# Produits de contraste et rein

**Yves Pirson**

**Staff du Service de Radiologie**
**St. Luc, 26.10.2012**

- **Insuffisance rénale aiguë (IRA) induite par les produits de contraste iodés (PCI)**
- **Fibrose systémique néphrogénique (FSN) induite par le gadolinium (Gd)**

- **Insuffisance rénale aiguë (IRA) induite par les produits de contraste iodés (PCI)**

- **Fibrose systémique néphrogénique (FSN) induite par le gadolinium (Gd)**

**Homme de 76 ans**

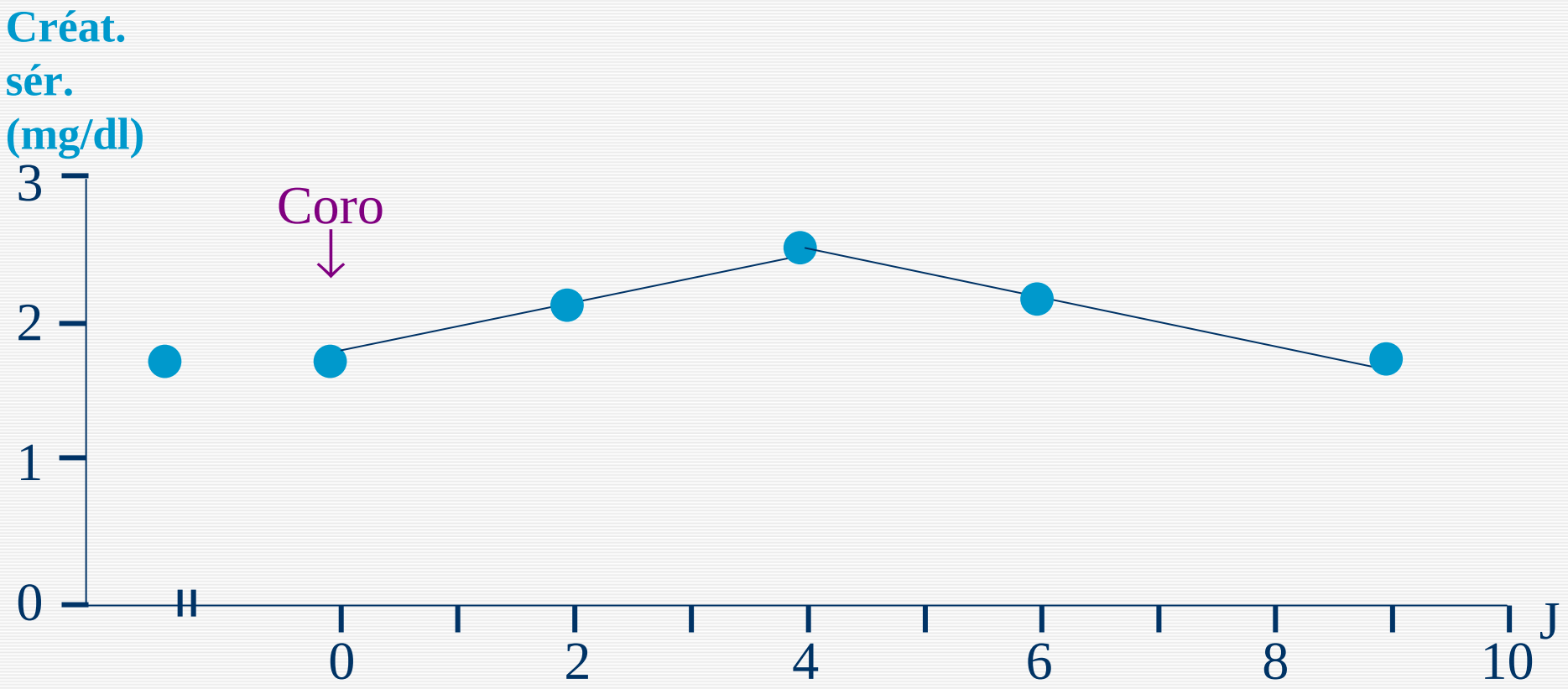
**Diabète de type 2**

**HTA traitée par enalapril 20 mg/j depuis 15 ans**

**Taux de créat. sér. à 1.8 mg/dl (Cockcroft: 34 ml/min), stable depuis 18 mois**

**Apparition d'angor → coronarographie prévue**

# Evolution de la fonction rénale



# Score prédictif d'insuffisance rénale aiguë (IRA) induite par les produits de contraste iodés (PCI)

| *Facteur de risque* | | *Score* |
|---|---|---|
| Age > 75 | | 4 |
| Diabète | | 3 |
| PA systolique < 80 mmHg durant > 1h et requérant support inotrope ou ballon intra-aortique | | 5 |
| Hypoperfusion rénale sévère | | 5 |
| DFG | 40 – 60 | 2 |
| | 20 – 39 | 4 |
| | < 20 ml/min | 6 |
| Volume de PCI | | 1/100 ml |

| *Total du score* | *Risque d'IRA (%)* | *Risque de dialyse (%)* |
|---|---|---|
| 2 – 5 | 7.5 | 0.04 |
| 6 – 10 | 14 | 0.12 |
| 11 – 15 | 26 | 1.09 |
| ≥ 16 | 57 | 12.6 |

*(Mehran R et al., J Am Coll Cardiol 2004; 44: 1393)*

# Prévalence de l'IRC aux USA

**Table 1 Prevalence of chronic kidney disease in the USA[1]**

| | | 1988-1994 | 1999-2004 |
|---|---|---|---|
| Stage 1 | (persistent albuminuria + normal GFR) | 1.7% | 1.8% |
| Stage 2 | (persistent albuminuria + GFR 60-89) | 2.7% | 3.2% |
| Stage 3 | (GFR 30-59) | 5.4% | 7.7% |
| Stage 4 | (GFR 15-29) | 0.2% | 0.4% |
| Stage 5 | (kidney failure) 0.2% | | |
| Total | | 10.0% | 13.1% |

*(Andersen PE, World J Radiol 2012; 4: 253)*

# Persistent Renal Damage After Contrast-Induced Acute Kidney Injury

## Incidence, Evolution, Risk Factors, and Prognosis

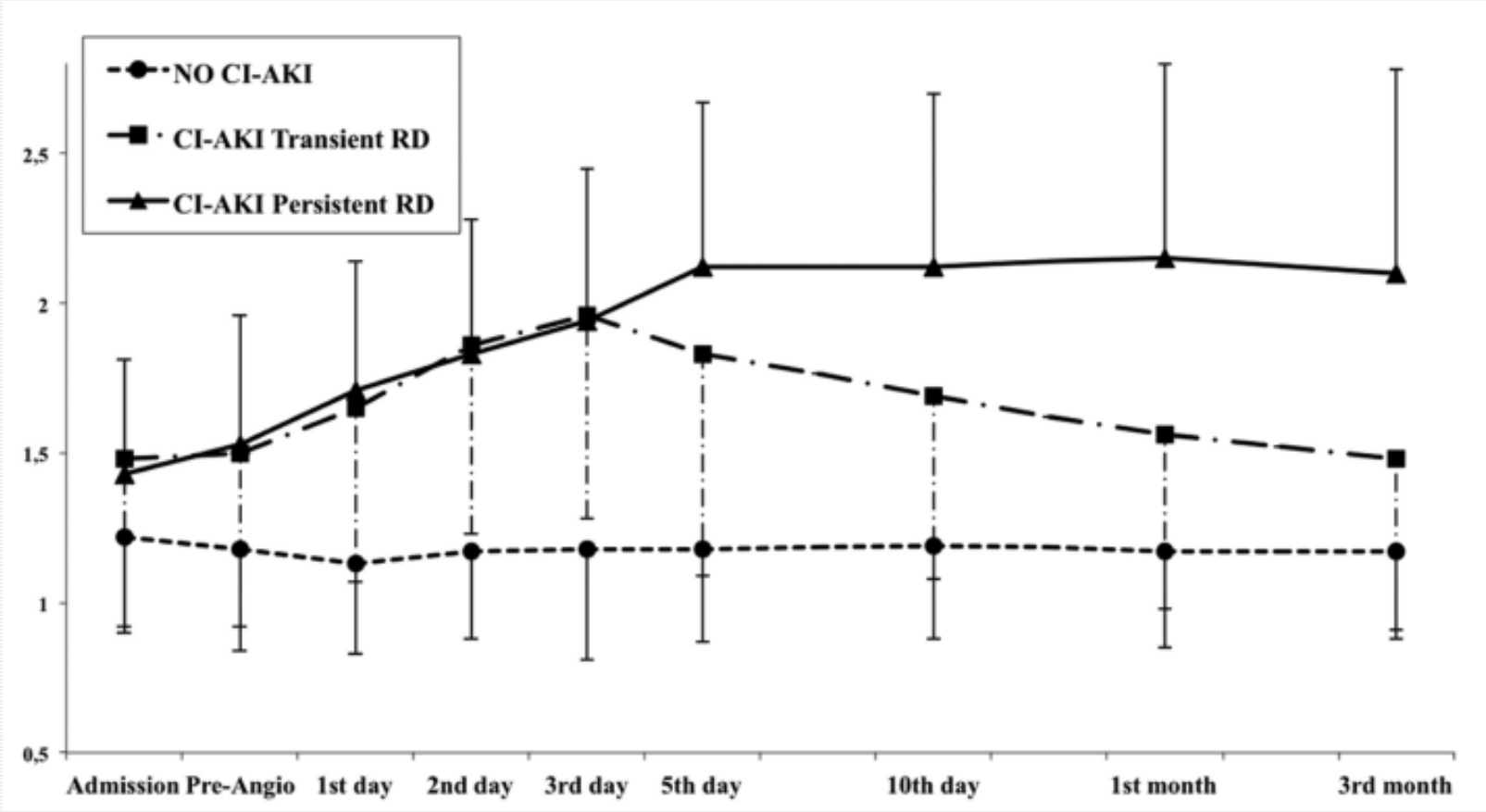
Mauro Maioli, MD; Anna Toso, MD; Mario Leoncini, MD; Michela Gallopin, MD; Nicola Musilli, MD; Francesco Bellandi, MD

***Background***—The temporal evolution of renal function in patients with acute kidney injury after contrast medium (CI-AKI) is not well known. The aim of this observational study was to evaluate the incidence, risk factors, and prognostic implications of persistent renal damage (RD) in patients with preexistent moderate-to-severe renal dysfunction.

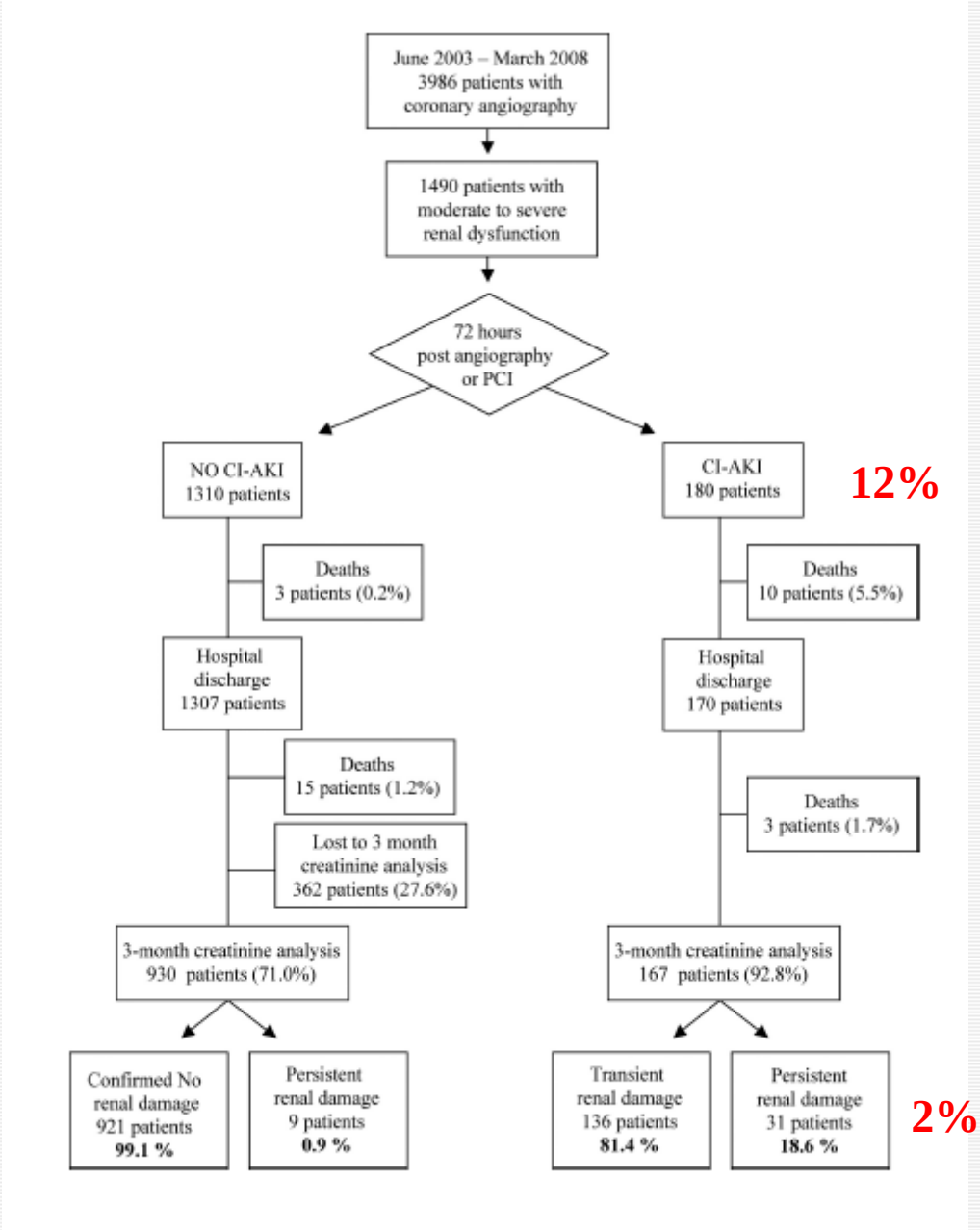
***Methods and Results***—From June 2003 to March 2008, 3986 patients underwent coronary angiography at our institution; 1490 of 3986 had an estimated creatinine clearance of <60 mL/min and were enrolled. CI-AKI was defined as an absolute increase ≥0.5 mg/dL over baseline serum creatinine within 3 days after the administration of contrast medium (iodixanol). In patients who developed CI-AKI, persistent RD was defined as a relative decrease of creatinine clearance ≥25% over baseline at 3 months. Patients whose creatinine clearance returned to baseline (or nearly) were classified as transient RD. The overall incidence of CI-AKI was 12.1%, and persistent RD occurred in 18.6% of CI-AKI patients. At Cox regression analysis, nephropathy risk score ≥17, left ventricular ejection fraction ≤30%, and increased value of serum creatinine ≥1.5-fold from baseline within 5 days were found to be significant risk factors for persistent RD. At 5 years, the incidence of death was significantly higher in patients with persistent RD than in both patients with transient RD ($P$=0.015) and those without CI-AKI ($P$=0.0001). A similar trend was observed for the combined end point of death, dialysis and cardiovascular events.

***Conclusions***—These results suggest that CI-AKI is not always a transient, benign creatininopathy, but rather a direct cause of worsening renal function. The occurrence of CI-AKI can identify patients at increased risk of cardiovascular events. (***Circulation*. 2012;125:3099-3107.**)

***(Maioli M, Circulation 2012; 125: 3099)***

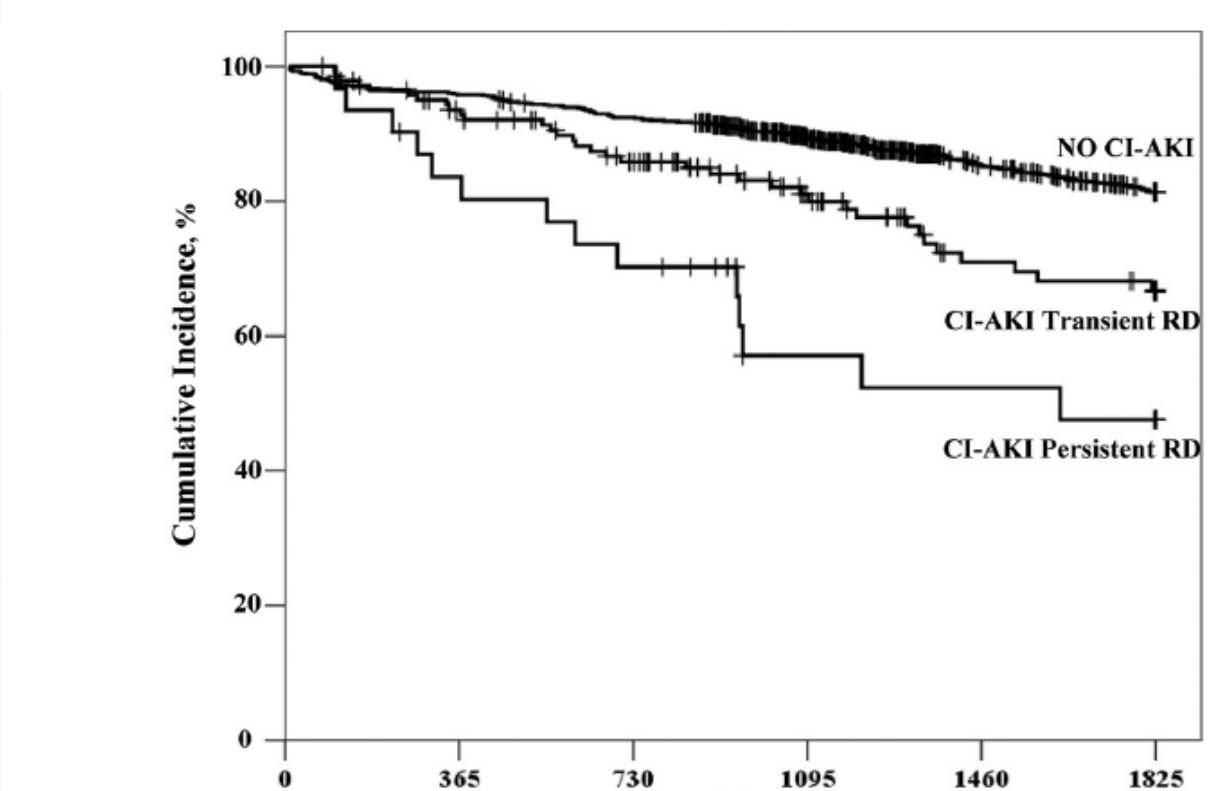



*(Maioli M, Circulation 2012;125:3099)*




*(Maioli M, Circulation 2012; 125: 3099)*

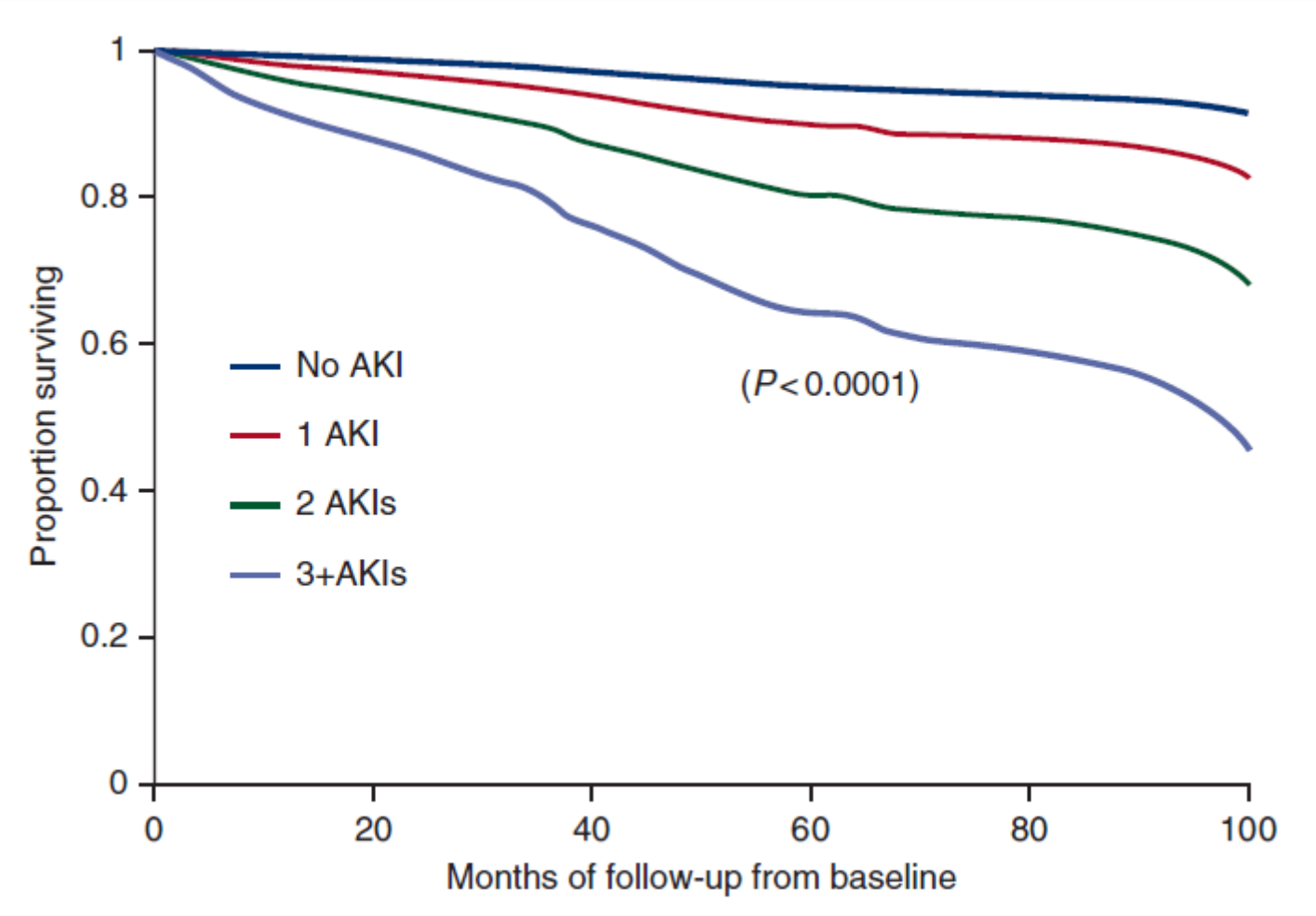
# Survival at 5 years, according to a previous CI-AKI



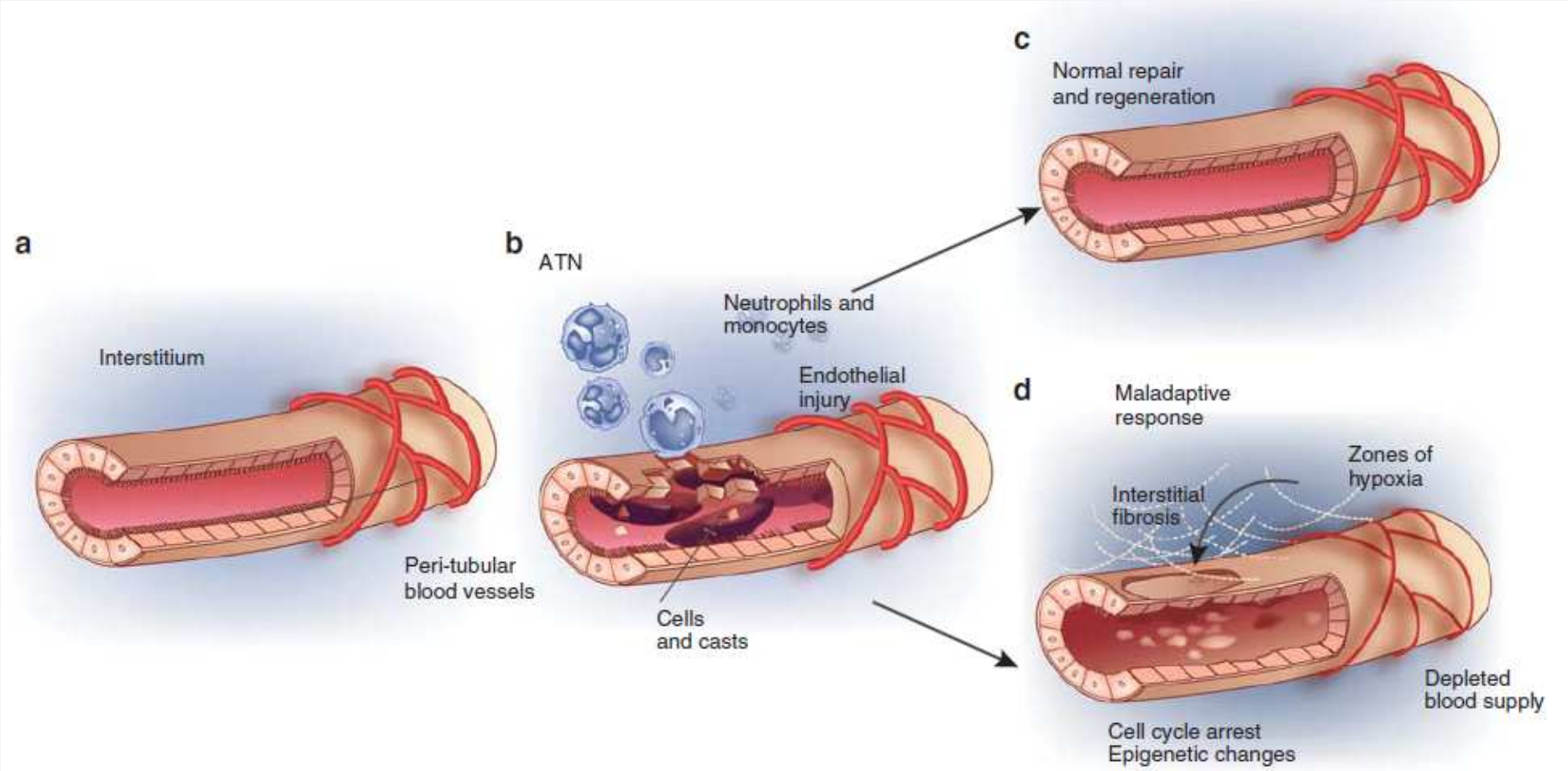

| No. at risk | | | | | | |
|---|---|---|---|---|---|---|
| NO CI-AKI | 1085 | 928 | 892 | 745 | 531 | 448 |
| CI-AKI Transient RD | 136 | 125 | 106 | 75 | 51 | 45 |
| CI-AKI Persistent RD | 31 | 26 | 22 | 12 | 11 | 10 |

*(Maioli M, Circulation 2012; 125: 3099)*

# Effect of acute kidney injury frequency on survival to stage 4 CKD



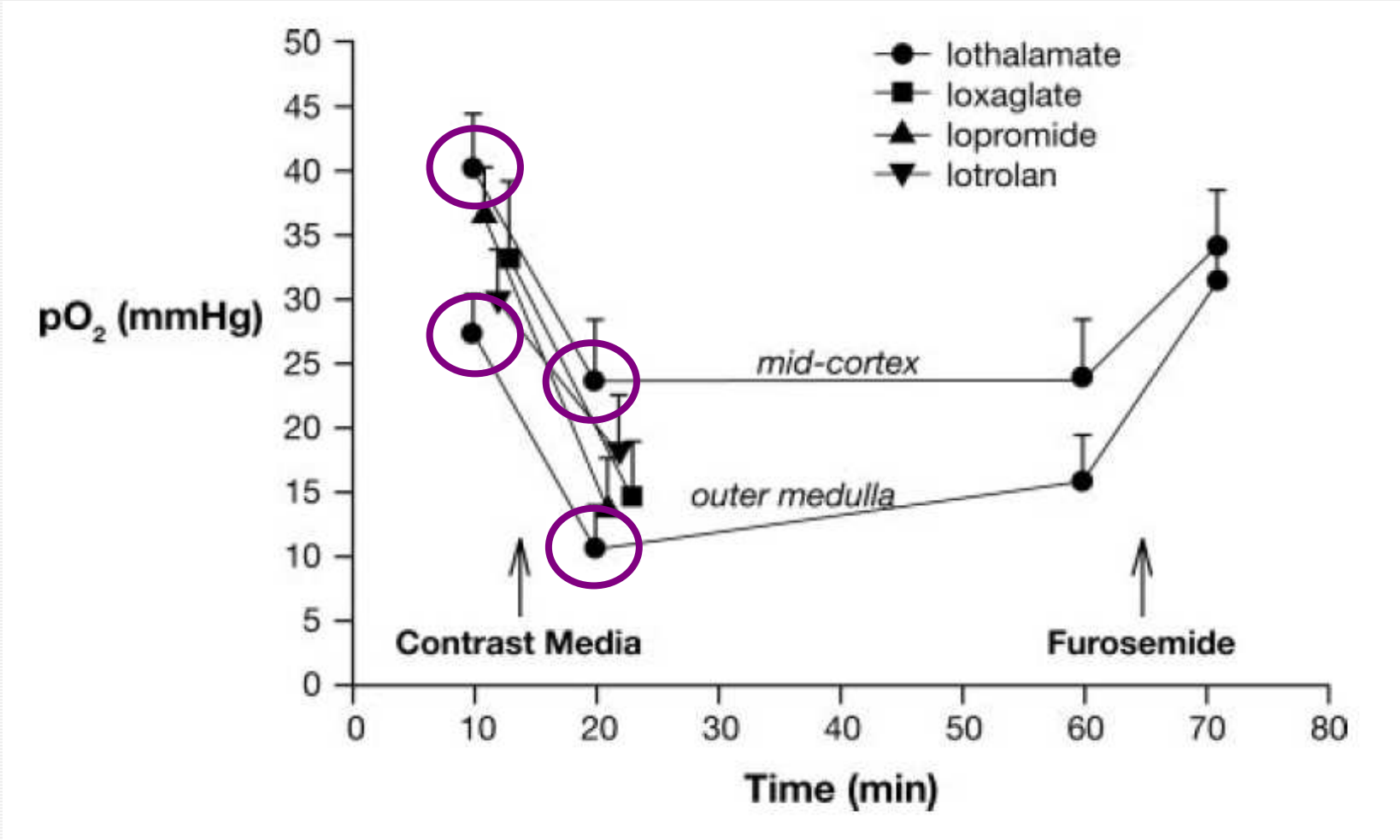

*(Chawla LS, Kidney Int 2012; 82: 516)*




*(Chawla LS, Kidney Int 2012; 82: 516)*

# Mécanisme de l'IRA induite par les PCI (1)

## Effet de l'injection de PCI sur la $pO_2$ intra-rénale chez le rat




*(Heyman SN et al. Clin J Am Soc Nephrol 2008: 288-296)*

# Mécanisme de l'IRA induite par les PCI (2)

**Facteurs favorisants :**

- **IRC**
- **Diabète**
- **Insuffisance cardiaque**
- **Déplétion**
- **AINS**
- **…**

→

**PCI**

↓

**Hypoxie du cortex et de la médullaire externe**

↓

**↓DFG**

# Evaluation et minimisation du risque

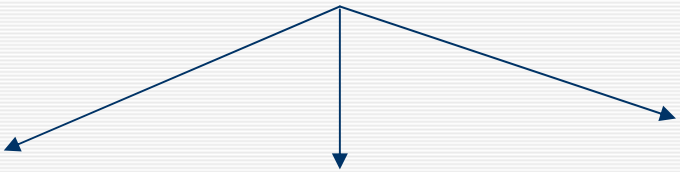
**Identification des patients à risque**

↓

**PCI indispensable ?** → **NON**

↓

**OUI**



**Avertir radiologue / cardiologue**

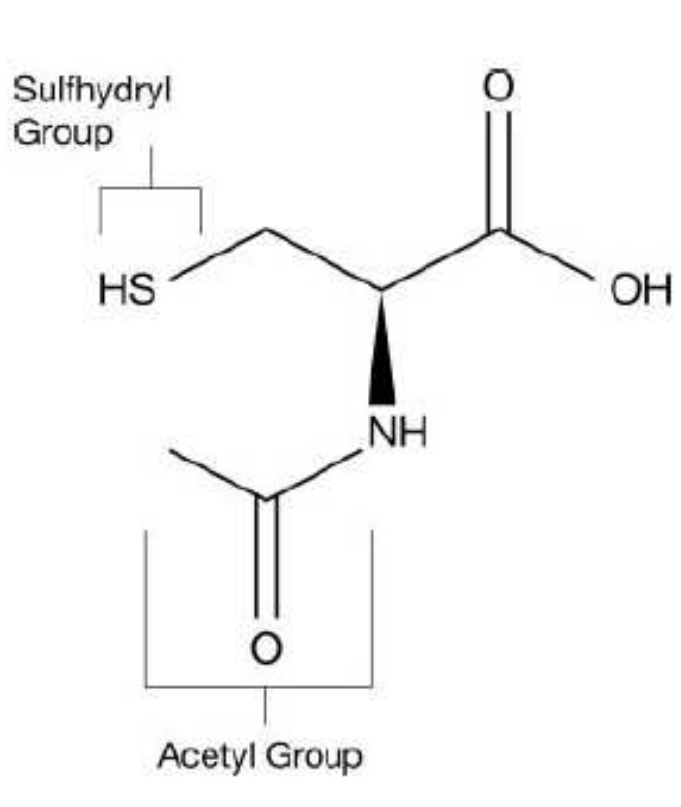
**Stop diurétiques AINS metformine 48h avant PCI**

**Protocole de prévention**

# Evaluation et minimisation du risque

- **N-acétylcystéine (LYSOMUCIL ®)**
- **« Diurèse forcée »**
- **Hydratation péri-PCI**
  **- NaCl**
  **- Bicarbonate de Na**

# Caractéristiques de la N-acétylcystéine (NAC)



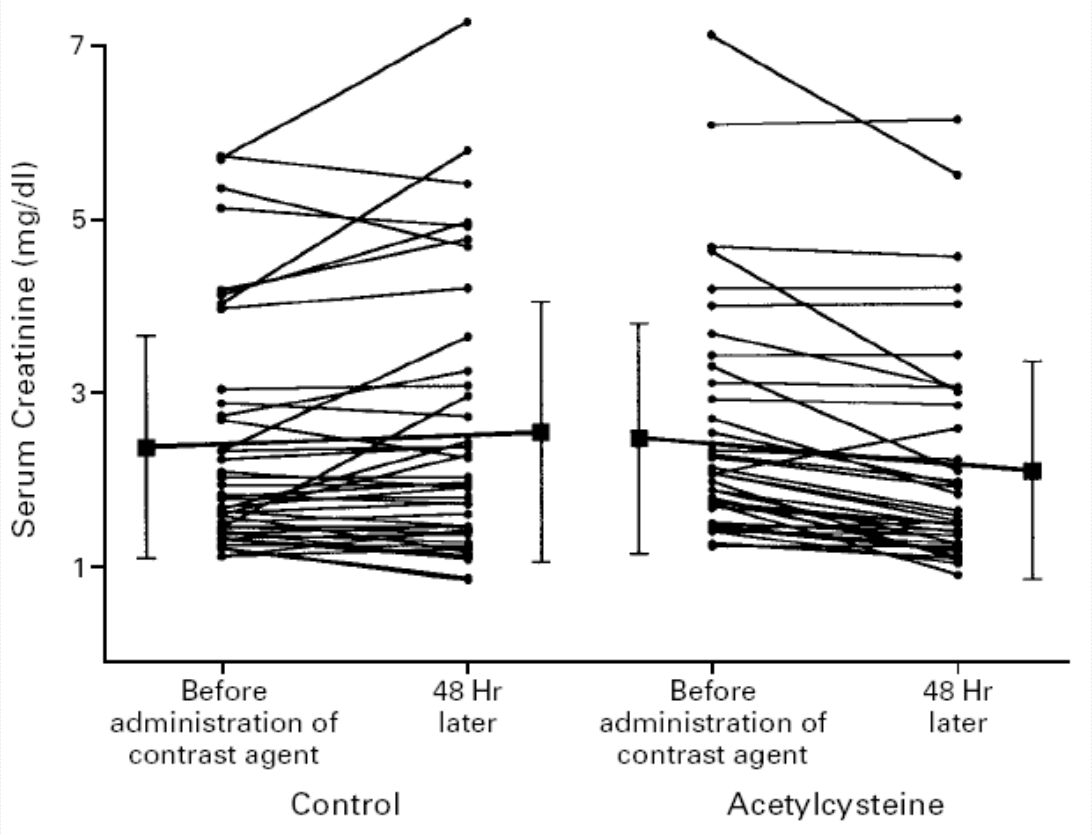

- **Biodisponibité médiocre (< 5 %)**
- **Métabolisation rapide (deacétylation)**
- **Effets recherchés:**
  - **anti-oxydant**
  - **néphroprotection**
    **(prévention déplétion glutathion) ?**
  - **vasodilatation intra-rénale ?**

The NEW ENGLAND JOURNAL of MEDICINE

# PREVENTION OF RADIOGRAPHIC-CONTRAST-AGENT–INDUCED REDUCTIONS IN RENAL FUNCTION BY ACETYLCYSTEINE

MARTIN TEPEL, M.D., MARCUS VAN DER GIET, M.D., CAROLA SCHWARZFELD, ULF LAUFER, M.D., DIETER LIERMANN, M.D., AND WALTER ZIDEK, M.D.

- **83 patients**
  **créat. sér ≃ 2,4 mg/dl**
  **CT scan + contraste**
- **NaCl 0.45 % i-v**
  **12h avant → 12h après**
  **± NAC 600 mg 2x/j**
  **J-1 et J0**




*(Tepel M et al. N Engl J Med 2000; 343: 180-4)*

# Depuis lors …jusqu'à ce jour (oct 2012)

**60 études  cliniques dont**

**…. 33 études randomisées (3.622 participants)**

**…. 15 méta-analyses**

**….  2 analyses des méta-analyses (!)**

**… non concordantes !!**

REVIEW | Annals of Internal Medicine

# Meta-analysis: Effectiveness of Drugs for Preventing Contrast-Induced Nephropathy

Aine M. Kelly, MD, MS; Ben Dwamena, MD; Paul Cronin, MD, MS; Steven J. Bernstein, MD, MPH; and Ruth C. Carlos, MD, MS

***The use of NAC is reasonable in high-risk patients who are to receive large or repeated volumes of contrast agents.***

*(Kelly AM, Ann Intern Med 2008; 148: 284)*

Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION

# Contrast-Induced Nephropathy

## A Clinical and Evidence-Based Approach

Martin Tepel, MD; Peter Aspelin, MD; Norbert Lameire, MD

***… At present there is limited evidence that NAC may be useful as standard prophylactic procedure in patients at high risk for contrast-induced nephropathy .***

*(Tepel M et al. Circulation 2006; 113: 1799-1806)*

**Preventing radiocontrast-induced nephropathy in chronic kidney disease patients undergoing coronary angiography**

Yao-Min Hung, Shoa-Lin Lin, Shih-Yuan Hung, Wei-Chun Huang, Paul Yung-Pou Wang

***… clinical data regarding the efficacy of NAC for RCIN remains debatable. However, considering the very low toxicity and cost of this drug, we recommend the use of oral NAC at a dose of 1.2 g twice daily on the day before and day of the procedure to patients at risk.***

*(Hung YM, World J Cardiol 2012; 4: 157)*

# A proscrire

**Toutes les manœuvres de « diurèse forcée »**

**(furosemide, mannitol, dopamine)**

**$\rightarrow \uparrow$ risque d'IRA**

# A recommander chez les patients à risque d'IRA sur PCI

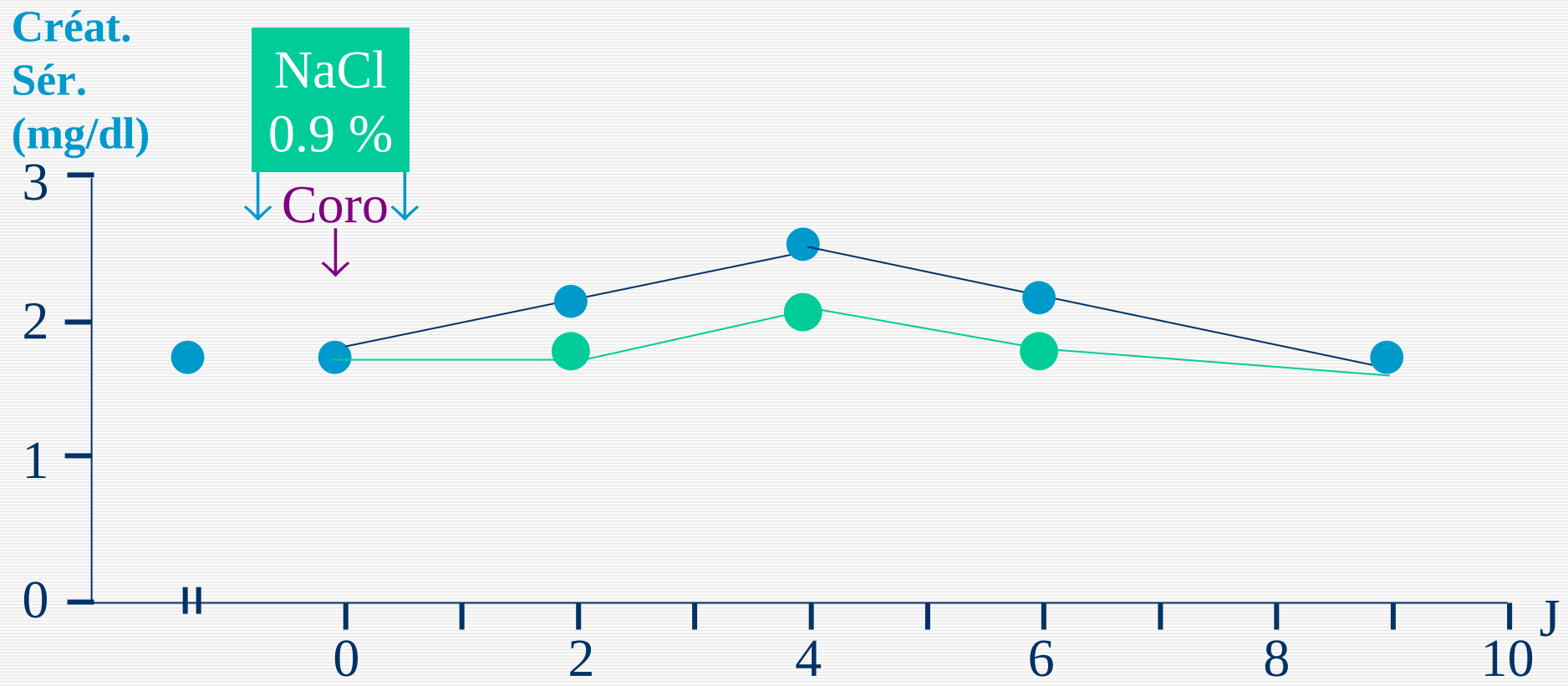
***… Although the optimal regimen is uncertain, available data support a regimen of 0.9 % saline at 1 ml/kg/h i-v from up to 12 hrs before administration of contrast and for up to 12 hrs after…***

***(Barrett 2006)***

***… Current evidence supports periprocedural hydration with preferably i-v administration of 0.9 % isotonic saline or an isotonic sodium bicarbonate solution.***

***(Tepel 2006)***

# A recommander chez les patients à risque

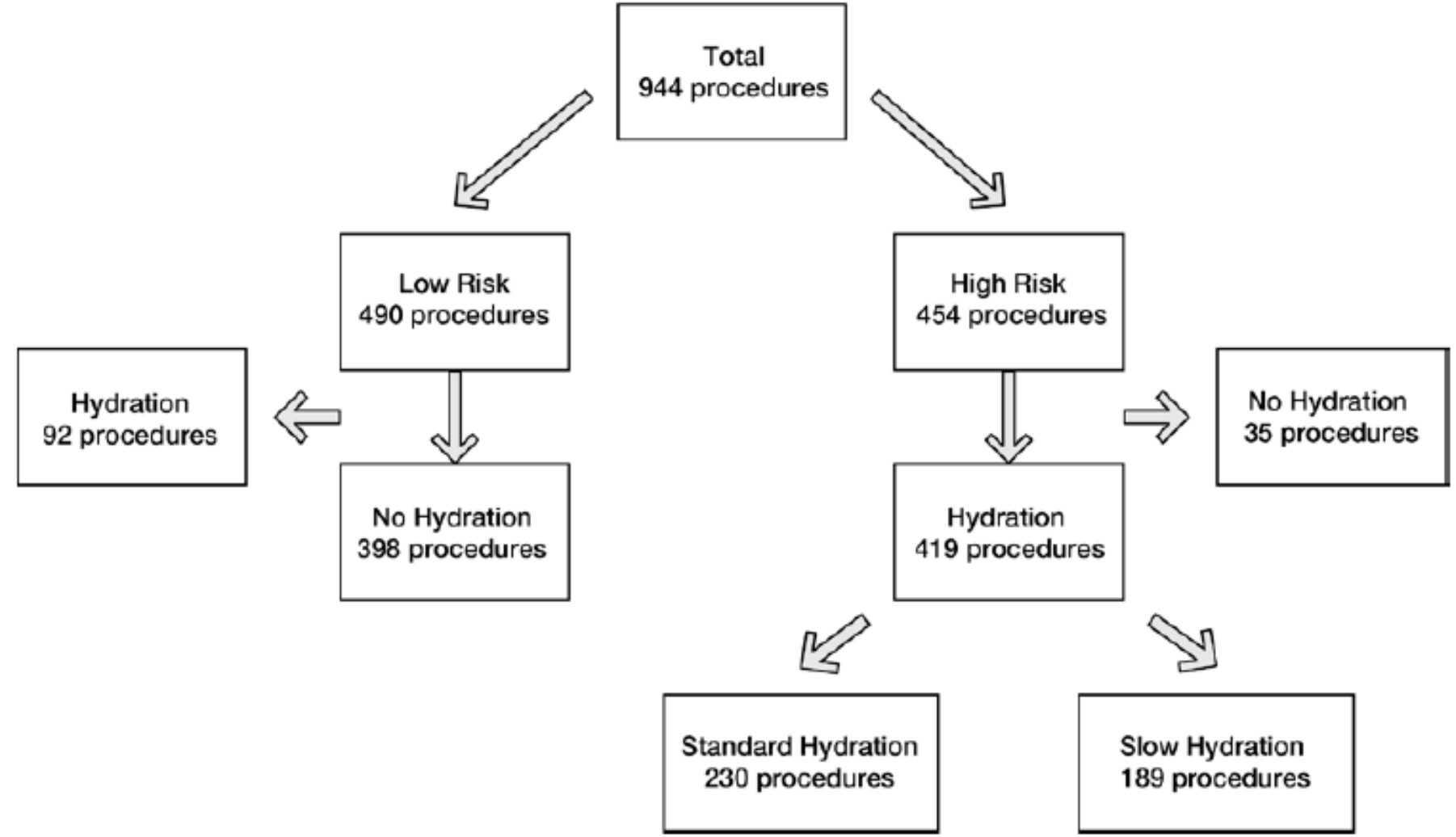


# Epidemiology of Contrast Material–induced Nephropathy in the Era of Hydration[1]

Corinne E. A. Balemans, MD
Louis J. M. Reichert, MD, PhD
Bert I. H. van Schelven, MD
Jan A. J. G. van den Brand, MSc
Jack F. M. Wetzels, MD, PhD

| High-Risk Group | Low-Risk Group |
|---|---|
| • eGFR <60 mL/min/min and diabetes mellitus | • eGFR >60 mL/min |
| • eGFR of 45–60 mL/min and at least two of the following risk factors: peripheral arterial disease, heart failure, patient age of more than 75 years, anemia (hematocrit level <0.39 in men and <0.36 in women), dehydration, and use of diuretics, nonsteroidal antiinflammatory drugs, or both | • eGFR of 45–60 mL/min and fewer than two risk factors |
| • eGFR <45 mL/min | |

*(Balemans CEA, Radiology 2012; 263: 706)*




*(Balemans CEA, Radiology 2012; 263: 706)*

## Incidence of CIN according to Risk Category

| Risk Group and Category | Incidence of CIN (%) |
|---|---|
| Low-risk group | |
| Absolute eGFR >60 mL/min ($n = 276$) | 2.5 (7) |
| Absolute eGFR 45–60 mL/min ($n = 214$)* | 2.3 (5) |
| High-risk group | |
| Absolute eGFR 45–60 mL/min ($n = 227$)* | 1.8 (4) |
| Absolute eGFR <45 mL/min ($n = 227$) | 3.1 (7) |

Note.—Data in parentheses are numbers of patients.

* In patients with absolute eGFR of 45–60 mL/min, risk was based on the number of confounding risk factors (Fig 1).

*(Balemans CEA, Radiology 2012; 263: 706)*

## Identified independent risk factors
## (multiple logistic regression analysis)

**-heart failure**
**-low BMI**
**-repeated contrast administration**

*(Balemans CEA, Radiology 2012;263:706)*

# Faut-il préférer le bicarbonate de Na ?

- **119 patients**
  **créat sér ~ 1.8 mg/dl**

- **perfusion**
  **1h avant → 6h après**
  **(3 ml/kg/h)**

**<u>% IRA</u>**

**NaCl 0.9 % : 13.6 %**
**bicar iso : 1. 7 %**

**P = .02**

*(Merten GS et al. JAMA 2004; 291: 2328)*

**Sodium bicarbonate for prevention of contrast-induced acute kidney injury: a systematic review and meta-analysis**

Eric A. J. Hoste[1,2], Jan J. De Waele[1], Sofie A. Gevaert[3], Shigehiko Uchino[4] and John A. Kellum[2]

Nephrol Dial Transplant (2010) 25: 747–758

***« …there is benefit favoring sodium bicarbonate … »***

**Sodium Bicarbonate for the Prevention of Contrast Induced-Acute Kidney Injury: A Systematic Review and Meta-analysis**

Somjot S. Brar,* Swapnil Hiremath,† George Dangas,* Roxana Mehran,* Simerjeet K. Brar,* and Martin B. Leon*

*Clin J Am Soc Nephrol* 4: 1584–1592, 2009.

***« no evidence of benefit for hydration with sodium bicarbonate … »***

# En pratique : prévenir le risque d'IRA induite par des PCI en cas d'examen RX programmé

**Chez les patients susceptibles de faire partie des catégories à risque (age > 75, diabète, notion de néphropathie, insuffisance cardiaque sévère, cirrhose décompensée…), mesurer le taux de créatinine sérique.**

**Si le DFG est < 40 ml/min:**

- **interrompre diurétique, AINS (et metformine ?) 48h avant l'examen**
- **en l'absence de contre-indication, perfuser du sérum physiologique, 1 ml/kg/h, durant 24h (de 12h avant à 12h après l'examen)**
- **utiliser le volume de PCI le plus faible possible**

# En pratique : que faire en cas d'examen RX urgent nécessitant un PCI chez un patient paraissant à risque ?

- **Protocole bicarbonate de Merten :**
  **154 mmol/L de bicarbonate Na dans solution glucosée**
  **- 3 ml/kg/h 1h avant le PCI**
  **- 1 ml/kg/h les 6h suivantes**

- **+ Lysomucil ® 300-600 mg i-v ?**

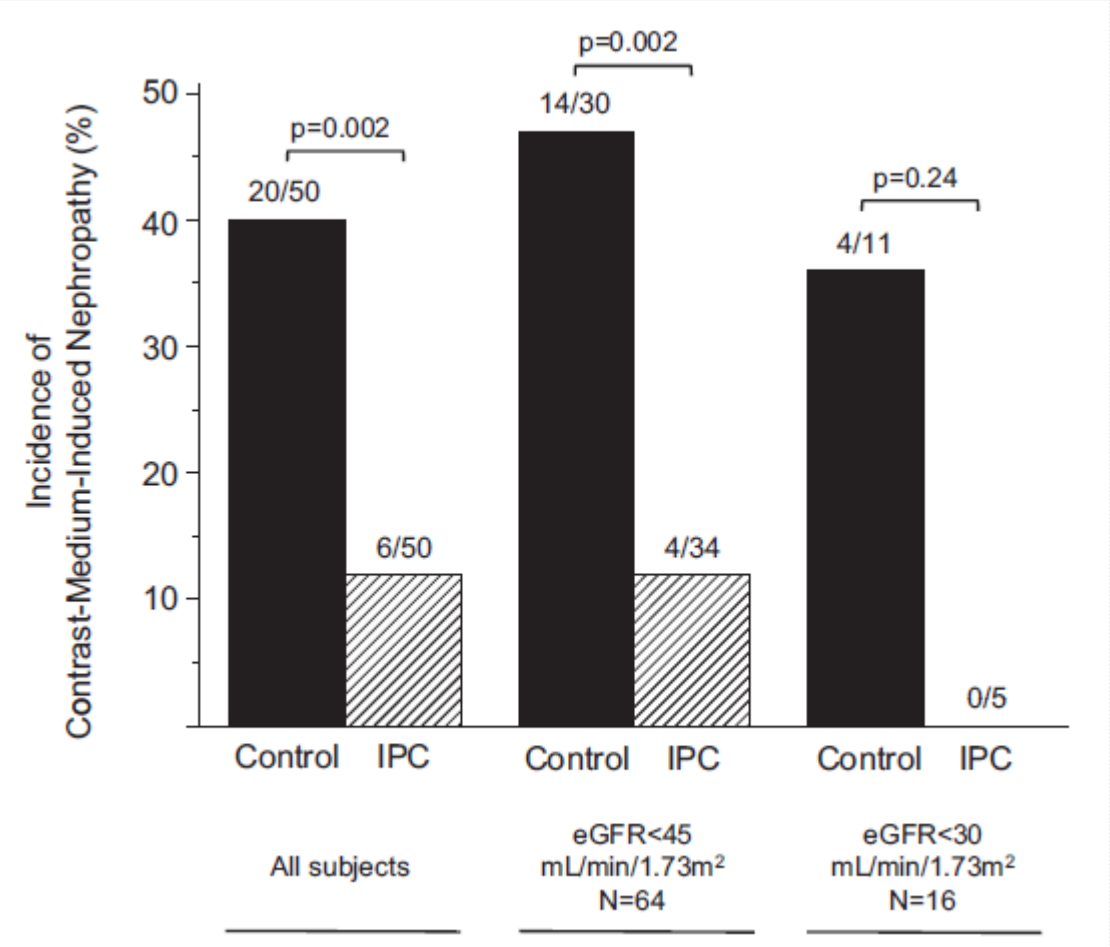
**Ischemic Preconditioning for Prevention of Contrast Medium–Induced Nephropathy : Randomized Pilot RenPro Trial (Renal Protection Trial)**

Fikret Er, Amir M. Nia, Henning Dopp, Martin Hellmich, Kristina M. Dahlem, Evren Caglayan, Torsten Kubacki, Thomas Benzing, Erland Erdmann, Volker Burst and Natig Gassanov

*Circulation.* 2012;126:296-303

- **Elective coronary angiography**
- **High-risk patients (mean Mehran score : 13)**
- **100 patients randomized to treatment**
  - **50 classic i-v hydration + NAC 600 mg**
  - **50 idem + ischemic preconditioning**

# Incidence of CIN in patients with ischemic preconditioning and control patients




*(Er F, Circulation 2012; 126: 296)*

- **Insuffisance rénale aiguë (IRA) induite par les produits de contraste iodés (PCI)**

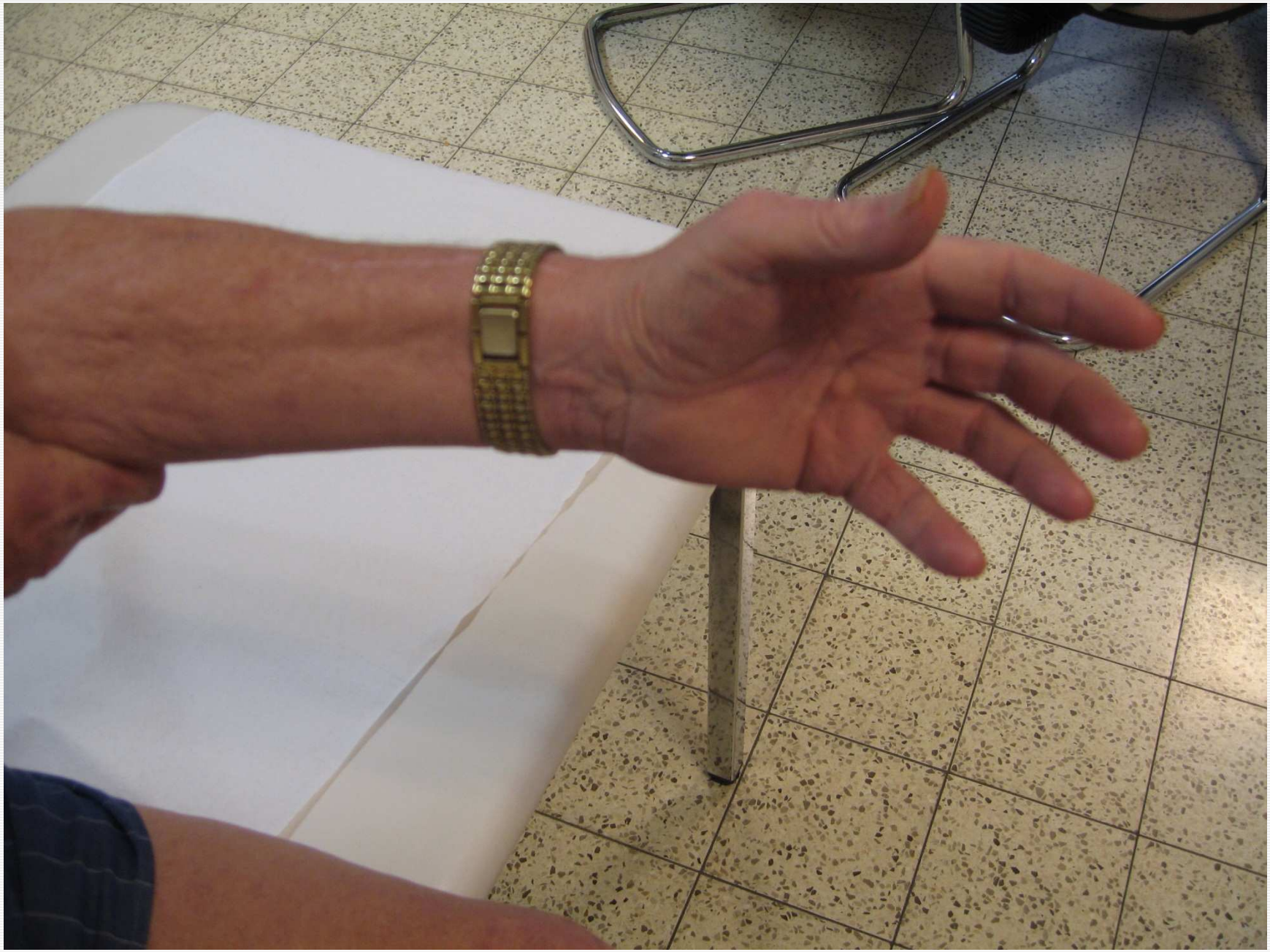
- **Fibrose systémique néphrogénique (FSN) induite par le gadolinium (Gd)**

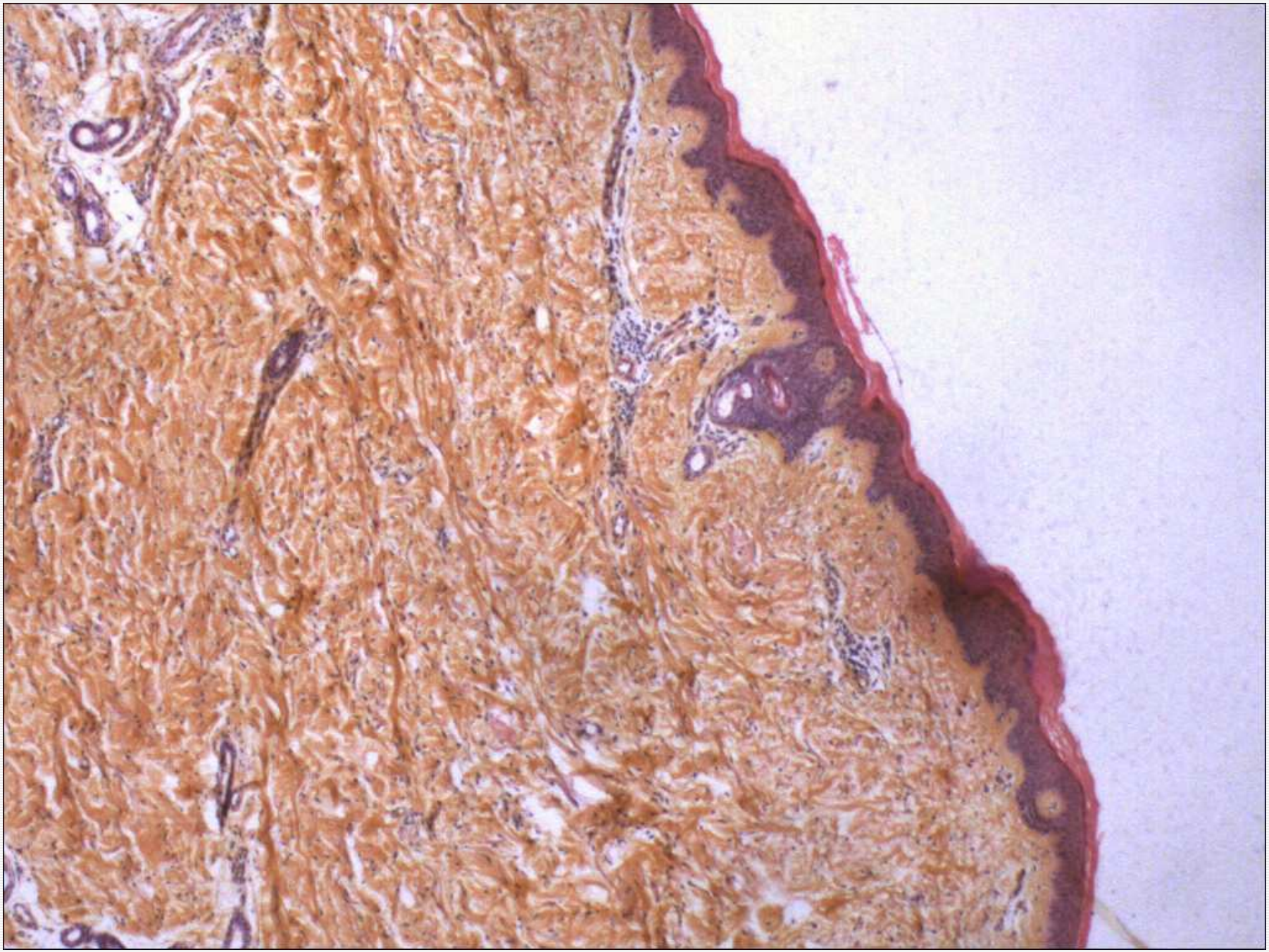
Homme de 57 ans

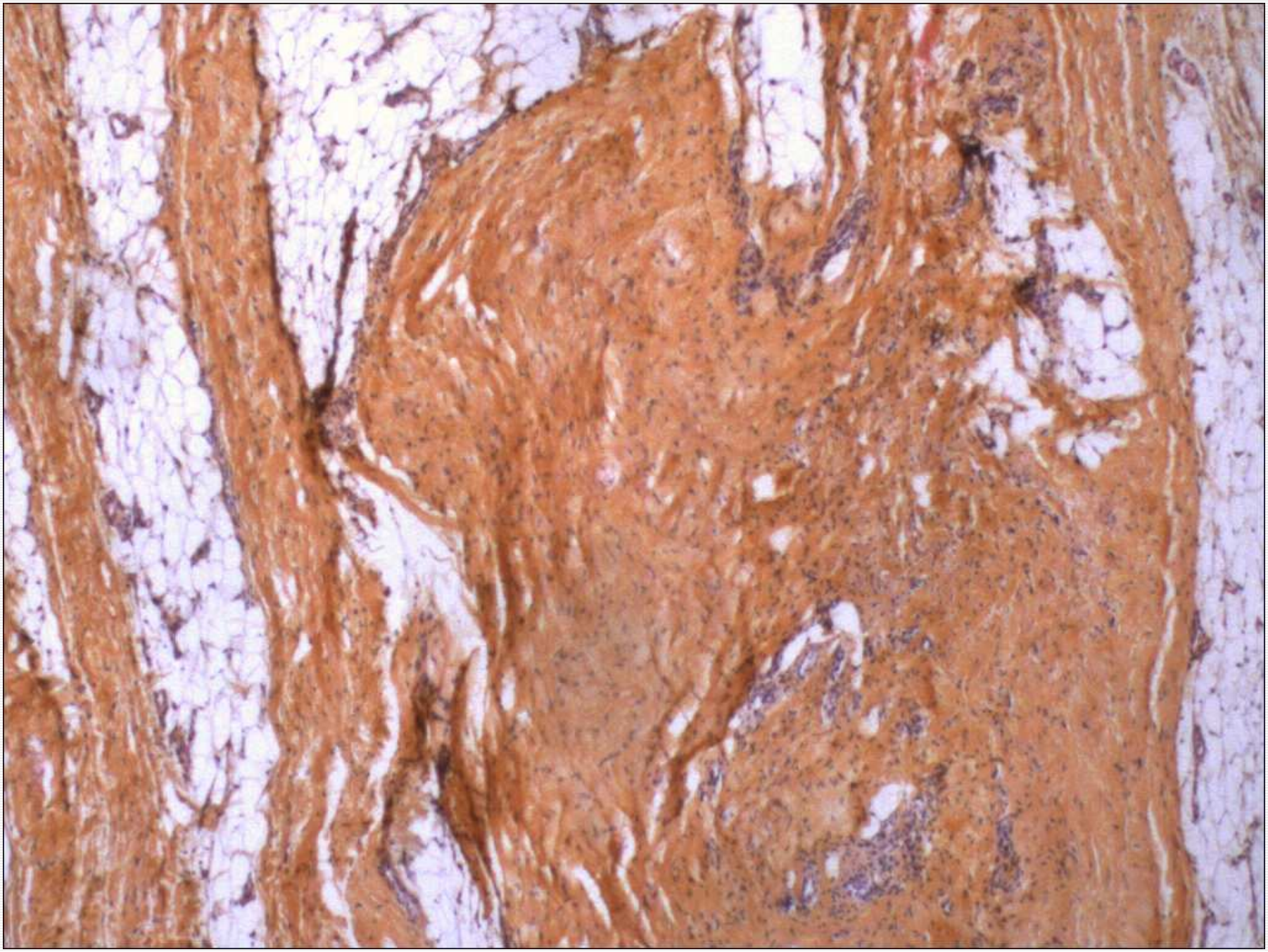
Diabète de type 2, multicompliqué

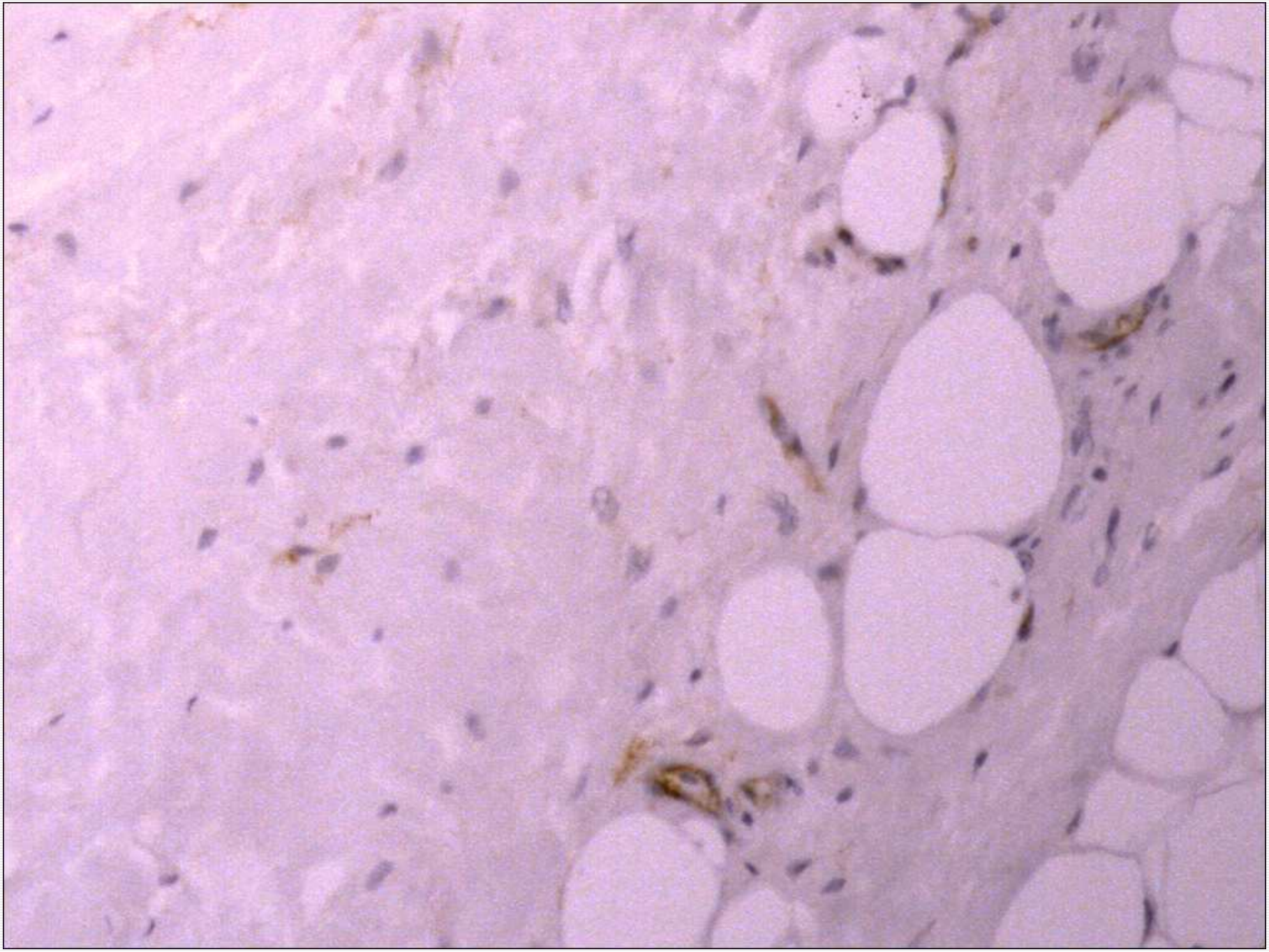
Hémodialyse depuis 2001 à Verviers

Admis à St. Luc, en Rhumatologie, en décembre 2003 pour bilan d'
« *un état sclérodermiforme essentiellement localisé au niveau des cuisses, des jambes et des avant-bras, limitant la flexion-extension des membres inférieurs et supérieurs, ainsi que d'une faiblesse musculaire extrême* »









THE LANCET.

# Scleromyxoedema-like cutaneous diseases in renal-dialysis patients

**Shawn E. Cowper MD, Howard S Robin MD, Steven M Steinberg MD, Lyndon D Su MD, Samardeen Gupta MD and Philip E LeBoit MD**

*(Cowper SH et al. The Lancet 2000; 356:1000-1001)*

Current Opinion in Rheumatology

# Nephrogenic fibrosing dermopathy (NFD): the first 6 years.

Cowper SE, Shawn E

***…The only immutable epidemiological association of NFD is renal insufficiency.***

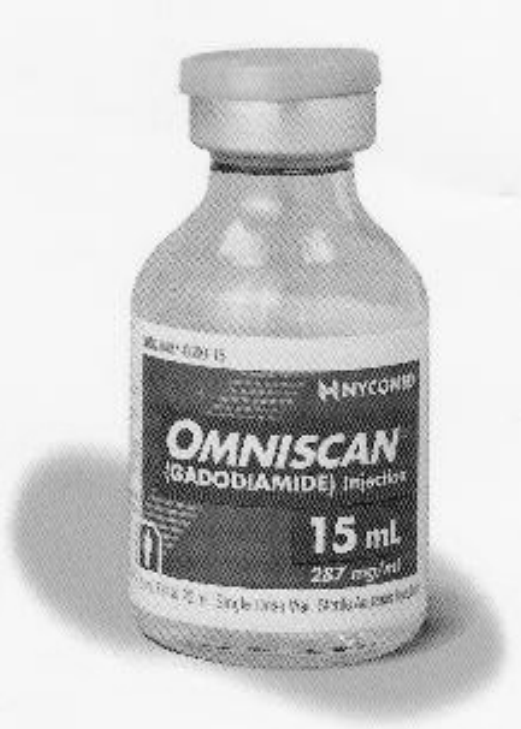
***…A baffling mystery is why no cases of NFD were reported before 1997. The abrupt appearance of a new disease process suggests that toxic exposures, new medications, new infectious agents, or new medical techniques may be involved. Intense study however, has failed to identify a common medication (or class of medications) among all patients with NFD, and no evidence of an infectious or toxic process….***

*(Curr Opin Rheumatol 2003; 15:785-790)*


Department of Health and Human Services
Centers for Disease Control and Prevention
CDC
CDC

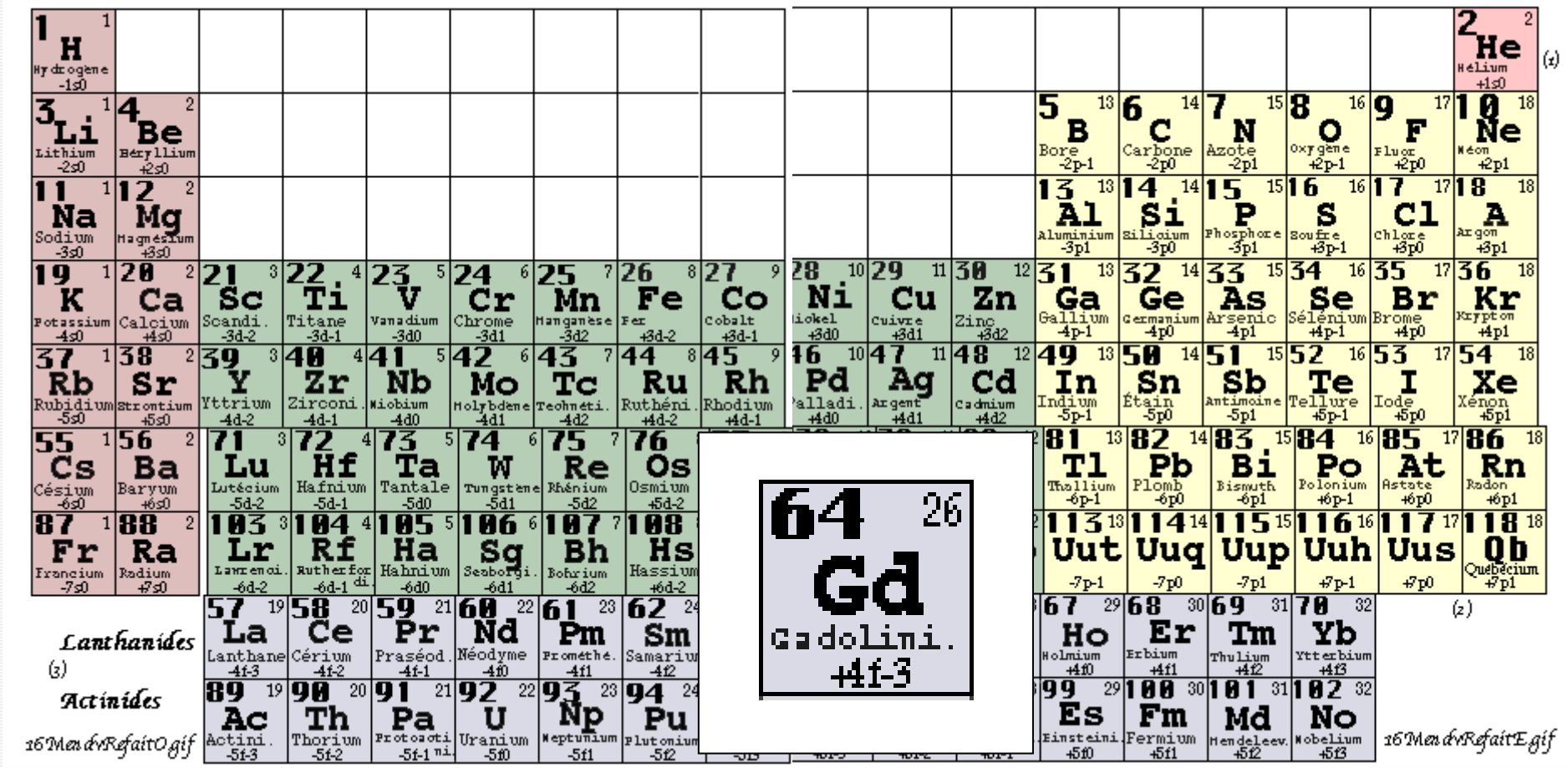

RV.GA 2..6

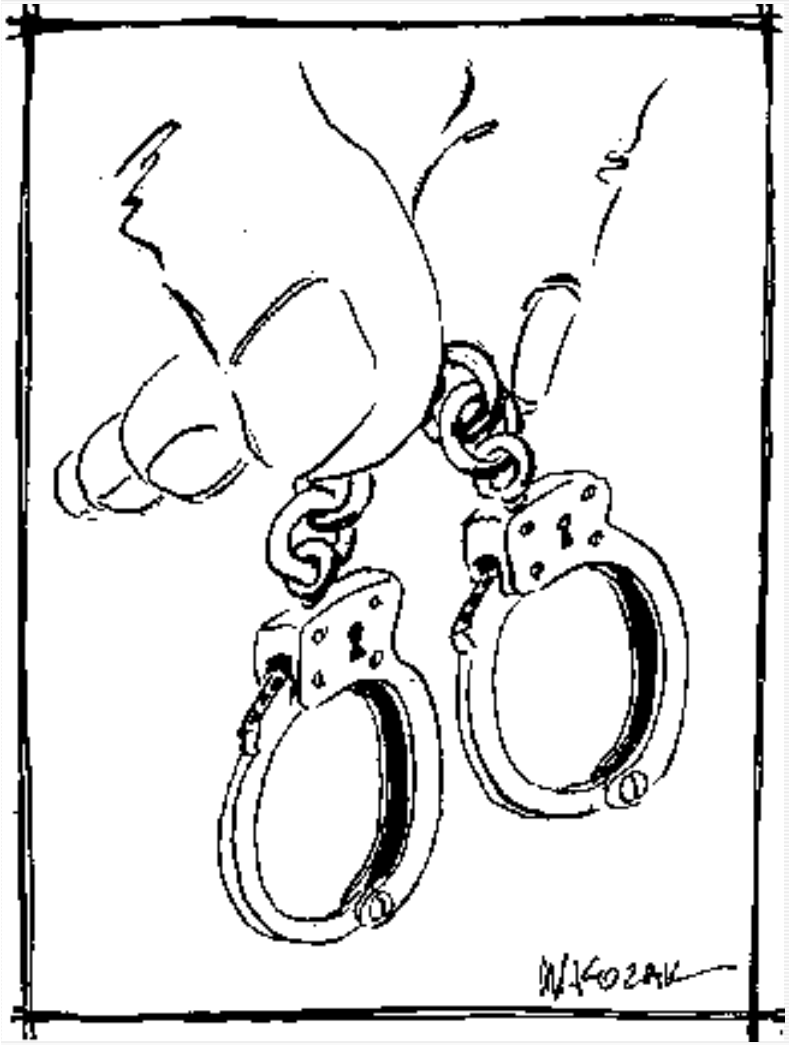

RX.GA2..6

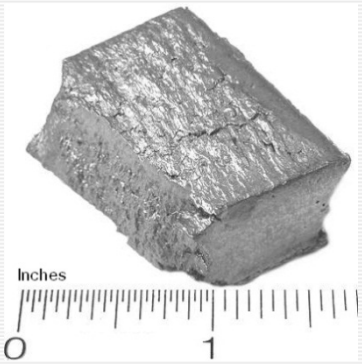

NYCOMED
OMNISCAN
(GADODIAMIDE) Injection
15 mL
287 mg/mL


NDC 50419-188-05 5mL
sterile solution
Magnevist®
(brand of gadopentetate dimeglumine)
injection
Rx only
2006
BERLEX

# Tableau de Mendeleev








Gadolinite

Johan Gadolin (1760 – 1852)

Nephrology Dialysis Transplantation

*Interesting Case*

# Gadolinium – a specific trigger for the development of nephrogenic fibrosing dermopathy and nephrogenic systemic fibrosis?

Thomas Grobner

Department of Nephrology, General Hospital of Wiener Neustadt, A-2700 Wiener Neustadt, Austria

***… All 9 affected patients underwent MR examination with the use of Gd-DTPA 2-4 weeks prior to development of the skin abdnormalities …***

*(Grobner Th. Nephrol Dial Transplant 2006; 21: 1104-1108)*

JASN® Journal of the American Society of Nephrology

# Nephrogenic Systemic Fibrosis: Suspected Causative Role of Gadodiamide Used for Contrast-Enhanced Magnetic Resonance Imaging

Peter Marckmann,* Lone Skov,† Kristian Rossen,‡ Anders Dupont,§
Mette Brimnes Damholt,* James Goya Heaf,* and Henrik S. Thomsen‖

*Departments of *Nephrology and ‖Diagnostic Radiology, Copenhagen University Hospital at Herlev, Herlev, Departments of †Dermatology and ‡Pathology, Copenhagen University Hospital at Gentofte, Hellerup, and §Faculty of Health Sciences, Copenhagen University, Copenhagen, Denmark*

***…All 13 had been exposed to gadodiamide before the development of nephrogenic systemic fibrosis…***

*(Marckmann P et al. J Am Soc Nephrol **2006**; 17: 2359-2362)*

American Journal of Roentgenology

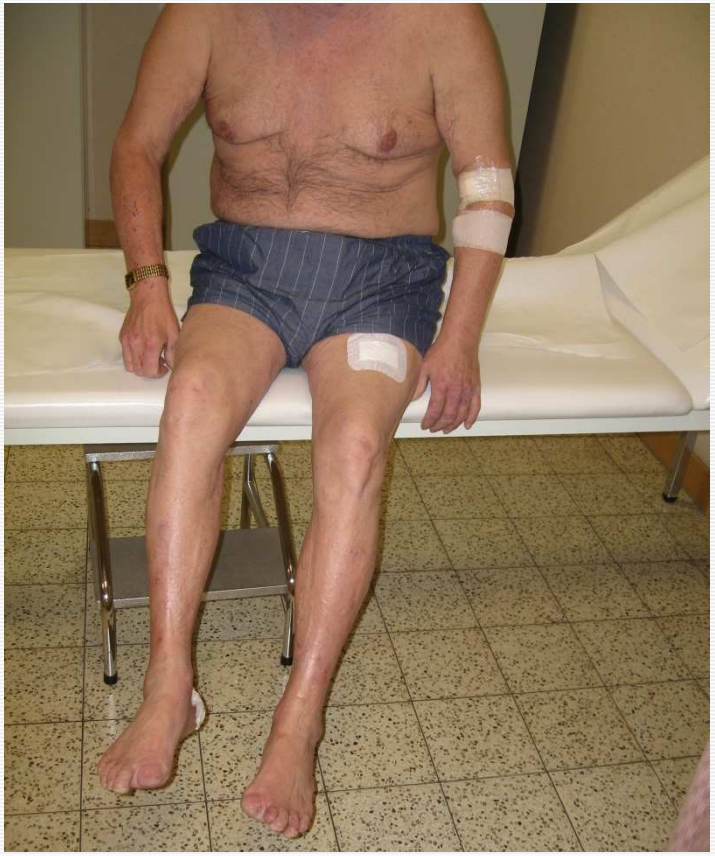
# Gadodiamide-Associated Nephrogenic Systemic Fibrosis: Why Radiologists Should Be Concerned

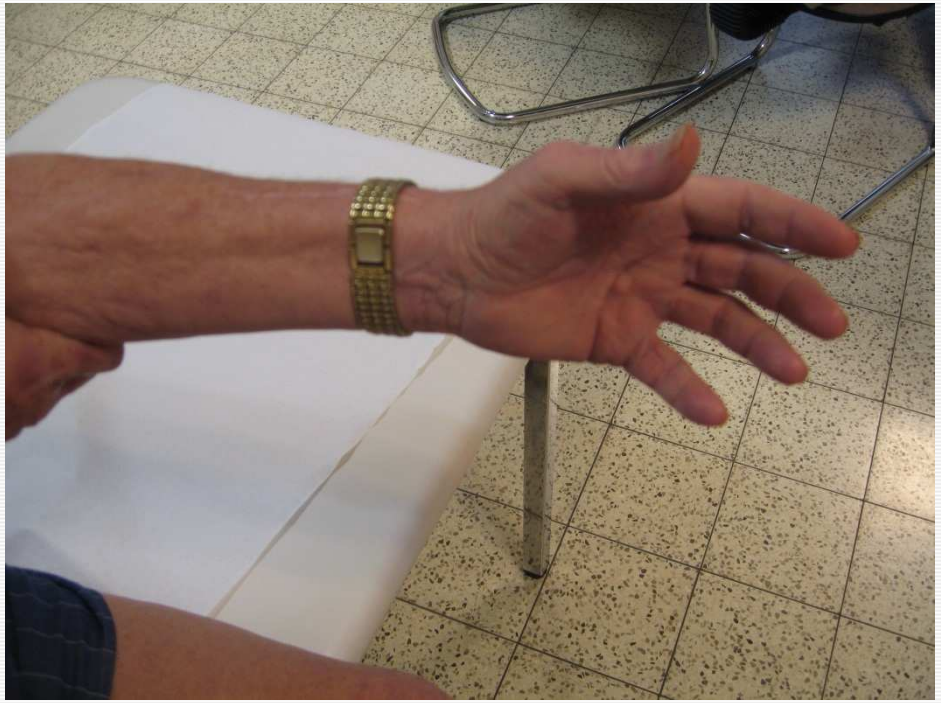
Dale R. Broome[1]
Mark S. Girguis[1]
Pedro W. Baron[2]
Alfred C. Cottrell[3]
Ingrid Kjellin[1]
Gerald A. Kirk[1]

***…All 12 patients developed skin fibrosis within 2-11 weeks after gadodiamide administration…***

*(Broome DR et al. AJR Am J Roentgenol **2007**; 188: 586-592)*

# Rappel: HD en 2001;  FSN apparue automne 2003

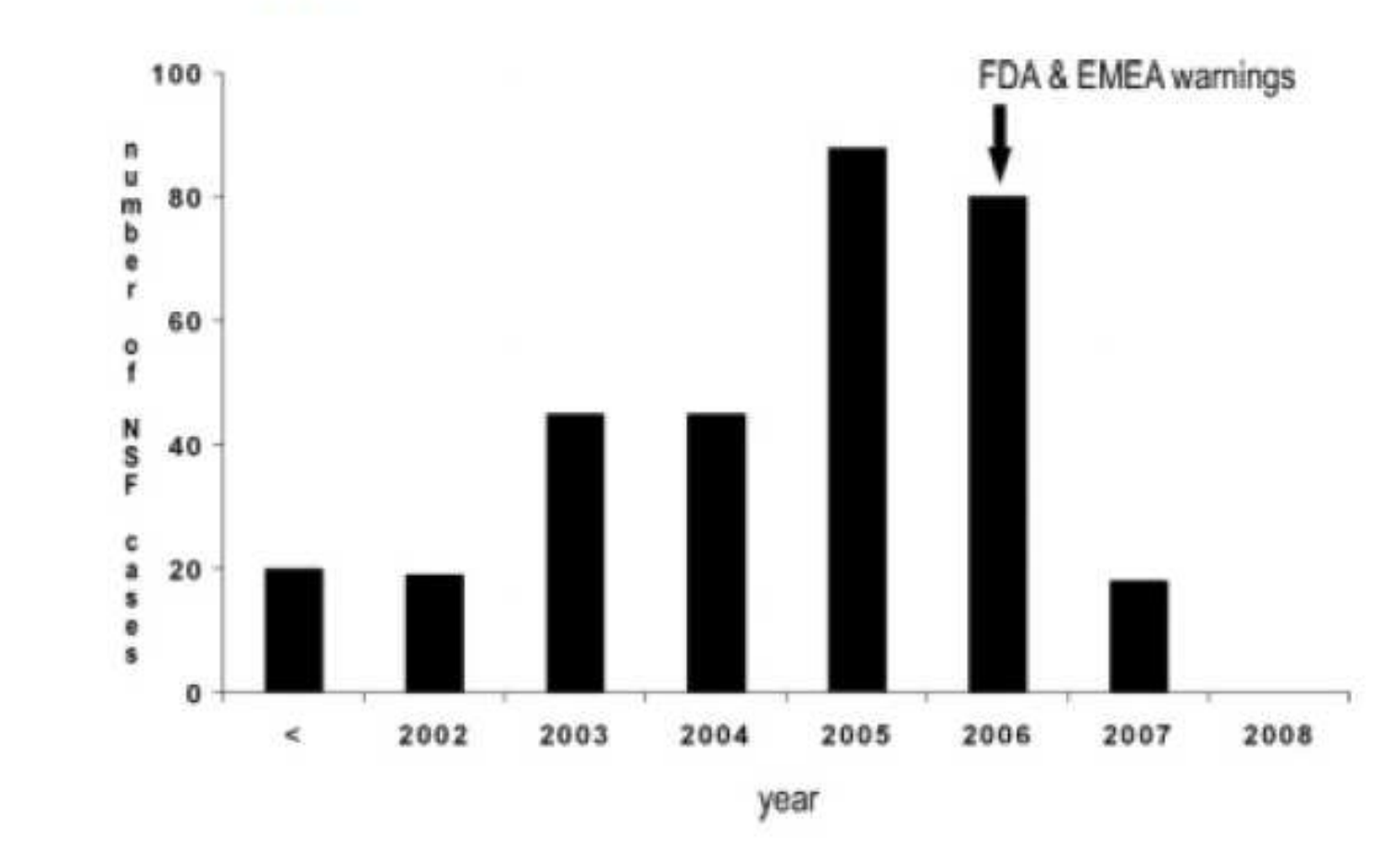




IRM avec injection de 10 à 30 ml de Magnevist ® à 4 reprises:

- décembre 2001 (membres supérieurs)
- juin 2002 (membres inférieurs)
- décembre 2002 (encéphale)
- juillet 2003 (membres inférieurs)

# Disparition des cas depuis l'application des recommandations prohibant l'utilisation du Gd en cas d'IRC sévère




*(Prince MR, J Magn Reson Imaging 2009; 30: 1298)*

Clin Kidney J (2012) 5: 82–88
doi: 10.1093/ckj/sfr172

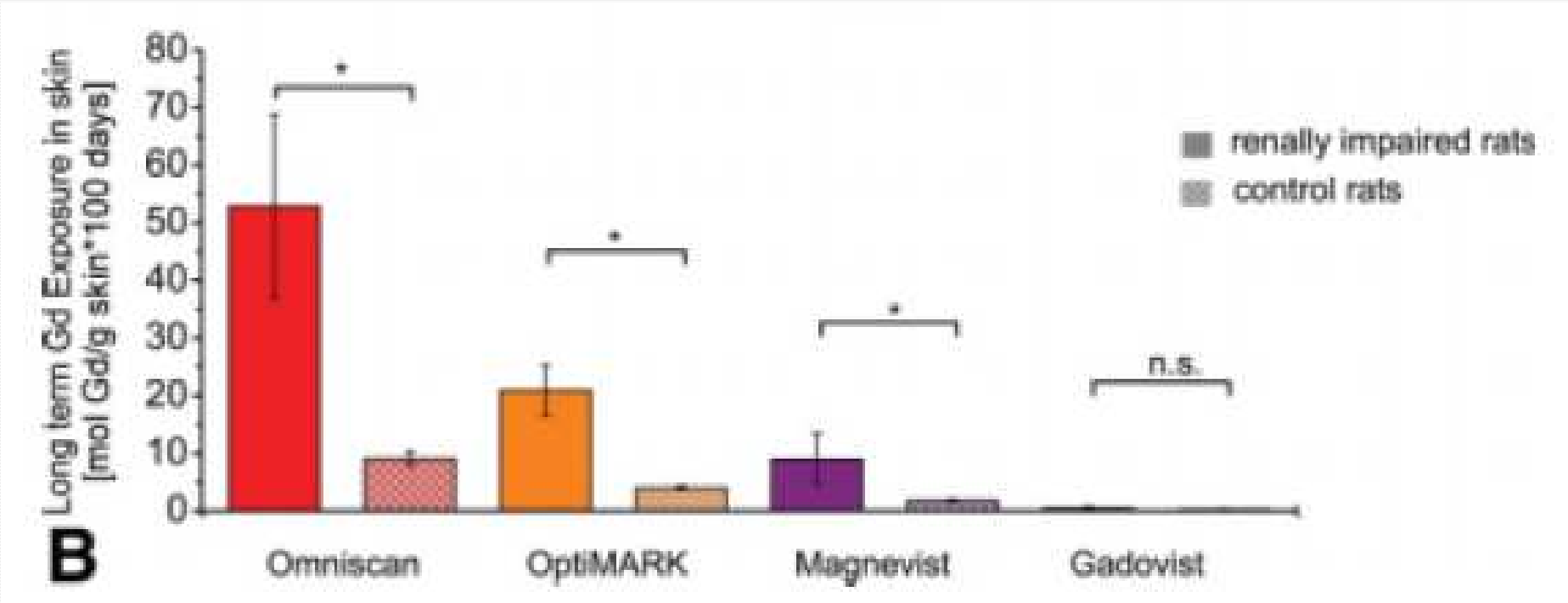
*History of Nephrology*

# Gadolinium-induced nephrogenic systemic fibrosis: the rise and fall of an iatrogenic disease

Charles L. Bennett[1,2,3,4], Zaina P. Qureshi[1,2,4], A. Oliver Sartor[5], LeAnn B. Norris[1,2], Alanna Murday[1,2], Sudha Xirasagar[5] and Henrik S. Thomsen[6]

[1]The Doris Levkoff Meddin Program on Medication Safety and the Southern Network on Adverse Reaction, South Carolina College of Pharmacy, University of South Carolina, Columbia, SC, USA, [2]The Doris Levkoff Meddin Program on Medication Safety and the Southern Network on Adverse Reaction, South Carolina College of Pharmacy, Medical University of South Carolina, Charleston, SC, USA, [3]The Hollings Cancer Center, Medical University of South Carolina, Charleston, SC, USA, [4]Health Services Policy and Management, Arnold School of Public Health, University of South Carolina, Columbia, SC, USA, [5]Department of Medicine: Section of Hematology & Medical Oncology and Department of Urology, Tulane School of Medicine and the Tulane Cancer Center, New Orleans, LA, USA, and [6]Department of Diagnostic Sciences, Faculty of Health Sciences, University of Copenhagen, Copenhagen, Denmark

# Induction de la FSN chez le rat traité par les chélates de Gd utilisés en IRM: effet de l'IR et du type de chélate




*(Sieber MA, J Magn Reson Imaging 2009; 30: 1266)*

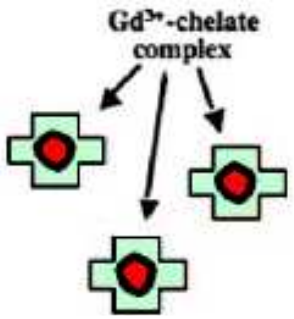
# Caractéristiques pharmacocinétiques du Gd

- **Eliminé à 97 % par le rein**
- **Demi-vie:**

| DFG (ml/min) | (h) |
|---|---|
| Nl | 1.3 |
| 60-30 | 5.6 |
| 29-15 | 9.2 |
| dialyse | 34-53 |

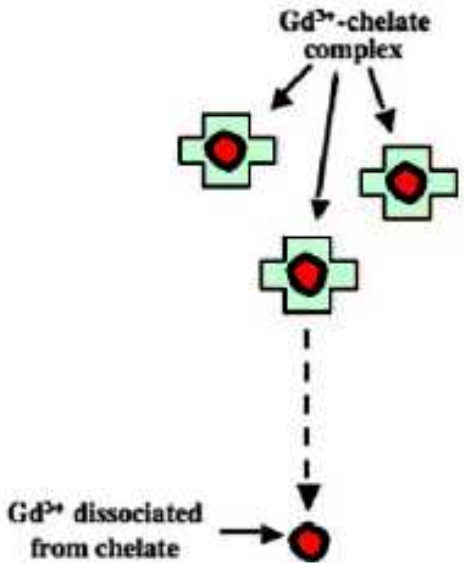
*(Joffe P, Acad Radiol 1998; 5: 491)*

# Mécanisme supposé de la FSN



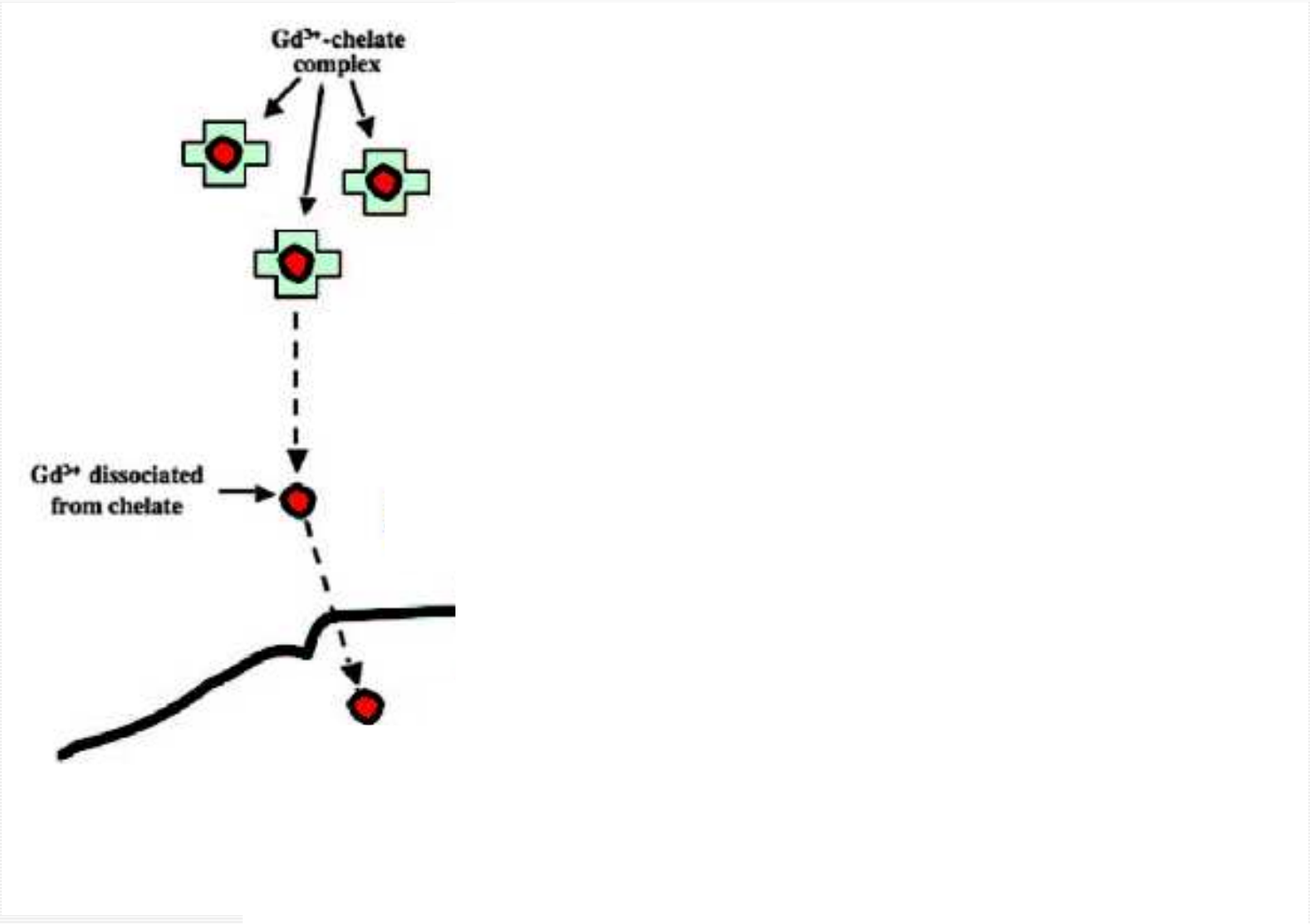

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN



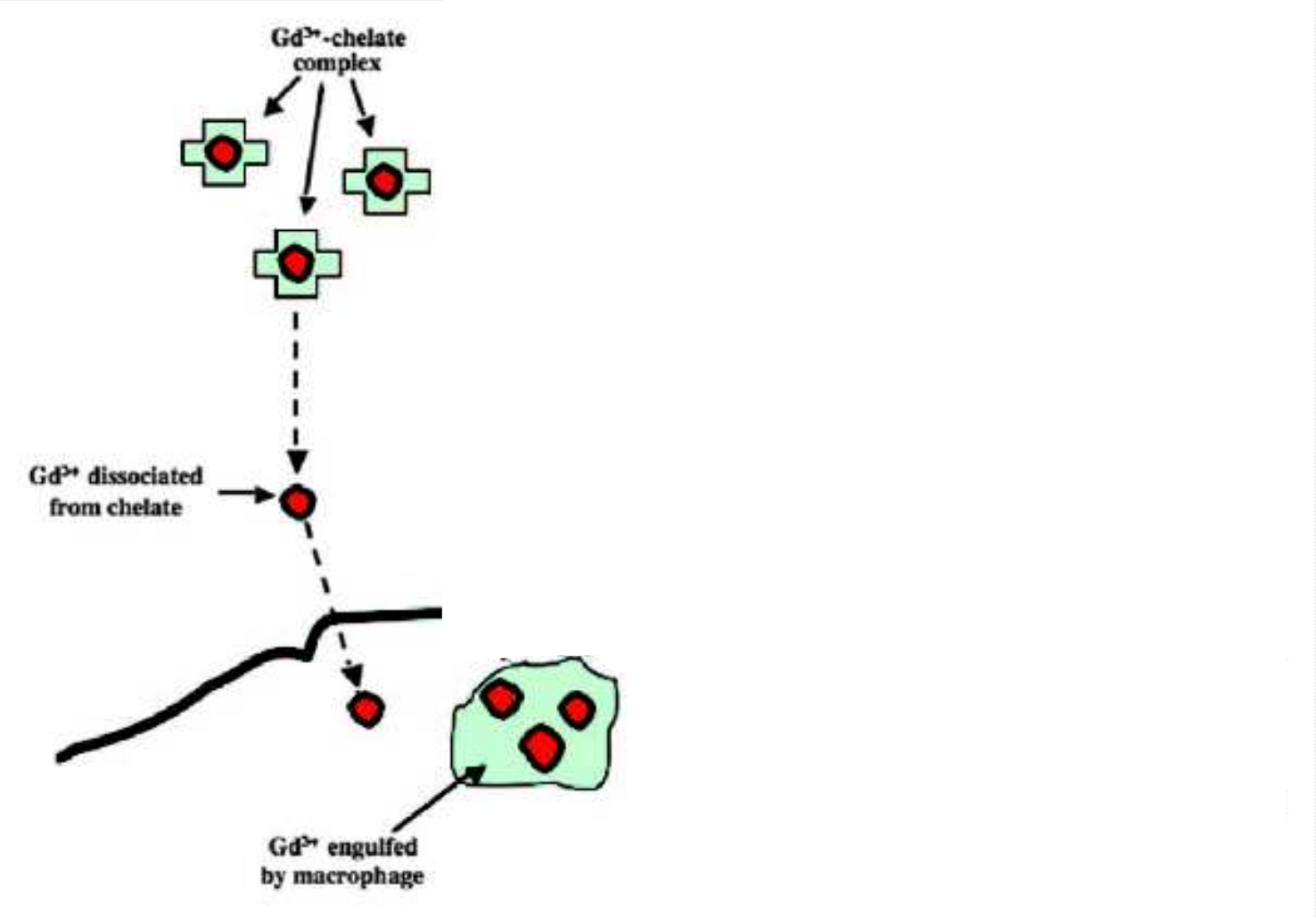

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN



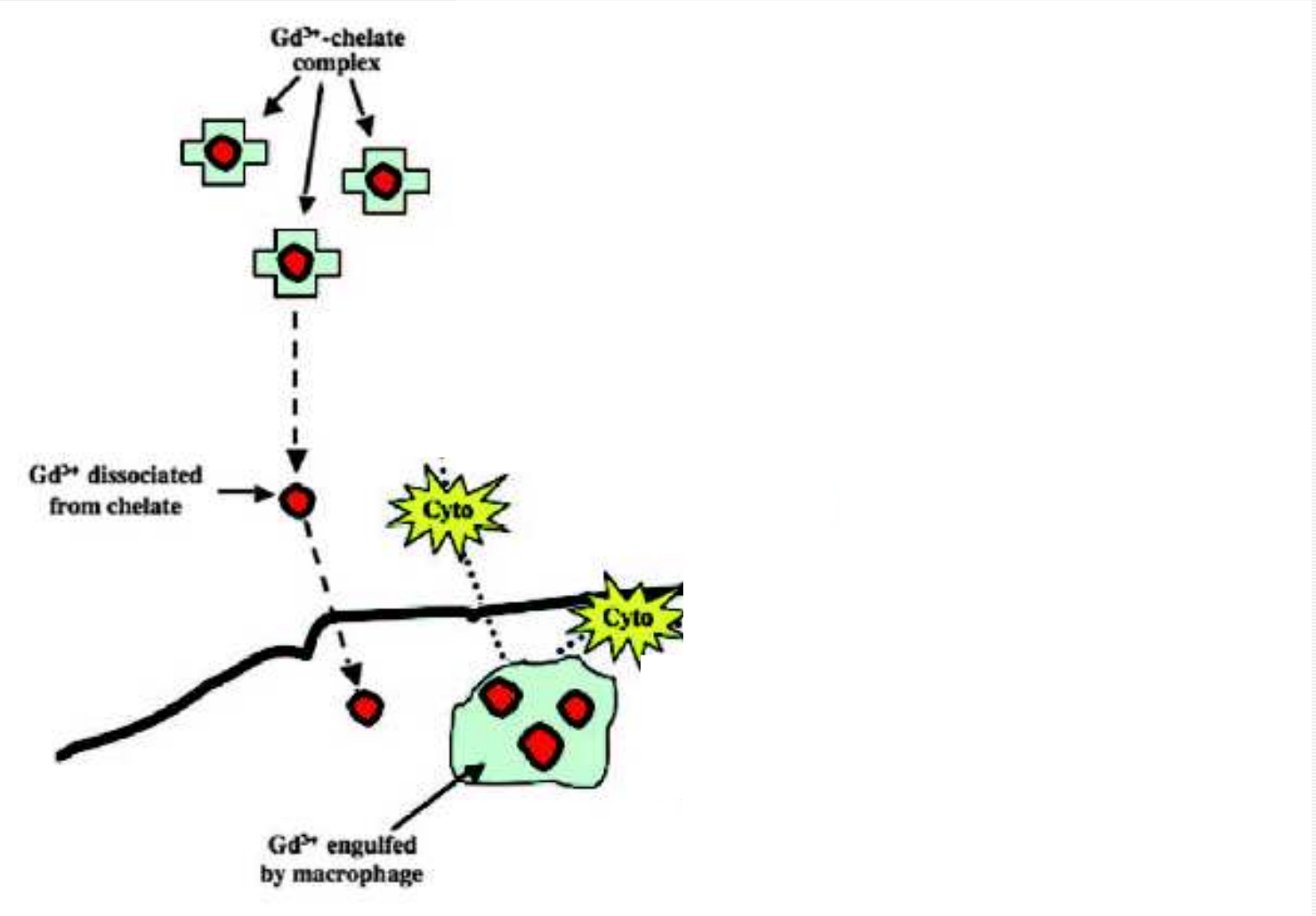

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN



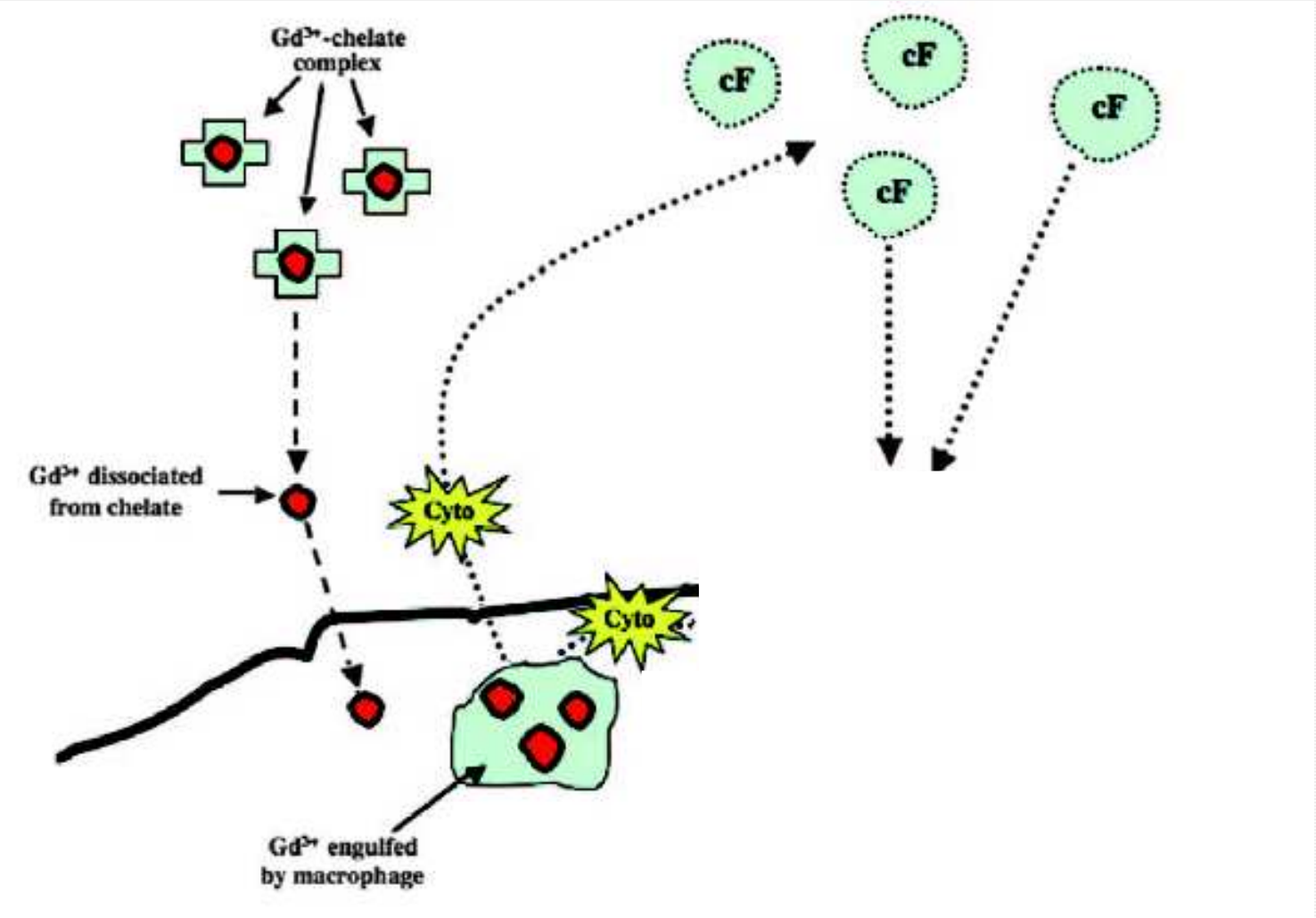

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN



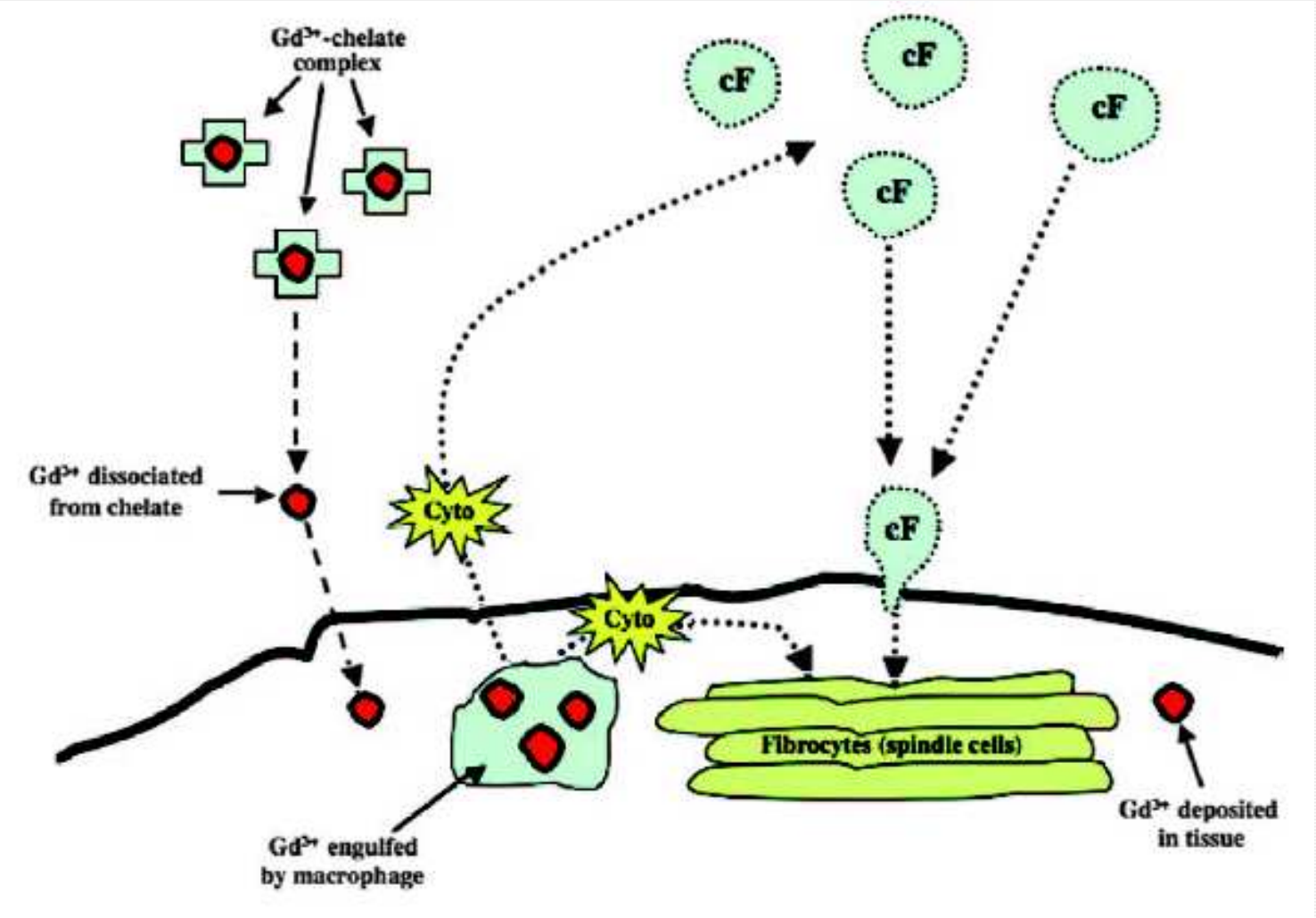

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN



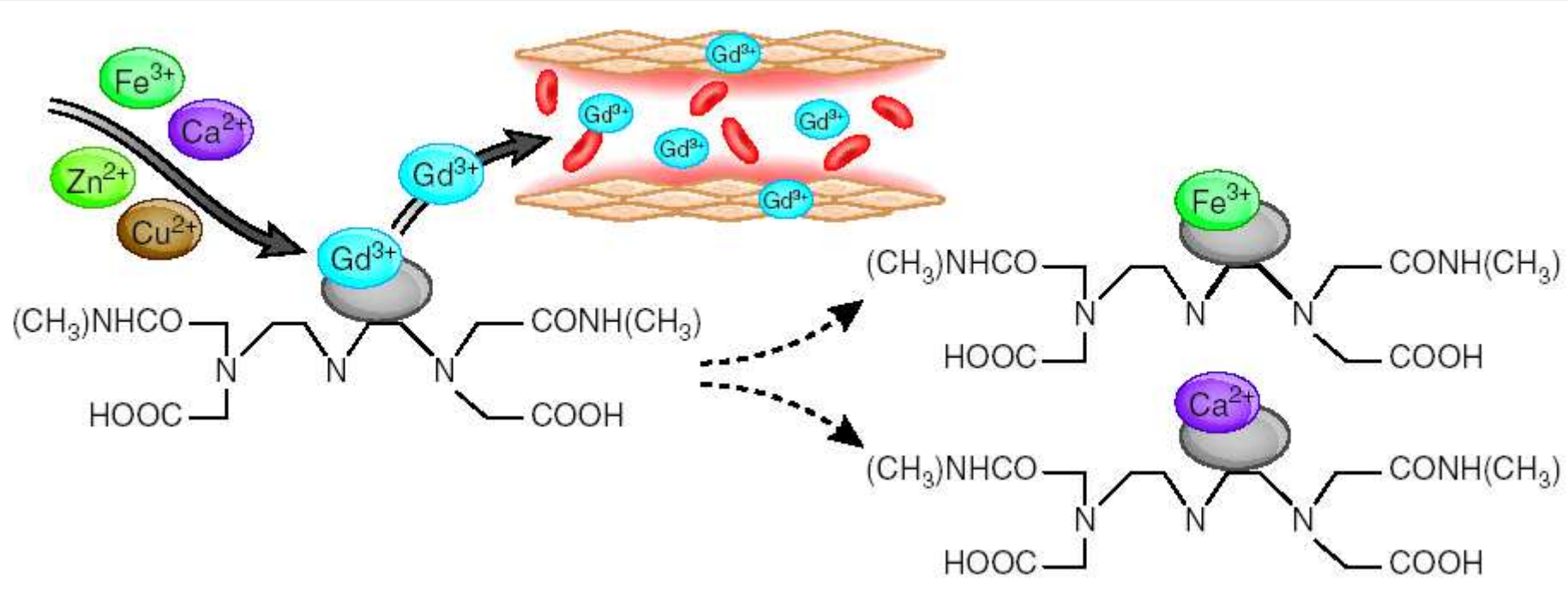

*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Mécanisme supposé de la FSN




*(Perazella MA. Clin J Am Soc Nephrol 2007: 2: 200-203)*

# Rôle de la transmetallation




*(Perazella MA. Clin J Am Soc Nephrol 2008; 3: 649-651)*

# Facteurs de risque de la FSN :

1. **IRC sévère ou IRA**

   **- 90 % des patients en dialyse**

   **- 10 % avec DFG < 30 ml/min ou en IRA**

# Facteurs de risque de la FSN :

2. **Dose de Gd**

   **- 90 % des cas ont reçu une quantité de Gd > dose standard**

   **- nb de cas chez des hémodialysés en fonction de la dose**

| | n total | n avec FSN |
|---|---|---|
| dose standard | 94 | 0 |
| dose supérieure | 210 | 12 |

*(Broone DR, AJR 2007; 188: 586)*

# Facteurs de risque de la FSN :

## 3. Type de Gd

**A ce jour, 467 cas de FSN rapportés à la FDA**

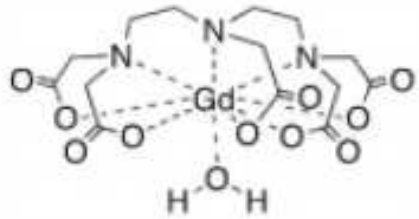
http://www.icnfdr.org)

*Table 4.* FDA MedWatch reported cases[a]

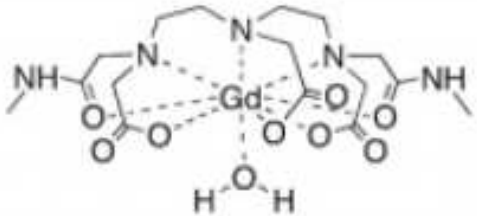
| Agent Generic Name | Agent Trade Name | Cases | Cases Alone | No. of Exposures |
|---|---|---|---|---|
| Gadoteridol | ProHance | 9 | 1 | 1.66 ± 1.66 |
| Gadobenate dimeglumine | MultiHance | 10 | 2 | 1.40 ± 0.70 |
| Gadoversetamide | OptiMARK | 20 | 8 | 1.10 ± 0.45 |
| Gadodiamide | Omniscan | 283 | 246 | 1.28 ± 1.07 |
| Gadopentetate dimeglumine | Magnevist | 125 | 96 | 2.70 ± 2.43 |

[a]FDA, Food and Drug Administration.

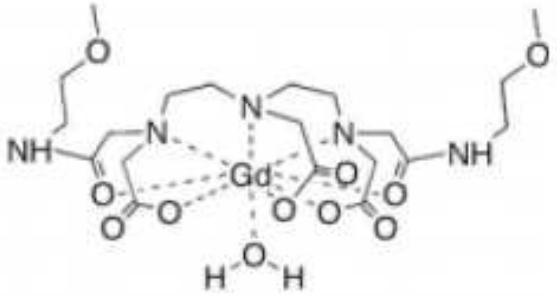
*(Reilly RF. Clin J Am Soc Nephrol 2008; 3: 747-751 )*



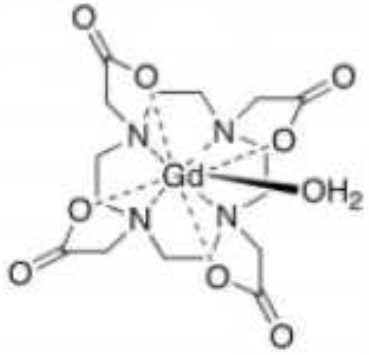
Gd-DTPA (Magnevist®)
Gadopentetate dimeglumine
linear ionic



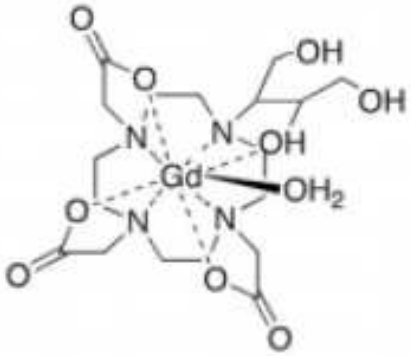
Gd-DTPA-BMA (Omniscan®)
Gadodiamide
linear neutral



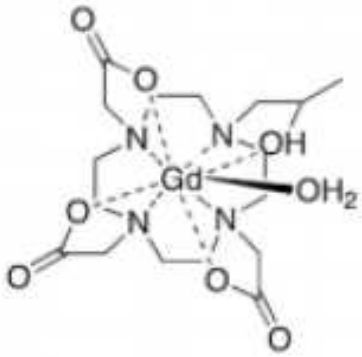
Gd-DTPA-BMEA (OptiMARK®)
Gadoversetamide
linear neutral



Gd-DOTA (Dotarem®)
Gadoterate meglumine
cyclic ionic



Gd-DO3A-butrol (Gadovist®)
Gadobutrol
cyclic neutral



Gd-HPDO3A (ProHance®)
Gadoteridol
cyclic neutral

*(Aime S, J Magn Reson Imaging 2009; 30: 1259 )*

# Aucun cas de FSN chez des dialysés (!) recevant une dose adéquate de Gadotérate (Dotarem ®)

- **Etude Pro-FINEST (France) :**
  - **préciser l'incidence de la FSN chez le dialysé chronique**
  - **après un examen IRM avec ou sans injection de Gd**

- **A 29 mois**
  - **571 patients, dont 50 % ont reçu Gd (Dotarem : 89 %)**
  - **aucun cas de FSN**

**(Amet S. pour le goupe ICAR, comm. Congrès SN 2012, Genève; http://dx.doi.org/10.1016/j.nephro.2012.07.049)**

# Facteurs de risque de la FSN :

4. **Etat « pro-inflammatoire » :**

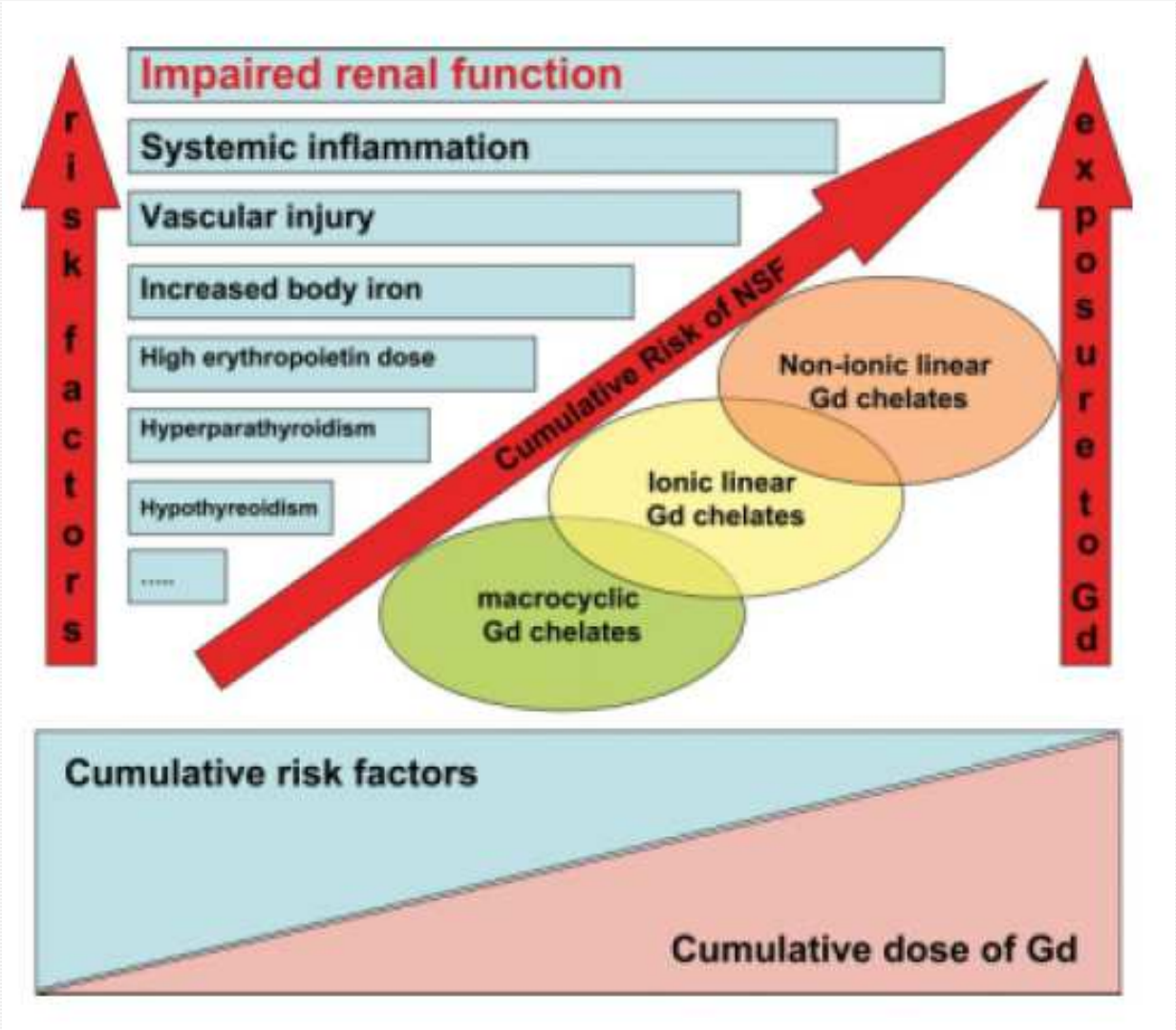
   **- intervention chirurgicale**

   **- infection**

   **- complication vasculaire**

*(Sadowski EA, Radiology 2007; 243: 148 )*

# Ces facteurs de risque sont cumulatifs




*(Mayr M, J Magn Reson Imaging 2009; 30: 1289 )*

# Recommandations pour l'utilisation des chélates de Gd en IRM

1. Eviter l'administration de Gd chez les patients présentant une insuffisance rénale (aiguë ou chronique)
   - au stade de la dialyse ou
   - avec un DFG < 30 ml / min

2. Si l'administration de Gd paraît néanmoins justifiée chez certains patients appartenant à cette catégorie:
   - recourir à un chélate de Gd macrocyclique (tel que le Dotarem ®)
   - si le patient est en dialyse (HD ou DP), prévoir une séance d'hémodialyse immédiatement après l'injection de Gd et une autre dans les 24h

# La FSN : une saga pleine de leçons !

- **Deux rappels salutaires :**
  **- attention, chez le patient en IR sévère, aux substances à élimination exclusivement rénale**
  **- utilité de la pharmacovigilance**

- **Un stimulant pour la recherche :**
  **- contribution à la compréhension du mécanisme de la fibrose**
  **- accélération de la mise au point de méthodes d'IRM sans Gd**