

## **Publications de l'U 507 / U 845(VW) (Inserm) (1999-2011)**

**2011**

**BUSSONE G., NOEL L.H., MOUTHON L.**

Renal involvement in patients with systemic sclerosis.

*Néphrol. Théor.*, 7 (3), 192-199, 2011

(Services cités : Anatomie Pathologique, U845 (VW))

Scleroderma renal crisis is characterized by malignant hypertension and oligo-anuric acute renal failure. Scleroderma renal crisis occurs in 2 to 5% of patients with systemic sclerosis, particularly those with diffuse cutaneous systemic sclerosis in the first years of disease evolution. High-dose corticosteroid therapy (>15mg/d) is associated with an increased risk of scleroderma renal crisis. Patients present with prominent left heart failure and hypertensive encephalopathy. Renal failure can be associated with moderate proteinuria, without hematuria. Thrombotic microangiopathy is detected in 43% of the cases. Anti-RNA polymerase III antibodies are present in one third of patients with scleroderma renal crisis. In case of renal failure, iatrogenic or functional origin must be investigated, as well as crescentic glomerulonephritis associated with antineutrophil cytoplasm antibodies (ANCA) or thrombotic microangiopathy. Renal biopsy is not necessary to establish the diagnosis in typical forms of scleroderma renal crisis. However, it can help to evaluate the prognosis and it is recommended when clinical presentation of scleroderma renal crisis is unusual. The prognosis of scleroderma renal crisis dramatically improved with the use of angiotensin-converting enzyme (ACE) inhibitors. However, 5-year survival of patients who developed a scleroderma renal crisis is only 65%. The treatment relies on the early control of blood pressure with increasing doses of ACE inhibitors, in association with calcium channel blockers if necessary. In case of severe renal failure and/or hypertension, dialysis can help to quickly control the vascular overload and the blood pressure. Dialysis can be stopped in about half of cases. After 2 years on dialysis, eligible patients should be considered for renal transplantation. The prevention of scleroderma renal crisis lacks consensus. Corticosteroids and/or nephrotoxic drugs should be avoided in patients with diffuse cutaneous systemic sclerosis.

**MOUTHON L., BEREZNE A., BUSSONE G., NOEL L.H., VILLIGER P.M., GUILLEVIN L.**

Scleroderma renal crisis: a rare but severe complication of systemic sclerosis.

*Clin. Rev. Allergy Immunol.*, 40 (2), 84-91, 2011

(Services cités : U845 (VW))

Scleroderma renal crisis (SRC) is a major complication in patients with systemic sclerosis (SSc). It is characterized by malignant hypertension and oligo/anuric acute renal failure. SRC occurs in 5% of patients with SSc, particularly in the first years of disease evolution and in the diffuse form. The occurrence of SRC is more common in patients treated with glucocorticoids, the risk increasing with increasing dose. Left ventricular insufficiency and hypertensive encephalopathy are typical clinical features. Thrombotic microangiopathy is detected in 43% of the cases. Anti-RNA-polymerase III antibodies are present in one third of patients who develop SRC. Renal biopsy is not necessary if SRC presents with classical features. However, it can help to define prognosis and guide treatment in atypical forms. The prognosis of SRC has dramatically improved with the introduction of angiotensin-converting enzyme inhibitors (ACEi). However, 5

years survival in SSc patients who develop the full picture of SRC remains low (65%). SRC is often triggered by nephrotoxic drugs and/or intravascular volume depletion. The treatment of SRC relies on aggressive control of blood pressure with ACEi, if needed in combination with other types of antihypertensive drugs. Dialysis is frequently indicated, but can be stopped in approximately half of patients, mainly in those for whom a perfect control of blood pressure is obtained. Patients who need dialysis for more than 2 years qualify for renal transplantation.

**MURAKAMI I., OKA T., KUWAMOTO S., KATO M., HAYASHI K., GOGUSEV J., IMAMURA T., MORIMOTO A., IMASHUKU S., YOSHINO T.**

Tyrosine phosphatase SHP-1 is expressed higher in multisystem than in single-system Langerhans cell histiocytosis by immunohistochemistry.

*Virchows Arch.*, 459 (2), 227-234, 2011

(Services cités : U845 (VW))

Langerhans cell histiocytosis (LCH) is a proliferative disorder of Langerhans cell (LC)-like CD1a-positive cell (LCH cell) with unknown causes. LCH consists of two subtypes: single-system LCH (LCH-SS) with favorable prognosis and multisystem LCH (LCH-MS) with poor prognosis. LCH has been indicated as a neoplastic disorder from monoclonal characteristics of LCH cells. This study aimed to investigate an expression of tyrosine phosphatase SHP-1 in LCH, since its expression levels were variously reported in many tumors, overexpression in ovarian cancers (a candidate oncoprotein), and downregulation by methylation in gastric cancers, prostate cancers, malignant lymphomas, and leukemias (a putative tumor suppressor). By immunohistochemistry (IHC), the SHP-1 expression in LCs and LCH cells was compared in LCH (two subtypes: LCH-SS = 21, LCH-MS = 12), dermatopathic lymphadenopathy (DLA) (n = 9) and normal epidermal LCs (n = 3) near LCH lesion. IHC results were analyzed semiquantitatively using a Photoshop software. The mean intensity score (IS) of DLA, LCH-SS, LCH-MS, and LCs were 47, 100, 139, and 167 (in arbitrary unit), respectively. The IS had significant differences among LCH-SS, LCH-MS, and DLA ( $p < 0.01$ ). SHP-1 is expressed significantly higher in LCH-MS than in LCH-SS. SHP-1 can be a progression marker of LCH. SHP-1 is also useful for differential diagnosis between LCH in lymph nodes and DLA.

**2010**

**ERMAK N., LACOUR B., GOIRAND F., DRUEKE T.B., VICCA S.**

Differential apoptotic pathways activated in response to Cu-induced or HOCl-induced LDL oxidation in U937 monocytic cell line.

*Biochem. Biophys. Res. Commun.*, 393 (4), 783-787, 2010

(Services cités : Biochimie Générale, U845 (VW))

We compared the apoptotic mechanism involved in U937 human monocytic cell line in presence of oxidized low-density lipoproteins (oxLDL) obtained after treatment with hypochlorous acid (HOCl) or copper (Cu). Both types of oxLDL induced U937 apoptotic cell death via the mitochondrial pathway. In contrast to HOCl-oxLDL, Cu-oxLDL induced apoptosis via a caspase-independent mechanism, with no activation of pro-caspase-3, but via the release of apoptosis inducing factor (AIF) from mitochondria. The apoptotic program of the monocyte differs depending on the mode of LDL oxidation, based on differences in the oxidatively modified components of the two oxLDL types.

**FAKHOURI F., FREMEAUX-BACCHI V., NOEL L.H., COOK H.T., PICKERING M.C.**

C3 glomerulopathy: a new classification.

*Nat. Rev. Nephrol.*, 6 (8), 494-499, 2010

(Services cités : U845 (VW))

Several distinct pathological patterns of glomerular inflammation are associated with abnormal regulation of the complement system, specifically, with dysregulation of the alternative pathway of the complement system. However, these conditions share the pathological finding of complement C3 (C3) deposited within the glomerulus in the absence of substantial immunoglobulin. This finding has alerted us and others to the possible presence of genetic and acquired complement dysregulation in individual patients. This article summarizes our current understanding of the relationship between dysregulation of the complement system and glomerular inflammation. Here, we suggest that glomerular pathologies that are characterized by the isolated deposition of C3 could usefully be classified by the term C3 glomerulopathy. In our view, this classification would alert the pathologist and nephrologist to the importance of screening for acquired and genetic abnormalities in complement regulation. In the future, it could help to identify individuals who might benefit from therapeutic inhibition of the complement system.

**HAJJAR E., BROEMSTRUP T., KANTARI C., WITKO-SARSAT V., REUTER N.**

Structures of human proteinase 3 and neutrophil elastase--so similar yet so different.

*FEBS J.*, 277 (10), 2238-2254, 2010

(Services cités : U845 (VW))

Proteinase 3 and neutrophil elastase are serine proteinases of the polymorphonuclear neutrophils, which are considered to have both similar localization and ligand specificity because of their high sequence similarity. However, recent studies indicate that they might have different and yet complementary physiologic roles. Specifically, proteinase 3 has intracellular specific protein substrates resulting in its involvement in the regulation of intracellular functions such as proliferation or apoptosis. It behaves as a peripheral membrane

protein and its membrane expression is a risk factor in chronic inflammatory diseases. Moreover, in contrast to human neutrophil elastase, proteinase 3 is the preferred target antigen in Wegener's granulomatosis, a particular type of vasculitis. We review the structural basis for the different ligand specificities and membrane binding mechanisms of both enzymes, as well as the putative anti-neutrophil cytoplasm autoantibody epitopes on human neutrophil elastase 3. We also address the differences existing between murine and human enzymes, and their consequences with respect to the development of animal models for the study of human proteinase 3-related pathologies. By integrating the functional and the structural data, we assemble many pieces of a complicated puzzle to provide a new perspective on the structure-function relationship of human proteinase 3 and its interaction with membrane, partner proteins or cleavable substrates. Hence, precise and meticulous structural studies are essential tools for the rational design of specific proteinase 3 substrates or competitive ligands that modulate its activities.

**LOCATELLI F., ECKARDT K.U., MACDOUGALL I.C., TSAKIRIS D., CLYNE N., BURGER H.U., SCHERHAG A., DRUEKE T.B.**

Value of N-terminal brain natriuretic peptide as a prognostic marker in patients with CKD: results from the CREATE study.

*Curr. Med. Res. Opin.*, 26 (11), 2543-2552, 2010

(Services cités : [U845 \(VW\)](#), [N<sup>2</sup>©phrologie Adulte](#))

This study assessed plasma N-terminal prohormone brain natriuretic peptide (NT-proBNP) as a prognostic marker of cardiovascular risk in patients with chronic kidney disease stages 3-4 and anaemia treated with epoetin beta to two haemoglobin target ranges. Of 603 patients enrolled in the Cardiovascular Risk Reduction by Early Anaemia Treatment with Epoetin Beta (CREATE) trial (baseline creatinine clearance 15-35 mL/min; haemoglobin 11.0-12.5 g/dL), 291 were included in this sub-study. Patients received subcutaneous epoetin beta either immediately after randomisation (target 13.0-15.0 g/dL; Group 1), or after their haemoglobin levels had fallen < 10.5 g/dL (target 10.5-11.5 g/dL; Group 2). Chronic heart failure New York Heart Association class III-IV was an exclusion criterion. (ClinicalTrials.gov Identifier: NCT00321919) Cardiovascular event rates were higher in patients with baseline NT-proBNP > 400 vs. 400 pg/mL (39 vs. 13 events;  $p = 0.0002$ ). Dialysis was initiated in 68 vs. 42 patients with NT-proBNP > 400 vs. 400 pg/mL ( $p = 0.0003$ ). Amongst patients with NT-proBNP > 400 pg/mL, there was no significant difference between treatment groups in risk of cardiovascular events (HR = 0.57;  $p = 0.08$ ) or time to dialysis (HR = 0.65;  $p = 0.08$ ). The overall interpretation of this substudy is, however, limited by its relatively small sample size which, together with low clinical event rates, result in a lack of statistical power for some analyses and should be viewed as being hypothesis-generating in nature. In chronic kidney disease patients with mild-to-moderate anaemia, elevated baseline plasma NT-proBNP levels are associated with a higher risk of cardiovascular events and an accelerated progression towards end-stage renal disease.

**NIKOLOV I.G., JOKI N., VICCA S., PATEY N., AUCHERE D., BENCHITRIT J., FLINOIS J.P., ZIOL M., BEAUNE P., DRUEKE T.B., LACOUR B.**

Tissue Accumulation of Lanthanum as Compared to Aluminum in Rats with Chronic Renal Failure - Possible Harmful Effects after Long-Term Exposure.

*Nephron Exp. Nephrol.*, 115 (4), e112-e121, 2010

(Services cités : [Anatomie Pathologique](#), [Biochimie Générale](#), [U845 \(VW\)](#))

Background: Lanthanum (La) carbonate is a new treatment for hyperphosphatemia. We tested

the effects of oral La carbonate and aluminum hydroxide, respectively, on tissue accumulation and liver function in rats with chronic renal failure (CRF). Methods: Adult male non-CRF and CRF rats were randomly assigned to 3 groups receiving either standard diet (St.D), or the same diet supplemented with 3% La carbonate (non-CRF La vs. CRF La) or 3% aluminum hydroxide (non-CRF Al vs. CRF Al). Results: After 12 weeks, serum phosphorus was decreased in both CRF La and Al groups. Urinary La and Al excretion was increased in these two groups, and so was liver and bone La content, and liver Al content. Both total body and liver weight were decreased in CRF La and CRF Al rats. Liver cell proliferation was decreased in these groups, while plasma total alkaline phosphatases and alanine aminotransferase were increased. Hepatic total cytochrome p450 content was reduced in CRF La, but not in CRF Al rats. Conclusion: Long-term oral La overload in rats with CRF was associated with a decrease in liver (and total body) weight and mild alterations of liver function, as was Al overload, possibly as a consequence of trace element accumulation.

### **NOEL L.H.**

Systemic amyloidosis: practical diagnosis.

*Néphrol. Théor.*, 6 (2), 88-96, 2010

(Services cités : [U845 \(VW\)](#))

Amyloid deposits have particular ultrastructural appearance with 7 to 10 nm-diameter fibrils. Amyloid is defined by its tinctorial affinity, which includes Congo red positivity, which must polarize and produce apple-green birefringence. Immunohistochemical characterization allows to know the origin of amyloidosis (characterization, which must be performed on frozen tissue). This restatement is proposed to renal pathologists in indicating the traps of studying amyloidosis, the tissues performing for the diagnosis, the specific staining and the immunohistochemical characterization.

### **PELLETIER S., ROTH H., BOUCHET J.L., DRUEKE T., HANNEDOUCHE T., LONDON G., FOUQUE D.**

Mineral and bone status in French maintenance hemodialysis patients: a comparison of June 2005 and June 2008.

*Néphrol. Théor.*, 6 (1), 11-20, 2010

(Services cités : [U845 \(VW\)](#))

**INTRODUCTION:** Because of the high associated morbi-mortality, phosphate and calcium disorders remain a major therapeutic challenge for nephrologists. Previous studies showed that only few patients had serum calcium, phosphate and parathyroid hormone within Kidney-Disease Outcomes Quality Initiative (K/DOQI) targets. **PATIENTS AND METHODS:** The French calcium and phosphate observatory monitors mineral metabolism at local, regional and national level and its follow-up every six months since 2005. More than 200 nephrologists collected more than 9000 patients' data. We compared the results recorded in June 2005 with those collected in June 2008. **RESULTS:** As compared with June 2005, in June 2008 fewer patients were hypercalcemic according to the K/DOQI targets (-26.2%,  $p < 0.001$ ) and hyperphosphatemic (-16.5%,  $p < 0.001$ ), more patients were hypocalcemic (+45.5%,  $p < 0.001$ ) and hypophosphatemic (+8.8%,  $p < 0.02$ ). A greater number of patients had elevated serum PTH above 300ng/l (+17.6%,  $p < 0.001$ ) and fewer had a PTH lower than 150ng/l (-25.4%,  $p < 0.001$ ). Serum 25OH vitamin D level was 21.7+/-20.0microg/l in June 2008. Overall, 10.5% of patients met all three K/DOQI targets, an improvement compared with June 2005 (6.8%,  $p < 0.001$ ). Between 2005 and 2008, cinacalcet, lanthane carbonate and native vitamin D derivatives prescription increased whereas calcium-based phosphate binders, sevelamer-HCL and active vitamin D derivatives decreased. **CONCLUSION:** Despite a

significant improvement between 2005 and 2008, only few patients reach the three K/DOQI targets (10.5%) in 2008. The prospective biannual follow-up during three years will allow us to identify the impact of different treatments on calcium and phosphate metabolic control and patient's survival.

**PELLETIER S., ROTH H., BOUCHET J.L., DRUEKE T., LONDON G., FOUQUE D.**

Mineral and bone disease pattern in elderly haemodialysis patients.

*Nephrol. Dialysis Transplant.*, 25 (9), 3062-3070, 2010

(Services cités : U845 (VW))

**BACKGROUND:** Although many studies have recently addressed the mineral and bone disorder of chronic kidney disease (CKD-MBD), only limited information is available for elderly dialysis patients. **METHODS:** We prospectively collected serum phosphorus, calcium, parathyroid hormone (PTH), 25(OH) vitamin D, albumin, C-reactive protein, protein intake and CKD-MBD treatments in 9169 maintenance haemodialysis patients in France in June 2008. We then compared biological and treatment patterns in 3403 patients aged 75 or over to their younger counterparts. **RESULTS:** Elderly patients exhibited lower serum phosphorus and parathyroid hormone concentrations (-8 and -18%, respectively) but slightly higher corrected serum calcium levels (+2%) compared to patients aged below 75 years. Elderly patients had higher mean C-reactive protein, lower serum albumin levels and reduced protein intake. Calcium and non-calcium phosphate binders as well as cinacalcet usage and dosage were significantly reduced in elderly patients, with a trend towards lower active vitamin D derivatives usage. Elderly patients were better controlled according to the Kidney Disease Outcome Quality Initiative (K/DOQI) targets compared to patients aged below 75.

**Conclusion.** In this large 2008 cohort of elderly haemodialysis patients, it appears easier to control serum parameters of CKD-MBD as compared to younger dialysis patients. A better control of serum phosphorus was observed, with less phosphate binder and reduced cinacalcet dosage.

**2009**

**BARRETO F.C., BARRETO D.V., LIABEUF S., DRUEKE T.B. et MASSY Z.A.**

Effects of uremic toxins on vascular and bone remodeling.

*Semin. Dialysis*, 22 (4), 433-437, 2009 ; (Facteur d'Impact 2008: 2,671)

(Services cités : U845 (VW))

**BOLLEE G., NOEL L.H., SUAREZ F., ROYAL V., GILARDIN L., de SERRE N.P., EL GHOUL B., LESAVRE P., ALYANAKIAN M.A. et FAKHOURI F.**

Pauci-immune Crescentic Glomerulonephritis Associated With ANCA of IgA Class.

*Amer. J. Kidney Dis.*, 53 (6), 1063-1067, 2009 ; (Facteur d'Impact 2008: 3,981)

(Services cités : Hématologie Adulte, Néphrologie Adulte, U845 (VW), Laboratoire d'Immunologie)

**BOLLEE G., PATEY N., CAZAJOUS G., ROBERT C., GOUJON J.M., FAKHOURI F., BRUNEVAL P., NOEL L.H. et KNEBELMANN B.**

Thrombotic microangiopathy secondary to VEGF pathway inhibition by sunitinib.

*Nephrol. Dialysis Transplant.*, 24 (2), 682-685, 2009 ; (Facteur d'Impact 2008: 3,167)

(Services cités : Anatomie Pathologique, Néphrologie Adulte, U845 (VW))

**DRUEKE T.B. et MASSY Z.A.**

Beta2-microglobulin.

*Semin. Dialysis*, 22 (4), 378-380, 2009 ; (Facteur d'Impact 2008: 2,671)

(Services cités : U845 (VW))

**DRUEKE T.B. et RITZ E.**

Treatment of secondary hyperparathyroidism in CKD patients with cinacalcet and/or vitamin D derivatives.

*Clin. J. Amer. Soc. Nephrol.*, 4 (1), 234-241, 2009 ; (Facteur d'Impact 2008: 2,236)

(Services cités : Néphrologie Adulte, U845 (VW))

**DRUEKE T.B. et TOUAM M.**

Calcium balance in haemodialysis--do not lower the dialysate calcium concentration too much (con-part).

*Nephrol. Dialysis Transplant.*, 24 (10), 2990-2993, 2009 ; (Facteur d'Impact 2008: 3,568)

(Services cités : U845 (VW))

**ECKARDT K.U., SCHERHAG A., MACDOUGALL I.C., TSAKIRIS D., CLYNE N., LOCATELLI F., ZAUG M.F., BURGER H.U. et DRUEKE T.B.**

Left ventricular geometry predicts cardiovascular outcomes associated with anemia correction in CKD.

*J. Amer. Soc. Nephrol.*, 20 (12), 2651-2660, 2009 ; (Facteur d'Impact 2008: 7,505)

(Services cités : U845 (VW))

**FERREIRA A., SARAIVA M., BEHETS G., MACEDO A., GALVAO M., D'HAESE P. et DRUEKE T.B.**

Evaluation of bone remodeling in hemodialysis patients: serum biochemistry, circulating cytokines and bone histomorphometry.

*J. Nephrol.*, 22 (6), 783-793, 2009 ; (Facteur d'Impact 2008: **1,211**)

(Services cités : Néphrologie Adulte, U845 (VW))

**FUKAGAWA M. et DRUEKE T.B.**

CKD-MBD as a systemic disorder.

*Bone*, 45 (Suppl.1), S1, 2009 ; (Facteur d'Impact 2008: **3,966**)

(Services cités : Néphrologie Adulte, U845 (VW))

**IVANOVSKI O., NIKOLOV I.G., JOKI N., CAUDRILLIER A., PHAN O., MENTAVERRI R., MAIZEL J., HAMADA Y., NGUYEN-KHOA T., FUKAGAWA M., KAMEL S., LACOUR B., DRUEKE T.B. et MASSY Z.A.**

The calcimimetic R-568 retards uremia-enhanced vascular calcification and atherosclerosis in apolipoprotein E deficient (apoE<sup>-/-</sup>) mice.

*Atherosclerosis*, 205 (1), 55-62, 2009 ; (Facteur d'Impact 2008: **4,601**)

(Services cités : Biochimie Générale, U845 (AE), U845 (VW))

**JEAN G., LAFAGE-PROUST M.H., MASSY Z.A. et DRUEKE T.B.**

Guidelines for vitamin D prescription in dialysis patients.

*Néphrol. Théor.*, 5 (6), 520-532, 2009 ; (Facteur d'Impact 2008: **X**)

(Services cités : Néphrologie Adulte, U845 (VW))

**JOKI N., NIKOLOV I.G., CAUDRILLIER A., MENTAVERRI S.R., MASSY Z.A. et DRUEKE T.B.**

Effects of calcimimetic on vascular calcification and atherosclerosis in uremic mice.

*Bone*, 45 (Suppl.1), S30-S34, 2009 ; (Facteur d'Impact 2008: **3,966**)

(Services cités : U845 (VW))

**MASSY Z.A., STENVINKEL P. et DRUEKE T.B.**

The role of oxidative stress in chronic kidney disease.

*Semin. Dialysis*, 22 (4), 405-408, 2009 ; (Facteur d'Impact 2008: **2,671**)

(Services cités : U845 (VW))

**MCCARRON D.A. et DRUEKE T.B.**

Salt wars.

*Nephrol. Dialysis Transplant.*, 29 (3), 1063, 2009 ; (Facteur d'Impact 2008: **3,167**)  
(Services cités : Néphrologie Adulte, U845 (VW))

**MEBAREK S., ERMAK N., BENZARIA A., VICCA S., DUBOIS M., NEMOZ G., LAVILLE M., LACOUR B., VERICEL E., LAGARDE M. et PRIGENT A.F.**

Effects of increasing docosahexaenoic acid intake in human healthy volunteers on lymphocyte activation and monocyte apoptosis.

*Br. J. Nutr.*, 101 (6), 852-858, 2009 ; (Facteur d'Impact 2008: **2,339**)

(Services cités : Biochimie Générale, U845 (VW))

**MORICEAU S., KANTARI C., MOCEK J., DAVEZAC N., GABILLET J., GUERRERA I.C., BROUILLARD F., TONDELIER D., SERMET-GAUDELUS I., DANEL C., LENOIR G., DANIEL S., EDELMAN A. et WITKO-SARSAT V.**

Coronin-1 is associated with neutrophil survival and is cleaved during apoptosis: potential implication in neutrophils from cystic fibrosis patients.

*J. Immunol.*, 182 (11), 7254-7263, 2009 ; (Facteur d'Impact 2008: **6,068**)

(Services cités : Pédiatrie Générale, U845 (VW), U845 (AE))

**NIKOLOV I.G., MOZAR A., DRUEKE T.B. et MASSY Z.A.**

Impact of Disturbances of Calcium and Phosphate Metabolism on Vascular Calcification and Clinical Outcomes in Patients with Chronic Kidney Disease.

*Blood Purif.*, 27 (4), 350-359, 2009 ; (Facteur d'Impact 2008: **1,822**)

(Services cités : U845 (VW))

**ROUMENINA L.T., JABLONSKI M., HUE C., BLOUIN J., DIMITROV J.D., DRAGON-DUREY M.A., CAYLA M., FRIDMAN W.H., MACHER M.A., RIBES D., MOULONGUET L., ROSTAING L., SATCHELL S.C., MATHIESON P.W., SAUTES-FRIDMAN C., LOIRAT C., REGNIER C.H., HALBWACHS-MECARELLI L. et FREMEAUX-BACCHI V.**

Hyperfunctional C3 convertase leads to complement deposition on endothelial cells and contributes to atypical hemolytic uremic syndrome.

*Blood*, 114 (13), 2837-2845, 2009 ; (Facteur d'Impact 2008: **10,432**)

(Services cités : U845 (VW))

**VANHOLDER R., ABOU-DEIF O., ARGILES A., BAURMEISTER U., BEIGE J., BROUCKAERT P., BRUNET P., COHEN G., de DEYN P.P., DRUEKE T.B., FLISER D., GLORIEUX G., HERGET-ROSENTHAL S., HORL W.H., JANKOWSKI J., JORRES A., MASSY Z.A., MISCHAK H., PERNA A.F., RODRIGUEZ-PORTILLO J.M., SPASOVSKI G., STEGMAYR B.G., STENVINKEL P., THORNALLEY P.J., WANNER C. et WIECEK A.**

The role of EUTox in uremic toxin research.

*Semin. Dialysis*, 22 (4), 323-328, 2009 ; (Facteur d'Impact 2008: **2,671**)

(Services cités : Néphrologie Adulte, U845 (VW))

**VANHOLDER R., ARGILES A., BEIGE J., BRUNET P., DRUEKE T.B., FLISER D., HERGET-ROSENTHAL S., HORL W.H., JORRES A., PERNA A., RODRIGUEZ-PORTILLO M., SPASOVSKI G., STEGMAYR B., STENVINKEL P., WANNER C., WIECEK A. et MASSY Z.A.**

Conservative treatment of the uremic syndrome.

*Semin. Dialysis*, 22 (4), 449-453, 2009 ; (Facteur d'Impact 2008: 2,671)

(Services cités : Néphrologie Adulte, U845 (VW))

**WESTENFELD R., SCHAFFER C., KRUGER T., HAARMANN C., SCHURGERS L.J., REUTELINGSPERGER C., IVANOVSKI O., DRUEKE T., MASSY Z.A., KETTELER M., FLOEGE J. et JAHNEN-DECHENT W.**

Fetuin-A Protects against Atherosclerotic Calcification in CKD.

*J. Amer. Soc. Nephrol.*, 20 (6), 1264-1274, 2009 ; (Facteur d'Impact 2008: 7,111)

(Services cités : U845 (VW))

**WITKO-SARSAT V., DANIEL S., NOEL L.H. et MOUTHON L.**

Neutrophils and B lymphocytes in ANCA-associated vasculitis.

*APMIS*, 127 (Suppl.), 27-31, 2009 ; (Facteur d'Impact 2008: 1,316)

(Services cités : U845 (VW))

**YAHIAOUI Y., JABLONSKI M., HUBERT D., MOSNIER-PUDAR H., NOEL L.H., STERN M., GRENET D., GRUNFELD J.P., CHAUVEAU D. et FAKHOURI F.**

Renal Involvement in Cystic Fibrosis: Diseases Spectrum and Clinical Relevance.

*Clin. J. Amer. Soc. Nephrol.*, 4 (5), 921-928, 2009 ; (Facteur d'Impact 2008: 2,236)

(Services cités : Néphrologie Adulte, U845 (VW))

2008

**BOYER O., NOEL L.H., BALZAMO E., GUEST G., BIEBUYCK N., CHARBIT M., SALOMON R., FREMEAUX-BACCHI V. et NIAUDET P.**

Complement factor H deficiency and posttransplantation glomerulonephritis with isolated C3 deposits.

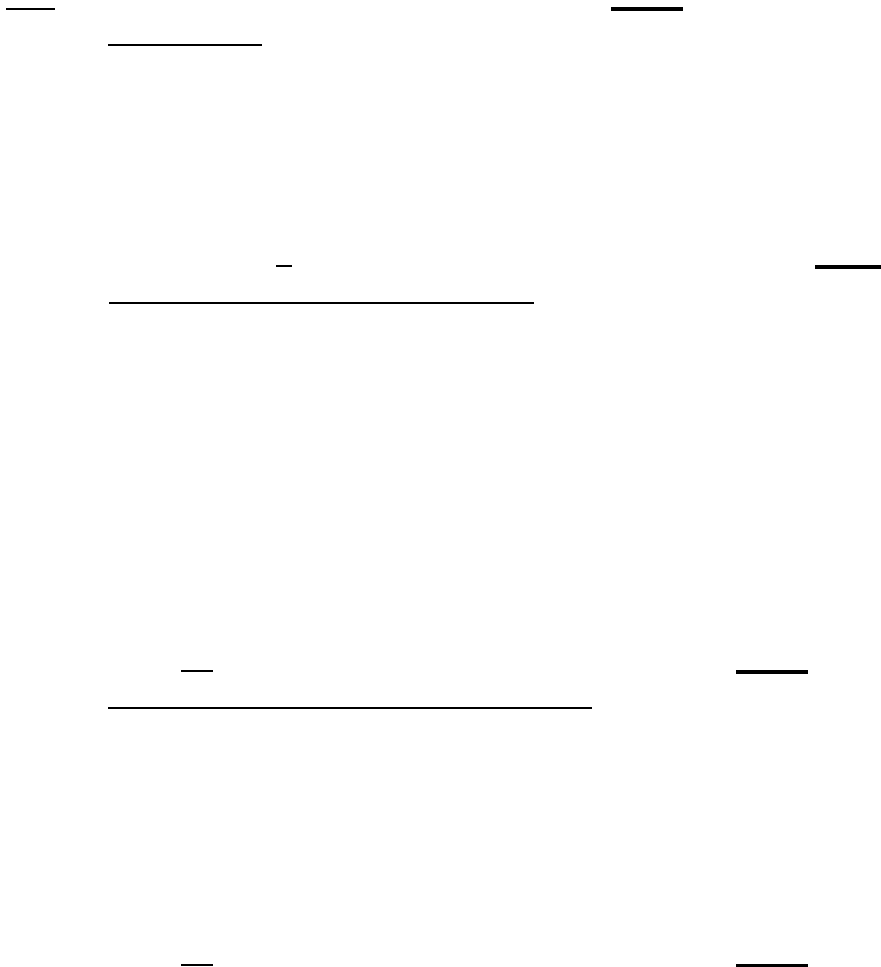
*Amer. J. Kidney Dis.*, 51 (4), 671-677, 2008 ; (Facteur d'Impact : **3,981**)

(Services cités : Néphrologie Pédiatrique, U845 (VW))

**CAMOUS L., MELANDER C., VALLET M., SQUALLI T., KNEBELMANN B., NOEL L.H. et FAKHOURI F.**

Complete remission of lupus nephritis with rituximab and steroids for induction and rituximab alone for maintenance therapy.

*Amer. J. Kidney Dis.*, 52 (2), 346-352, 2008 ; (Facteur d'Impact : **3,981**)



(Services cités : Néphrologie Adulte, U845 (VW))

**DANIEL L., DOU L., BERLAND Y., LESAVRE P., HALBWACHS-MECARELLI L. et DIGNAT-GEORGE F.**

Circulating microparticles in renal diseases.

*Nephrol. Dialysis Transplant.*, 23 (7), 2129-2132, 2008 ; (Facteur d'Impact : **3,167**)

(Services cités : Néphrologie Adulte, U845 (VW))

**DRUEKE T.B.**

Arterial Intima and Media Calcification: Distinct Entities with Different Pathogenesis or All the Same ?

*Clin. J. Amer. Soc. Nephrol.*, 3 (6), 1583-1584, 2008 ; (Facteur d'Impact : **2,236**)

(Services cités : Néphrologie Adulte, U845 (VW))

**DRUEKE T.B.**

Is parathyroid hormone measurement useful for the diagnosis of renal bone disease ?

*Kidney Int.*, 73 (6), 674-676, 2008 ; (Facteur d'Impact : **4,922**)

(Services cités : Néphrologie Adulte, U845 (VW))

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