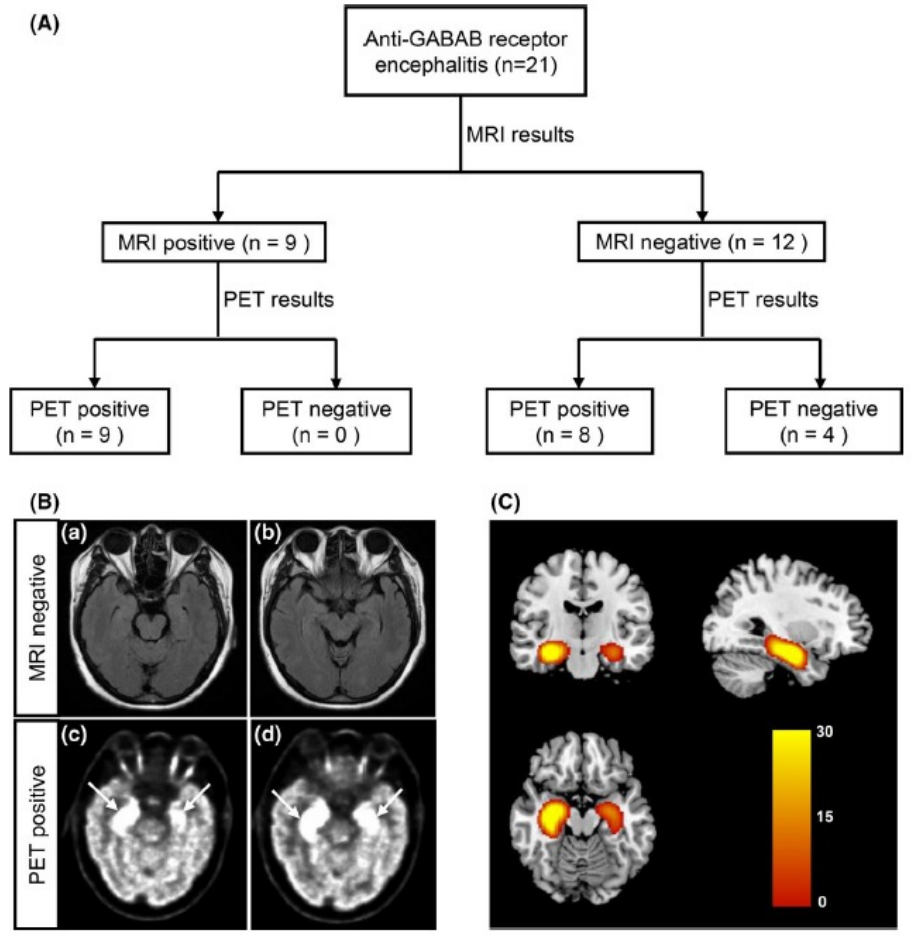
➔ Anomalies régressives avec la guérison

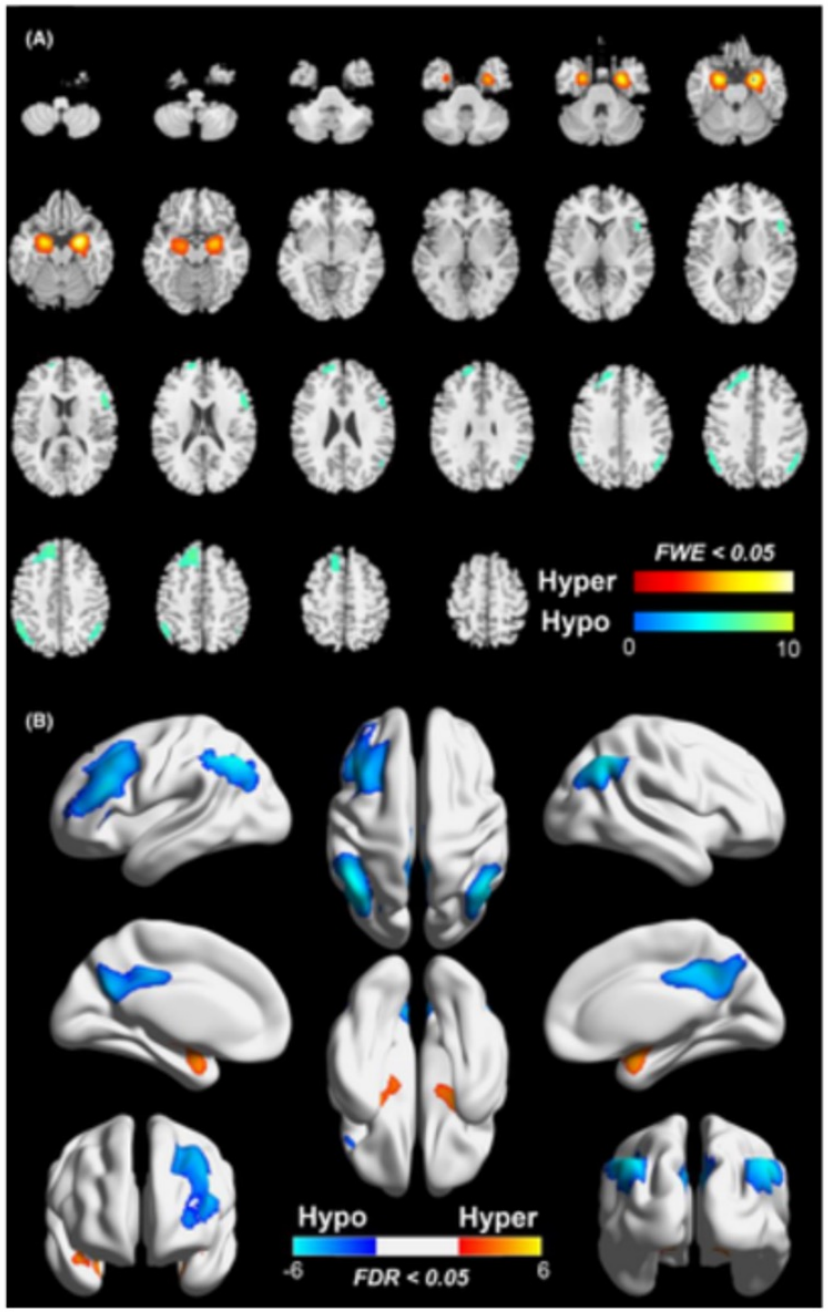
Encéphalite à anti-GABAB

50% paranéoplasique (**CBPC** >>>…)

Clinique :

- Crises convulsives réfractaires
- Déficit cognitif
- Trouble psychiatrique (dépression, confusion, mutisme)





^{18}F-fluorodeoxy-glucose positron emission tomography pattern and prognostic predictors in patients with anti-GABAB receptor encephalitis

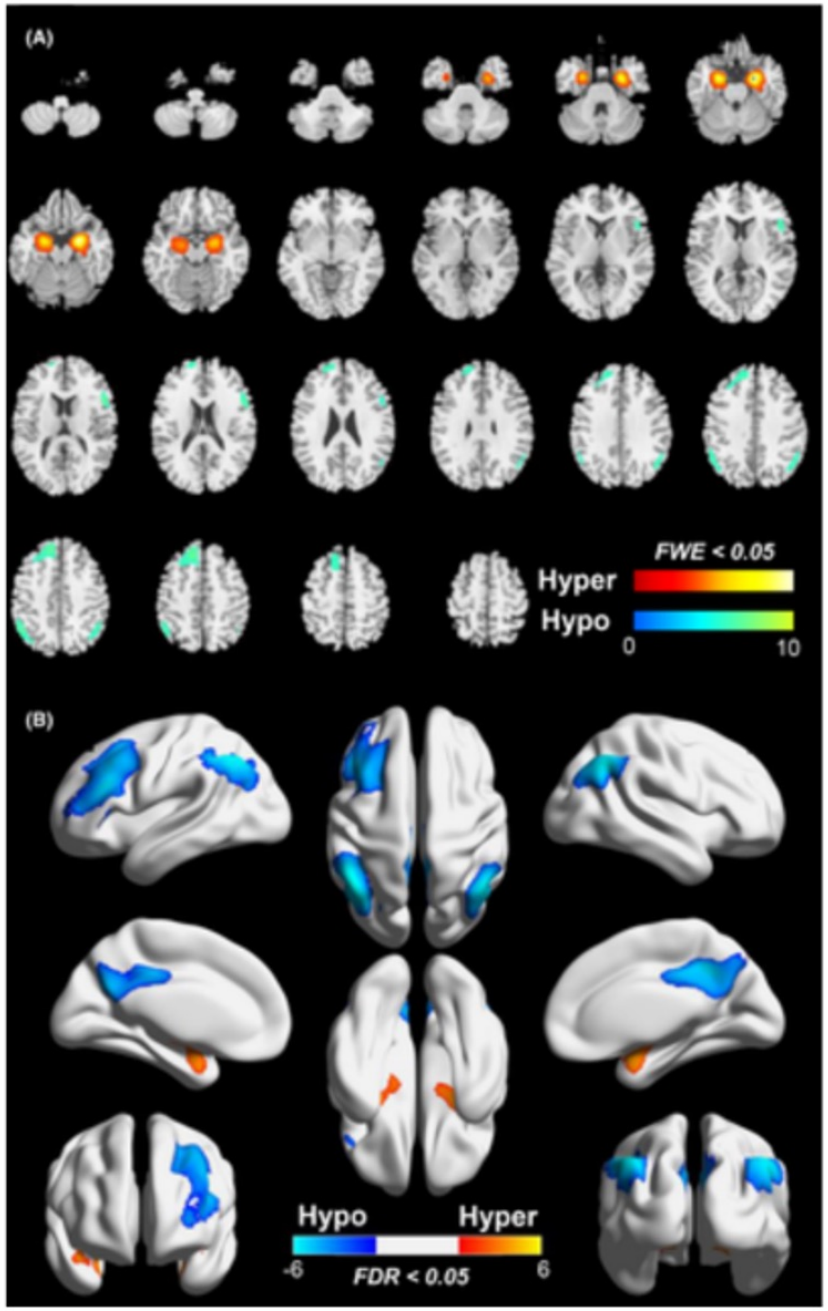
Xiao Liu, Tingting Yu, Xiaobin Zhao, Gongfei Li, Ruijuan Lv, Lin Ai, Qun Wang

Encéphalite à anti-GABAB

Pattern :

- Hypermétabolisme MTL
- Hypométabolisme fronto-parietal
- +/- Hypométabolisme gyrus cingulaire
- **Epargne striatale**

Valeur pronostique *(à 33 mois)*



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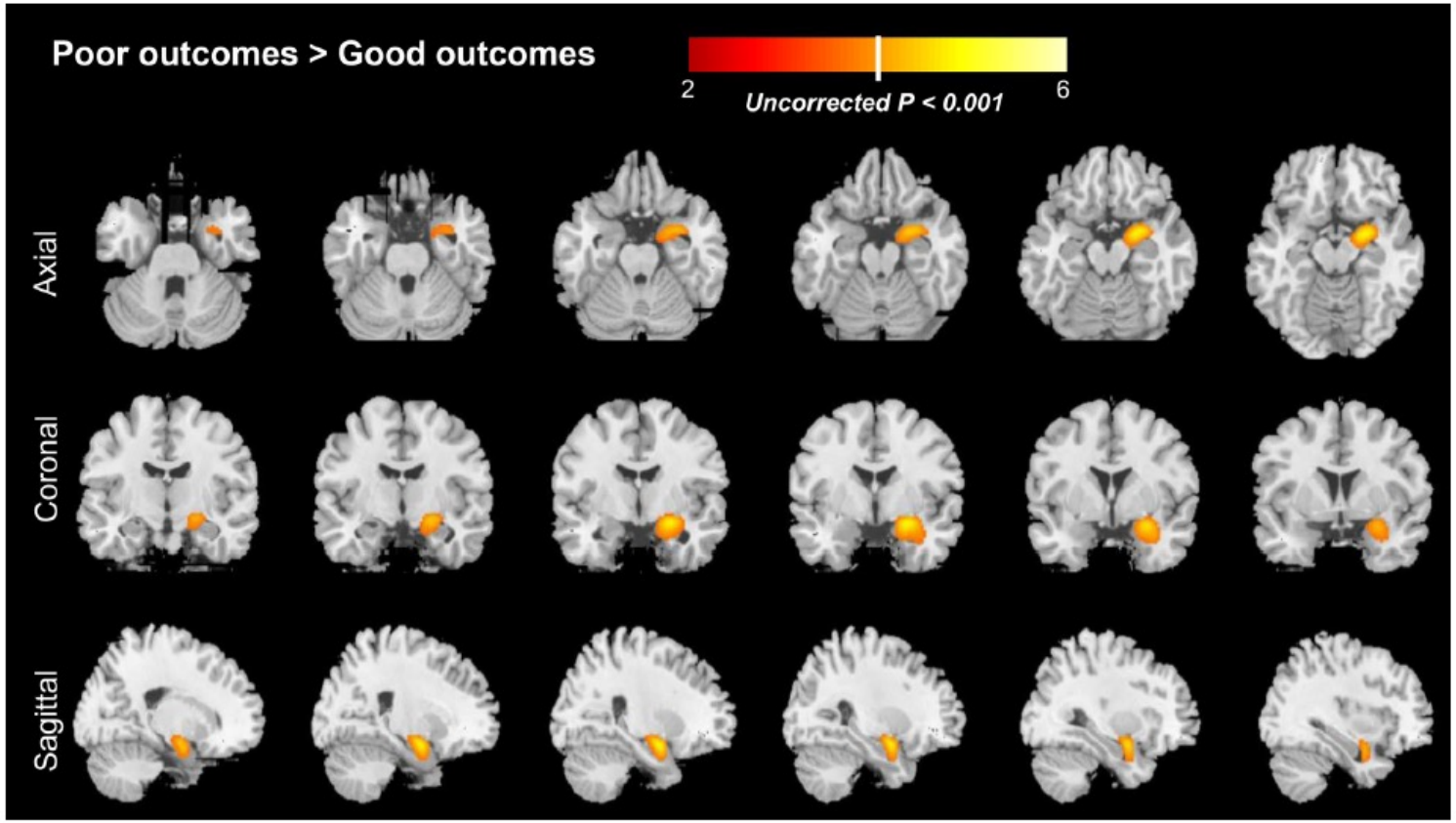
Xiao Liu, Tingting Yu, Xiaobin Zhao, Gongfei Li, Ruijuan Lv, Lin Ai, Qun Wang

Encéphalite à anti-GABAB



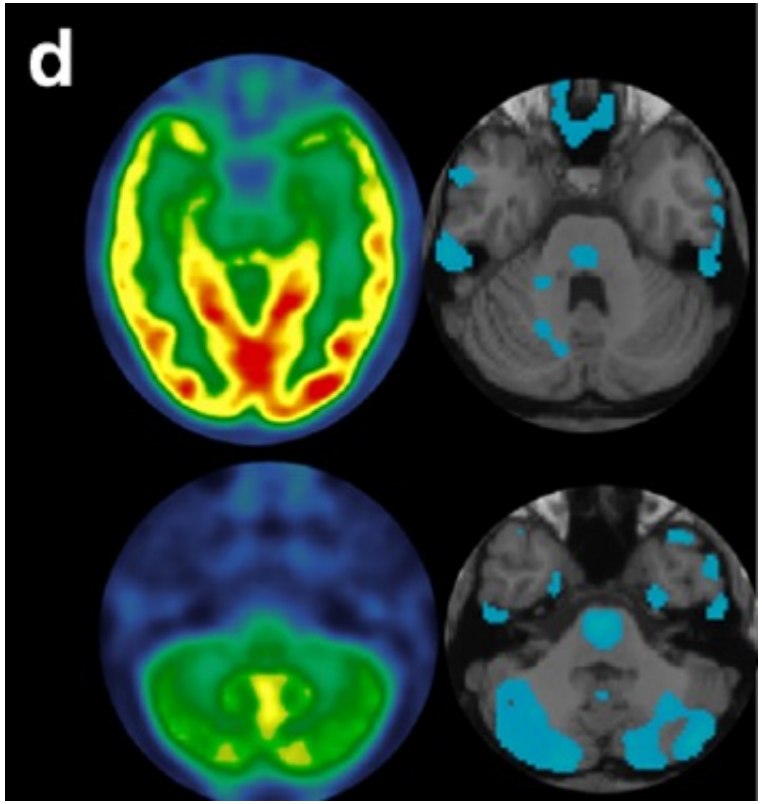
Autres encéphalites dysimmunitaires

Encéphalite anti-GAD (d)

- Cible : Ag synaptiques intra-cellulaires

- Clinique : « *stiff person syndrom* »
  - Ataxie cérébelleuse
  - Sd akinéto-rigide
  - Crise comitiales réfractaires (temporales)

- TEP : **hypo**métabolisme MTL (si tardif)

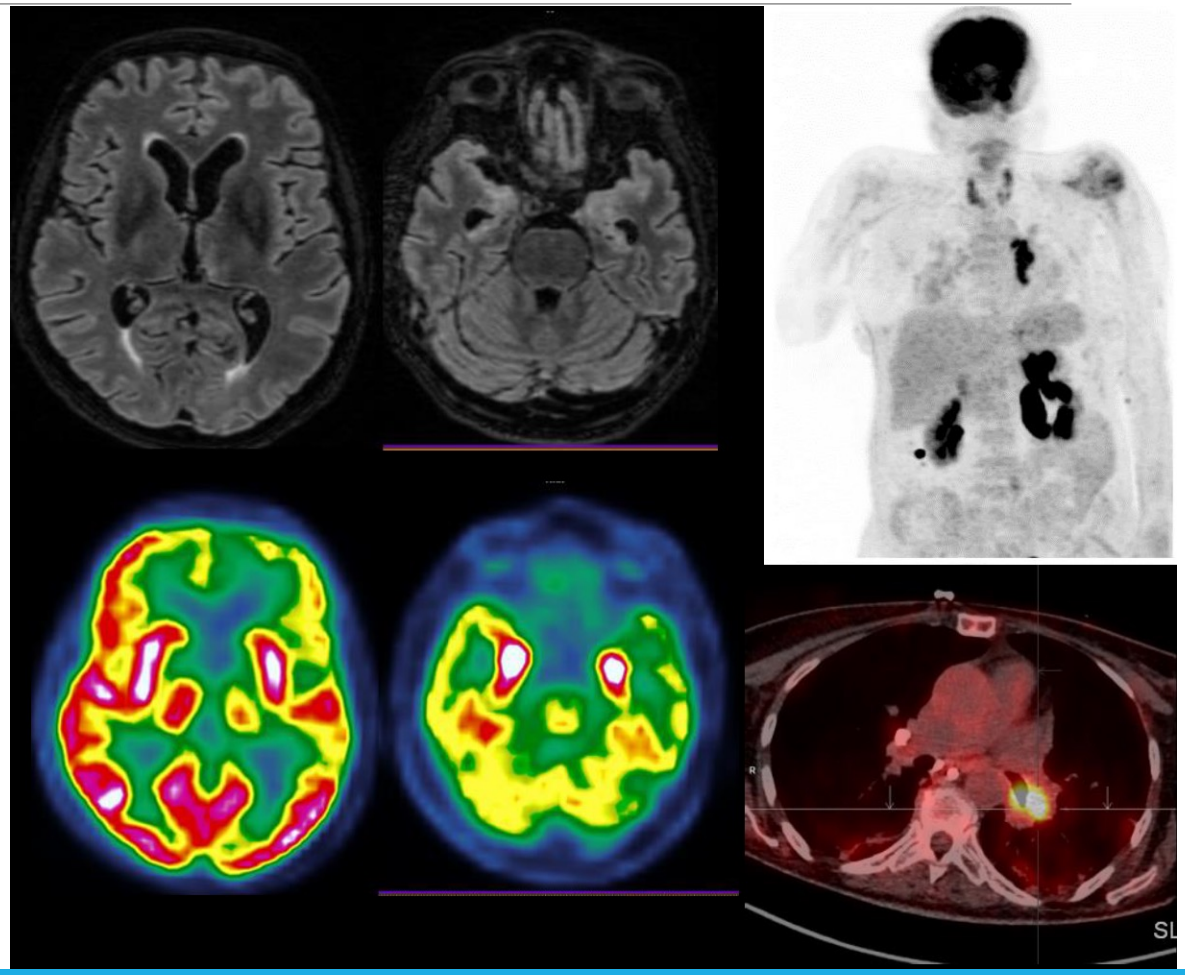


Autres encéphalites dysimmunitaires

Anticorps onco-neuronaux :

Anticorps anti-Hu :

- Troubles moteurs
- Troubles neuropsychiatriques
- Crises convulsives
- +/- Atteinte cérébelleuse



Autres encéphalites dysimmunitaires

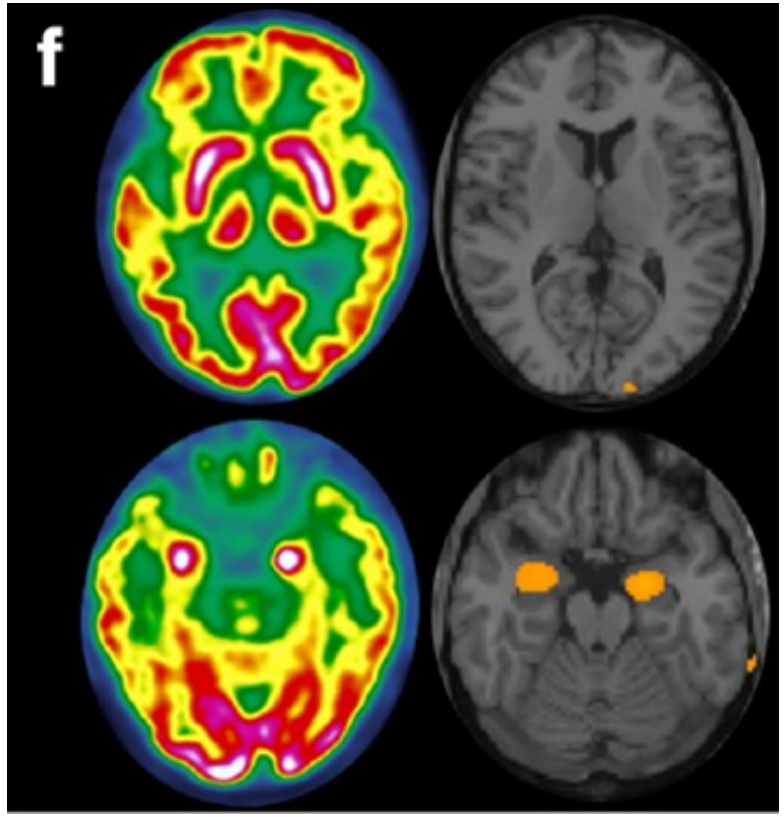
Anticorps onco-neuronaux :

Anticorps anti-Hu :

- Hypométabolisme cortical diffus
- ± Hypermétabolisme temporal interne

Anticorps anti-Ma :

- hypométabolisme temporal interne



Encéphalite de Rasmussen

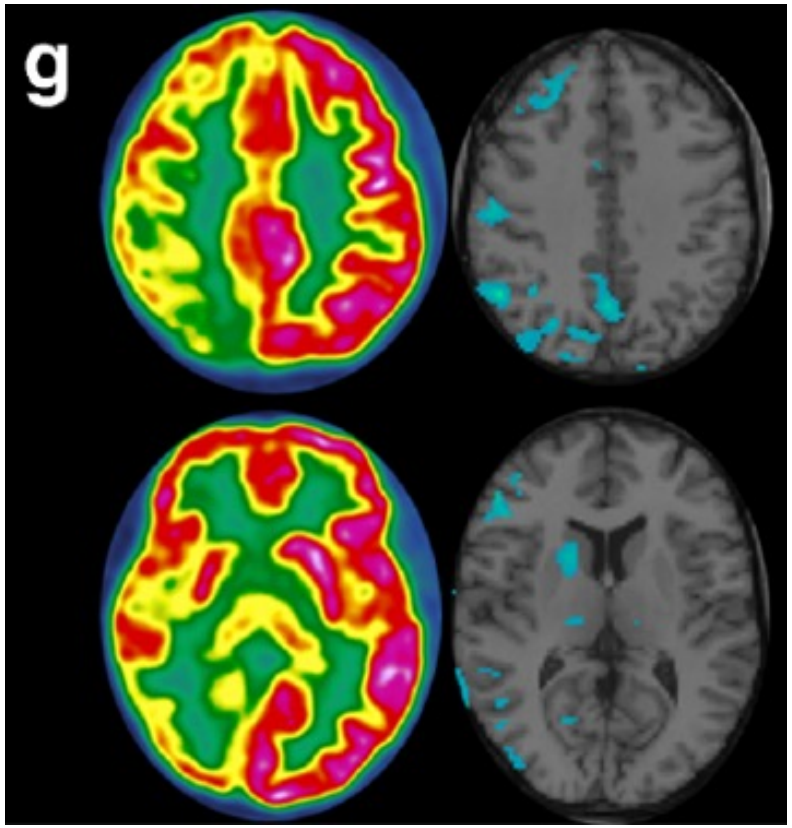
Origine inconnue, auto-immune suspectée

Début dans l'enfance

Evolution vers épilepsie progressive avec encéphalite unilatérale chronique

TEP :

- Hypométabolisme hémisphérique
- Métabolique > morphologique

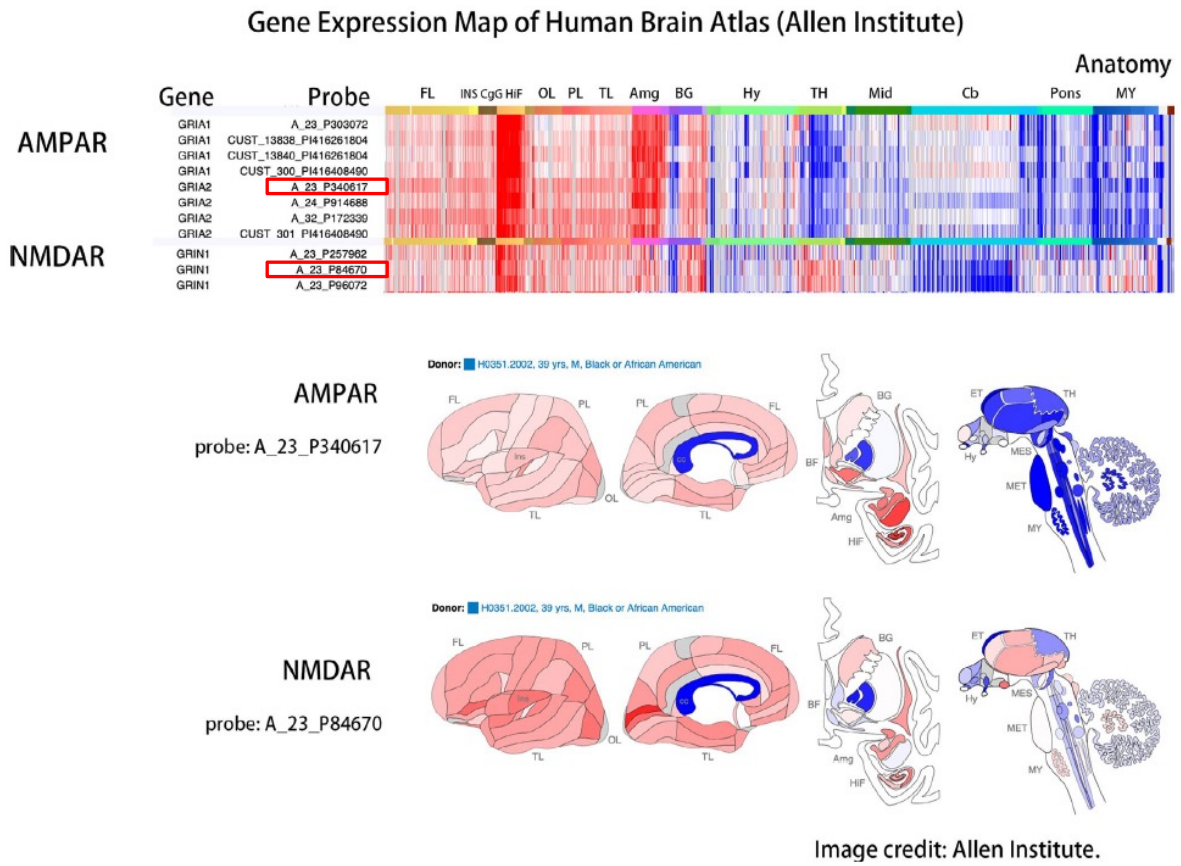


Perspectives

- Nouveaux traceurs : cibles antigéniques
- Pattern semi-quantitatifs
- Apports de l'IA

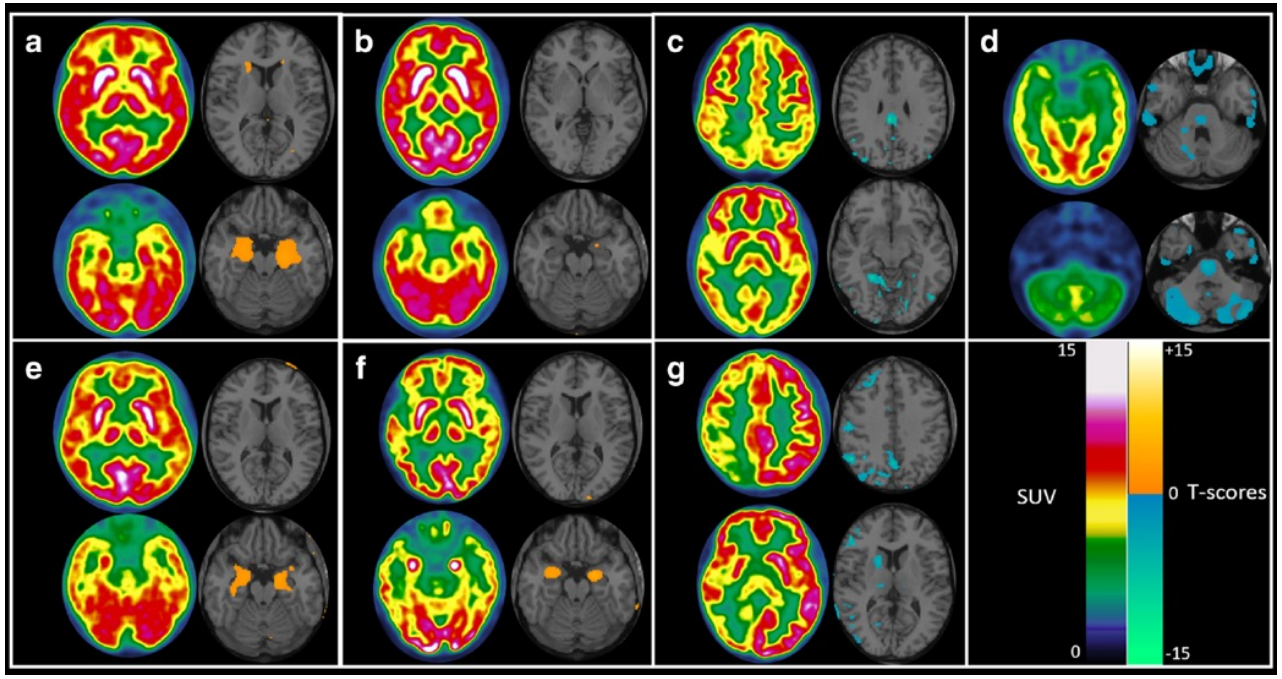
[^{11}C]-DPA-713 and [^{18}F]-DPA-714 as New PET Tracers for TSPO: A Comparison with [^{11}C]-(*R*)-PK11195 in a Rat Model of Herpes Encephalitis

Janine Doorduin,[1] Hans C. Klein,[1,2] Rudi A. Dierckx,[1] Michelle James,[3] Michael Kassiou,[3,4,5] Erik F. J. de Vries[1]



Conclusion

- Performances diagnostiques de la TEP
- Place croissante dans la **stratégie diagnostique :** positif et étiologique
- Importance de l'**analyse semi-quantitative** (ratio C/S)
- Identification de **patterns spécifiques** des cibles antigéniques
- Intérêt dans le **pronostic**



Brain ^{18}F-FDG PET for the diagnosis of autoimmune encephalitis: a systematic review and a meta-analysis

Manon Bordonne[1] · Mohammad B. Chawki[1] · Matthieu Doyen[1,2] · Aurelie Kas[3] · Eric Guedj[4] · Louise Tyvaert[5] · Antoine Verger[1,2]

A clinical approach to diagnosis of autoimmune encephalitis

Prof. Francesc Graus, MD, Maarten J Titulaer, MD, Ramani Balu, MD, Susanne Benseler, MD, Prof. Christian G Bien, MD, Tania Cellucci, MD, Irene Cortese, MD, Prof. Russell C Dale, MD, Jeffrey M Gelfand, MD, Michael Geschwind, MD, Carol A Glaser, MD, Prof. Jerome Honnorat, MD, Romana Höftberger, MD, Takahiro Iizuka, MD, Sarosh R Irani, MD, Eric Lancaster, MD, Frank Leypoldt, MD, Harald Prüss, MD, Alexander Rae-Grant, MD, Prof. Markus Reindl, PhD, Prof. Myrna R Rosenfeld, MD, Kevin Rostásy, MD, Albert Saiz, MD, Arun Venkatesan, MD, Prof. Angela Vincent, FRS, Prof. Klaus-Peter Wandinger, Patrick Waters, PhD, and Prof. Josep Dalmau, MD

Usefulness of brain FDG PET/CT imaging in pediatric patients with suspected autoimmune encephalitis from a prospective study

Yafu Yin[1] · Jing Wu[2] · Shuqi Wu[1] · Suyun Chen[1] · Weiwei Cheng[1] · Ling Li[2] · Hui Wang[1]

^{18}F-fluorodeoxy-glucose positron emission tomography pattern and prognostic predictors in patients with anti-GABAB receptor encephalitis

Xiao Liu, Tingting Yu, Xiaobin Zhao, Gongfei Li, Ruijuan Lv, Lin Ai, Qun Wang

Semi-quantitative FDG-PET Analysis Increases the Sensitivity Compared With Visual Analysis in the Diagnosis of Autoimmune Encephalitis

Rui-Juan Lv, Jian Pan, Guifei Zhou, Qun Wang, Xiao-Qiu Shao, Xiao-Bin Zhao and Jiangang Liu

Merci de votre attention !

Decreased occipital lobe metabolism by FDG-PET/CT

An anti–NMDA receptor encephalitis biomarker

John C. Probasco, MD
Lilja Solnes, MD
Abhinav Nalluri, BS
Jesse Cohen, MD
Krystyna M. Jones, MD
Elcin Zan, MD
Mehrbod S. Javadi, MD
Arun Venkatesan, MD, PhD

The Clinical Value of ^{18}F-FDG-PET in Autoimmune Encephalitis Associated With LGI1 Antibody

Xiao Liu, Wei Shan, Xiaobin Zhao, Jiechuan Ren, Guoping Ren, Chao Chen, Weixiong Shi, Ruijuan Lv, Zhimei Li, Yaou Liu, Lin Ai and Qun Wang

Distinct cerebral ^{18}F-FDG PET metabolic patterns in anti-N-methyl-D-aspartate receptor encephalitis patients with different trigger factors

Jingjie Ge*, Bo Deng*, Yihui Guan, Weiqi Bao, Ping Wu, Xiangjun Chen and Chuantao Zuo

[^{11}C]-DPA-713 and [^{18}F]-DPA-714 as New PET Tracers for TSPO: A Comparison with [^{11}C]-(*R*)-PK11195 in a Rat Model of Herpes Encephalitis

Janine Doorduin,[1] Hans C. Klein,[1,2] Rudi A. Dierckx,[1] Michelle James,[3] Michael Kassiou,[3,4,5] Erik F. J. de Vries[1]

Decrease in the cortex/striatum metabolic ratio on [^{18}F]-FDG PET: a biomarker of autoimmune encephalitis

Nicolas De Leiris, Berangère Ruel, Jean Vervandier, José Boucraut, Stephan Grimaldi, Tatiana Horowitz, Jean Pelletier, Frederique Fluchere, Jacques-Yves Campion, Alzheimer's Disease Neuroimaging Initiative, Elsa Kaphan & Eric Guedj