1. Schier RW, Wang W. Acute renal failure: definitions, diagnosis, pathogenesis and therapy. J Clin Invest 2004;114:5-14.
2. Liaño F, Pascual J. Fracaso renal agudo: concepto, epidemiología y aspectos socioeconómicos. En: Liaño F, Pascual J (eds). Fracaso renal agudo. Barcelona: Masson SA, 2000;3-13.
3. Tovar JL, Pascual J, Liaño F. Fracaso renal agudo. En: Liaño F, Pascual J (eds). Barcelona: Masson SA, 2000;103-25

A. Rodríguez[1], R. Guerra[2], D. Silva[1]
Nefrology department.[1] General Hospital of Fuerteventura. [2] Gran Canaria Island University Hospital. Las Palmas de Gran Canaria, Spain
Correspondence: Alejandra Rodríguez
Servicio de Nefrología. Hospital General de Fuerteventura. Las Palmas de Gran Canaria, Spain.
jairarodriguez@hotmail.com

Henoch-Schönlein nephritis triggered by *Salmonella enteritidis* infection

Nefrologia 2010;30(3):374
doi: 10.3265/Nefrologia.pre2010.Mar.10261

Dear Editor,
Henoch-Schönlein purpura (HSP) is a common type of vasculitis in children. Renal involvement is frequent and not always benign. Prognostic factors have been recently reviewed by García et al. on your Journal, older age and relapses being related to a poorer renal prognosis.[1]

We present a case of 5-years old boy who was admitted to our hospital with a history of fever-chills, vomiting, abdominal cramping and diarrhoea. He received IV and subsequent oral rehydration and his conditions improved in three days. A stool culture yielded *Salmonella enteritidis*. After an interval of one week by the onset of gastrointestinal symptoms he developed symmetrical purpuric papules and plaques at the lower extremities and arthralgia of the tibio-tarsal joints. Two days later appeared frank hematuria lasting one day only and followed by microhematuria with mild proteinuria. Blood pressure was always normal. Among laboratory investigations creatinine was 77 µmol/l, platelet count, C3 and C4 levels were normal, antinuclear antibody and rheumatoid factor were absent, serum IgA levels were increased for his age (232 mg/dl). Characteristic skin manifestations, joint involvement and hematuria led us to the diagnosis of HSP nephritis (HSPN). Purpura and arthritis resolved in three weeks. Nephritis had a benign evolution. After six months the boy was normotensive without residual microhematuria nor proteinuria and his renal function was normal.

Pathogenesis of HSPN has very recently reviewed.[2] High levels of galactose-deficient IgA1 (Gd-IgA1) has been found in children with HSPN, but not in HSP affected patients without nephritis. Gd-IgA1 seems to have a pivotal role both in HSPN and IgA nephritis. Gd-IgA1 is recognised by anti-glycan antibodies and form large molecular immune complexes. Their deposit in renal mesangium is thought to initiate glomerular inflammation.

Many factors may activate IgA1 overproduction and subsequent disease: a list that includes various infective agents and medications has been published in a review by Rai et al., but it does not contain *Salmonella enteritidis*.[3] Afterwards a case of HSP nephritis in a 50-year-old woman with *Salmonella typhi* septicaemia has been described.[4] At our knowledge our case of HSP nephritis induced by *Salmonella enteritidis* is the first described in literature. This pathogen, very common in children, should be included in the number of infectious agent that can trigger HSPN.

1. Lucas García J, Álvarez Blanco O, Sanahuja Ibáñez MJ, Ortega López PJ, Zamora Martí I. Evolución de la nefropatía de Schönlein-Henoch en pacientes pediátricos. Factores pronósticos. Nefrologia 2008;28(6):627-32.
2. Lau KK, Suzuki H, Novak J, Wyatt RJ. Pathogenesis of Henoch-Schönlein purpura nephritis. Pediatr Nephrol 2010;25(1):179.
3. Rai A, Nast C, Adler S. Henoch-Schönlein Purpura Nephritis. J Am Soc Nephrol 1999;10:2637-44.
4. Youmbissi TJ, Malik TQ, Ajit Kumar S, Rafi A, Al Khursanny AI, Karkar A. Henoch-Schönlein nephritis and *Salmonella typhi* septicaemia. Nephrol Dial Transplant 2001;16(5):1081-2.

A. Venuta[1], F. Balli[2]
[1] Paediatric Department. Dipartimento materno-infantile. Modena University Hospital. Policlinico di Modena. Modena. Italy. [2] Paediatric Department. Modena University Hospital. Modena. Italy.
Correspondence to: Andrea Venuta
Paediatric Department.
Dipartimento materno-infantile.
Modena University Hospital.
Policlinico di Modena. Modena. Italy.
andrea.venuta@unimore.it

Acute rejection of pancreatic grafts

Nefrologia 2010;30(3):374-6
doi: 10.3265/Nefrologia.pre2010.Apr.10313

Dear Editor,
Simultaneous renal and pancreatic transplantation is the best treatment option for young patients (< 45 years) who have type 1 diabetes mellitus (DM) and advanced stages of diabetic nephropathy, in the absence of other cardiovascular risk factors, as long as the transplantation waiting time is not unreasonably prolonged.[1]

Due to the characteristics of this type of donors and recipients, patient and graft survival is similar to living donor transplant cases.[2] The pancreatic graft in this case has a survival rate of around 70% five years after transplantation.

We report a case of a 47-year-old man who underwent simultaneous pancreas and kidney transplantation and came in for follow-up; he was asymptomatic except for mild discomfort in the area

of the pancreatic graft. He was sent from the outpatient clinic for admission in order to obtain laboratory data related to pancreatic injury.

His medical history included hypertension and type 1 DM with diabetic retinopathy and nephropathy; he had received Continuous Ambulatory Peritoneal Dialysis (CAPD) for 18 months. Ischaemic heart disease had been ruled out and he received pancreatic and kidney transplants 7.5 years ago, both of which are currently functional.

The donor was a 21-year-old man who died of multiple trauma, with the same blood group as the patient, and no HLA compatibility among those that were tested. The pancreas was transplanted with exocrine drainage into the duodenum. Both grafts were initially functional, the patient did not require insulin starting in the first 24 hours and plasma creatinine was 0.8mg/dl on the third day. Thymoglobulin induction was performed during the first 4 days after transplantation, and then suspended due to lymphopoenia and thrombocytopoenia. Tacrolimus was started, which, along with MMF and prednisone, became the patient's maintenance immunosuppressive therapy.

On the current admission the patient has good blood glucose control and blood pressure. On physical examination, the patient had no pathological findings except for discomfort on palpation of the epigastric area. The main laboratory data obtained were: plasma creatinine 1mg/dl, amylase 440mg/dl, lipase 403 U and tacrolimus levels of 3.1ng/ml. Ultrasonography was performed which showed slight oedema of the pancreatic graft and resistive index (RI) within normal limits, without abnormalities in the renal graft.

The clinical symptoms were interpreted as being consistent with acute rejection of the pancreatic graft, probably related to low levels of the anti-calcineurin agent. In the first days after admission, the tacrolimus levels were adjusted to 10ng/ml and the patient was treated with 4 boluses of 6-methylprednisolone, with reduction but not normalisation of pancreatic enzymes. Treatment was initiated with thymoglobulin (3mg/kg) via a jugular catheter. This medication was poorly tolerated and the patient developed fever, myalgias, gastrointestinal intolerance, and general worsening, which improved with symptomatic treatment. The pancreatic enzyme levels normalised after three doses of thymoglobulin, and renal function remained stable. Prophylactic treatment with septrim and oral valganciclovir was started at the time of discharge.

Acute rejection is 1.5 to 2 times more common in combined pancreas-kidney transplantation than in simple renal transplantation. It also occurs later and is more often resistant to steroids.[3] However, graft loss due to a pancreatic rejection is more uncommon in the case of combined transplantation than in either of the other two methods (pancreas after kidney and pancreas alone).[4] Pancreatic rejection may or may not be associated with renal graft rejection and it can occur synchronously or asynchronously. Confirmation by renal biopsy is sufficient when it occurs simultaneously.

Pancreatic rejection initially occurs against acinar cells, such that the islets of Langerhans continue functioning at first. Thus, uncontrolled blood glucose is a late event in the course of acute pancreatic rejection, when more than 90% of the graft has been affected.

In combined transplantation of the pancreas and kidney, renal graft rejection is more common and more serious than pancreatic rejection, and therefore monitoring of serum creatinine is usually used to detect rejection of both organs. However, up to 15% of cases of pancreatic rejection occur without any damage to the renal graft.[5]

In the case of enteric drainage, pancreatic function cannot be monitored via amylasuria, so it is important to pay attention to more nonspecific data, such as serum pancreatic amylase and lipase levels. Ultrasound-guided pancreatic biopsy does not require laparotomy, but it carries a 3 to 15% risk of non-diagnosis due to sampling failure.[6] The diagnostic alternative is laparoscopic biopsy. Information provided by Doppler ultrasound or CT is often non-specific.

1. Gruessner RW, Dunn DL, Gruessner AC, Matas AJ, Najarian JS, Sutherland DE. Recipient risk factors have an impact on technical failure and patient and graft survival rates in bladder-drained pancreas transplants. Transplantation 1994;57(11):1598-606.
2. Gaston RS, Basadonna G, Cosío FG, Davis CL, Kasiske BL, Larsen J, et al. Transplantation in the diabetic patient with advanced chronic kidney disease: a task force report. Am J Kidney Dis 2004;44(3):529-42.
3. Douzdjian V, Abecassis MM, Corry RJ, Hunsicker LG. Simultaneous pancreas-kidney versus kidney-alone transplants in diabetics: increased risk of early cardiac death and acute rejection following pancreas transplants. Clin Transplant 1994;8(3 Pt 1):246-51.
4. Gruessner AC, Sutherland DE. Pancreas transplant outcomes for United States (US) and non-US cases as reported to the United Network for Organ Sharing (UNOS) and the International Pancreas Transplant Registry (IPTR) as of June 2004. Clin Transplant 2005;19(4):433-55.
5. Reddy KS, Davies D, Ormond D, Tuteja S, Lucas BA, Johnston TD, et al. Impact of acute rejection episodes on long-term graft survival following simultaneous kidney-pancreas transplantation. Am J Transplant 2003;3(4):439-44.
6. Kuhr CS, Davis CL, Barr D, McVicar JP, Perkins JD, Bachi CE, et al. Use of ultrasound and cystoscopically guided pancreatic allograft biopsies and transabdominal renal allograft biopsies: safety and efficacy in kidney-pancreas transplant recipients. J Urol 1995;153(2):316-21.

A. Guitián Pena[1], A. Alonso Hernández[2]
[1] Nephrology Department. [2] Nephrology Department and Renal Transplant Unit. University Hospital Commplex of A Coruña. La Coruña, Spain.
Correspondence: Ana Guitián Pena
Servicio de Nefrología. Complejo Hospitalario Universitario de A Coruña, Spain.
Canceliña, 1, 4.º A. 15011 La Coruña, Spain.
ana.guitian.pena@sergas.es

Intestinal pseudo-obstruction due to lanthanum carbonate

Nefrologia 2010;30(3):376
doi: 10.3265/Nefrologia.pre2010.Mar.10311

Dear Editor,
Currently, we have a wide range of options regarding the treatment of bone metabolism in haemodialysis.[1] Recently, even with limited time that sevelamer has been used to control hyperphosphataemia, many nephrologists have opted for lanthanum carbonate based largely on the fewer number of tablets required for this therapy.[2] The most frequent side effect of these drugs is manifested in the gastrointestinal tract, primarily affecting gastrointestinal transit, since they are binding compounds.

In our unit, after one year of treating hyperphosphataemia with lanthanum carbonate, our level of control may be considered acceptable in terms of the percentage of patients with phosphate levels less than 5.5. Tolerance has been good, in general, and we have radiologically confirmed the residual presence of this substance in the colon on more than one occasion, which is to be routinely expected and has been previously described.[3]

Here we present the case of a patient who presented with severe abdominal pain with intestinal paralysis, in whom lanthanum carbonate could not be excluded as a causal or contributing agent.

A 75-year-old man, diagnosed with ischaemic nephropathy on haemodialysis for the past 5 years, was admitted to the emergency room with pain in the right iliac fossa. The patient was afebrile, without vomiting, but did have constipation. On physical examination, there was absence of peristalsis and tenderness to palpation in the right iliac fossa. The laboratories were unremarkable (no leukocytosis, amylase, or lipase within the ranges adjusted to the degree of uraemia, etc.). Plain abdominal radiography showed remains of lanthanum carbonate in the colon, dilated loops of bowel, and, overall, a pseudobstructive pattern. Surgical intervention was decided upon for suspicion of an obstructed bowel loop, with a preoperative diagnosis of intestinal ischaemia. The patient underwent surgery during which no signs of mesenteric thrombosis were seen, bowel loops appeared normal, as well as the appendix and the abdominal environment.

Clearly, although we are unable to state anything conclusively, we must suggest a possible iatrogenic aetiology related to lanthanum ingestion, as previously reported, and the importance of remaining alert to the occurrence of processes similar to those described.

1. Block GA, Klassen PS, Lazarus JM, et al. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. J Am Soc Nephrol 2004;15:2208-18.
2. Martín de Francisco AL. ¿Debemos considerar el costo-efectividad de los distintos tratamientos al aplicar las recomendaciones sobre los captores (quelantes) de fósforo? Nefrologia 2008;28(2):129-34.
3. Cerny S, Kunzendorf U. Images in clinical medicine. Radiographic appearance of lanthanum. N Engl J Med 2006;355:1160.

A. Pobes Martínez de Salinas[1], I. Blanco Rodríguez[2], L. Quiñones Ortiz[1], A. Suárez Laurés[1]
[1] Nefrology and [2] Anaesthesia departments. Cabueñes Hospital. Gijón. Asturias, Spain.
Correspondence: Ana Suárez Laurés
Servicio de Nefrologia. Hospital Cabueñes. Gijón. Asturias, Spain.
luysquio@hotmail.com

Pregnancy and advanced chronic kidney disease

Nefrologia 2010;30(3):376-8
doi: 10.3265/Nefrologia.pre2010.Apr.10354

Dear Editor,
The ability to become and remain pregnant in patients with chronic kidney disease depends on its stage. In early stages of the disease, there are practically no differences from a normal pregnancy.[1] On the other hand, the difficulties that pregnancy poses to renal replacement therapy (RRT) are well known, and better outcomes have been described in patients who have undergone renal transplantation.[2] However, the presence of advanced chronic kidney disease (stage 3-4) and pregnancy is an uncommon occurrence. Here we present the progression and treatment of a pregnant woman with stage 4 chronic kidney disease, which is especially unusual.

The patient is a 23-year-old female with epilepsy and chronic renal failure secondary to interstitial nephropathy. She was not hypertensive and presented, at one month of gestation, with the following laboratory findings: Hb: 13.1g/dl, Cr: 2.7mg/dl, urea: 101mg/dl, Ca: 9.1, P: 3.8mg/dl, HCO3: 19mmol/l, PTH: 480pg/ml, estimated glomerular filtration rate (eGFR) (MDRD-4): 21ml/min/1.73m^2, proteinuria: 2.23g/day; other tests without significant abnormalities. Weight 45.8kg and blood pressure (BP) 113/75mmHg. The progression of laboratory values can be seen in Figure 1. Clinical progression, BP control, presence of urea less than 100mg/dl or serum creatinine less than 4mg/dl, and ultrasound follow-up were established as the parametres to be assessed at the beginning of the RRT. These values