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An atypical pneumonia in a renal transplant patient

We report the case of a 27-year-old man who suffered from end-stage renal disease secondary to medullary cystic kidney disease and who received a kidney transplant in November 2007. The post-transplant immunosuppressive regimen consisted of methylprednisolone, tacrolimus and mycophenolate mofetil. One year later, the patient presented with a non-productive cough and pain at the right side of the chest. He did not have fever. Physical examination revealed breath sounds reduction and rales over the right lung. On blood examination, C-reactive protein (CRP) was elevated at 52.3 mg/L (normal range <5 mg/L), and white blood cell (WBC) count was $13 \times 10^3/mm^3$ (normal range 3.6–9.6 × $10^3/mm^3$) with $9.2 \times 10^3/mm^3$ neutrophils (normal range 1.4–6.7 × $10^3/mm^3$). Serum creatinine was 177 µmol/L (2.0 mg/dL) (normal range 0.5–1.5 mg/dL), and urea was 8.5 mmol/L (51 mg/dL) (normal range 15–40 mg/dL). Tacrolimus level was in the therapeutic range. Chest radiography showed the presence of an opacity at the right lung. Empirical treatment with intravenous amoxicillin clavulanate and clarithromycin was initiated. After a few days, the patient's symptoms resolved, and CRP and WBC count returned to normal. Antibiotic therapy was continued orally. However, the infiltrate remained radiologically unchanged.

Two months later, our patient was readmitted with general malaise. Laboratory tests at that time showed CRP of 145.5 mg/L, urea of 70 mg/dL, creatinine of 2.5 mg/dL and WBC count of $6.9 \times 10^3/mm^3$. Chest computed tomography (CT) scan showed no reduction of the pulmonary infiltrate. Therapy with amoxicillin clavulanate was reinitiated. Two sputum cultures and one blood culture yielded growth of Gram-positive coccobacilli, later identified as *Rhodococcus equi*. After susceptibility testing, therapy was switched to oral doxycycline and ciprofloxacin [1], and the patient could be discharged. A week later, he was readmitted for an *Escherichia coli* sepsis of unknown origin. Based on advanced literature study [1,2], it was decided to treat the patient with oral ciprofloxacin (2 × 500 mg/day) and rifampicin (2 × 450 mg/day) for at least 6 months. A CT-guided biopsy of the lesion was performed 7 months after initial diagnosis and showed the presence of granulomatous inflammation without necrosis. Additional stains and tissue cultures remained negative, namely for mycobacteria.

Currently, after 1 year of therapy, the patient's symptoms have completely resolved, and chest CT scan shows limited atelectasis but no active pulmonary infiltrate anymore. Immunosuppressive therapy has not been changed during the whole course of the infection.

R. equi is a bacterium identified in soil and animals [2,3], but since our patient had a negative anamnesis for direct exposure, it still remains unknown how he acquired this type of infection.

This case illustrates the difficulty to establish the diagnosis of causative pathogens in immunocompromised patients, since clinical presentation can be very atypical and the spectrum of possible pathogens is extended. In conclusion, *R. equi* is a rare pathogen that should be considered in the differential diagnosis of atypical pneumonia in transplant patients.

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Should we recommend precautions during a hantavirus endemic?

Sir,

The number of notified hantavirus infections in southwestern Germany increased considerably in the beginning of the year 2007; therefore, the German health institutions put out a press release in the areas with the highest incidence in the southwestern part (Baden-Wuerttemberg) to establish precautions in the case of a possible contact with rodents [1].

During this significant endemic burst of infections, a 43-year-old male patient presented in Baden-Wuerttemberg with fever (>39°C), low back pain and acute renal failure. He told the doctors at the emergency department that he most likely had a hantavirus because he had cleaned his garden cabin 10 days before the presentation in the emergency department, and he had observed several rodents in his garden in the past. Because of the mentioned press release of an increased number of hantavirus infections with a broad discussion in the public, he followed some of the recommended precautions of the gov-

ernmental health institutions and used a disposable respirator of category 1 (FFP1) during the cleaning procedure [2]. On clinical examination, the patient was afebrile, his blood pressure was 135/90 mmHg, and the initial laboratory investigations showed a low platelet count (65×10^9/L), normal electrolytes and an elevated serum creatinine (4.8 mg/dL). Hantavirus-specific IgM antibodies (Puumala) were strongly positive and IgG slightly positive. The infection was self-limiting, with a maximum serum creatinine of 4.8 mg/dL and a normalization of the kidney function tests in the follow-up.

Hantaviruses comprise one of five genera of the family *Bunyaviridae*, and the natural reservoirs are rodent-borne pathogens [3]. Human infection occurs most commonly through the inhalation of infectious, aerosolized saliva or rodent faeces. In Europe and Asia, hantavirus infections can present with a haemorrhagic fever and renal syndrome (HFRS). The main hantavirus species in Germany is Puumala, and their main reservoir is bank voles (*Myodes glareolus*), which predominantly live in temperate forests of Western and Central Europe or in the boreal forests (taiga) in Northern Europe. It is reported that, in endemic areas, the incidence of hantavirus infections among humans is related to the size of the bank vole population and prevalence of the virus [4,5]. Every 3–4 years, there are peaks in the bank vole population, and this may result in an increase in human infection rates. In Germany, laboratory-confirmed symptomatic hantavirus infections are mandatorily notifiable since 2001. In the period from 1 January 2007 to 3 June 2007, 526 symptomatic hantavirus cases were reported to the Robert Koch Institute. In the comparable time period of the previous years, the mean number of reported cases was only 71 (minimum in 2006 with 17 cases and maximum in 2005 with 171 cases), this is a seven times higher incidence, and the vast majority of cases (77%, 405 cases) were reported from Baden-Wuerttemberg [2]. The Robert Koch Institute and a recent overview of Clement *et al*. from the Hantavirus Reference Center in Belgium explained this significant increase of hantavirus infection by an extraordinarily mild winter 2006/2007 with no snow cover on the ground and, additionally, an abundant supply of beech mast in the autumn of 2006 (the so-called 'mast hypothesis') [5]. This could have resulted in an early increase in the bank vole population because of higher rodent survival rates, and the breeding could have started earlier. Furthermore, during the mild winter 2006/2007, humans may have been more exposed to rodents because of increased outdoor activities [1]. During this extraordinary increase, the issue of infection prevention was discussed in the public media (press and television), and the government health institutions sent bulletins to the physicians in the endemic areas, especially in Baden-Wuerttemberg [6].

The prevention–recommendation of the Robert Koch Institute in Germany in a high-risk situation was

(i) reduce dust development in contaminated areas, e.g. by moistening;

(ii) use disposable gloves and, if dust development is not avoidable, use disposable respirators;

(iii) if you have observed rodents, ventilate closed rooms for 30 mins and eventually use disposable respirators and gloves; and

(iv) use disinfection fluids.

The Center for Disease Control and Prevention (CDC) recommendations in the USA are more detailed; one reason could be the more serious clinical manifestation by the different hantavirus species in the USA [3,7].

Our well-informed patient considered the planned cleaning of his garden cabin as a high-risk situation (observed rodents in his garden in the past) and tried to prevent an infection by wearing a disposable respirator and gloves as proposed by the health institutions [2,7]. This prevention failed, and he developed, after the usual incubation period, the feared infection.

This case raises several questions: Did our patient use incorrect preventative measures, or not enough of the preventative measures needed? What is the value of the proposed prevention recommendations? Should we recommend precautions during a hantavirus endemic?

The most likely explanation in the failed prevention in our patient is the missing ventilation of his garden cabin for 30 mins before starting to clean. Ventilation of a confined space (hut, cabin, attic, etc.) with a presumed presence of rodents (e.g. during the past winter) is the most effective, easiest and least expensive protective measure. The floors must be sprayed with water and bleach; afterwards, it is recommended to flush with water and to avoid sweeping in order to avoid breathing rodent excreta.

The value of the proposed FFP1 has to be discussed because in theory, protection against a biohazard requires at least a FFP2 mask with a high-efficiency particulate absorption or arrestance (HEPA) filter. HEPA filters remove at least 99.97% of airborne particles 0.3 μm in diameter. Although an individual virus particle ranges in size from 0.005 to 0.1 μm, viruses generally only survive to travel through the air as part of larger particles (0.3 μm or larger), for example, attached to mucous particles. FFP1 masks are not 100% effective in protecting against aerosolized viruses, as apparently proven in this case. The use of FFP2 masks in the general public, however, is deemed to expensive and impractical.

Up to now, it is difficult to give exact recommendations. On the one hand, the incidence of a severe hantavirus is still low, and hysteric precautions during all leisure activities will not be helpful, especially because gardening in urban areas and exercising/recreational activities in the forest (like jogging) showed no increased risk in a case-control study [8]. On the other hand, a well-informed society can try to avoid high-risk situations by simple and cheap measures [9], and hopefully, the infection rates can be reduced: avoid camping or sleeping on grounds with many rodent burrows, turn your back to the wind when working on such grounds or when cutting wood in the forest, ventilate indoor locations with signs of rodent manifestations, and after ventilation, wet mop floors with a bleach solution.

In summary, in order to establish more detailed prevention recommendations, the nephrology community has

to learn more about risk factors and detailed infection route circumstances.

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High risk of chronic kidney disease: results of the screening during World Kidney Day 2010

The aim of World Kidney Day (WKD) is to raise awareness of the importance of the kidneys to the overall health and to reduce the frequency and impact of kidney disease and its associated health problems worldwide. The objective of the 2010 WKD was to highlight that diabetes and high blood pressure are key risk factors for developing chronic kidney disease (CKD).

We therefore organized an anonymous screening in the lobby of our hospital during WKD. The screened population was composed of passers-by (employees, visitors of hospitalized patients and outpatients) of the University Hospital of the Free University of Brussels. They were invited to have their blood pressure (BP) and blood glucose measured by trained nurses and under standardized conditions. We also gathered information on personal and familial history of diabetes (DM), hypertension (HT) and CKD. Participants were asked about their smoking habits and how they estimate their actual weight (normal, overweight or obese). Educative information regarding causes and prevention of CKD was distributed.

Hypertension was defined as a BP of at least 140 and/or 90 mmHg. Controlled BP was defined as a BP <140 and <90 mmHg. Diabetes and impaired glucose tolerance (IGT) were defined as a blood glucose level of at least 200 mg/dL and between 140 and 200 mg/dL, respectively, in patients with no known diabetes.

In total, 325 people were examined (Table 1), of these 56% were women. The most frequently self-reported risk factor for developing CKD was excess weight, followed by HT and smoking. Excess weight was significantly more prevalent in men than in women. Self-reported diabetes was present in 6.2% of the studied persons. Significantly more men belonged to the older age group. Women had a lower systolic BP and had more frequently a controlled BP, when treated.

The prevalence of self-reported hypertension was 20%, but the prevalence of hypertension increased to 54% if we also took into account the 108 patients with a high BP who were unaware of an elevated BP. The prevalence is comparable with commonly reported values worldwide especially in the Belgian adult population [1–4]. Also, the percentage of patients (43%) unaware of their hypertensive status were very similar to previously reported values [4]. Of the 63 pa-

Table 1. Characteristics of participants

	All subjects (n = 325)	Men (n = 99)	Women (n = 184)	P
n (%)[a]				
Hypertension	66 (20.3%)	24 (24.2%)	30 (16.3%)	NS
Diabetes	20 (6.2%)	7 (7.1%)	10 (5.4%)	NS
Excess weight	78 (24%)	27 (27.3%)	44 (23.9%)	NS
Smokers	64 (19.7%)	17 (17.2%)	35 (19.0%)	NS
Age >50 years	137 (42.2%)	54 (54.5%)	67 (36.4%)	<0.02
Kidney disease	7 (2.2%)	1 (1%)	5 (2.7%)	NS
Family history, DM	102 (31.4%)	28 (28.3%)	66 (35.9%)	NS
Family history, KD	84 (25.8%)	23 (23.2%)	54 (29.3%)	NS
Mean (SD)				
SBP (mmHg)	138 (21)	142 (20)	135 (20)	<0.01
DBP (mmHg)	82 (13)	83 (12)	82 (13)	NS
PP (mmHg)	56 (16)	59 (17)	54 (16)	0.01
Glycaemia (mg/dL)	107 (30)	106 (30)	106 (28)	NS
BMI (kg/m^2)	25.5 (4.7)	25.9 (3.9)	25.2 (5.1)	NS
<20	27 (8.5%)	5 (5.1%)	19 (10.6%)	<0.01
20–<25	146 (45.8%)	36 (36.4%)	87 (48.6%)	
25–<30	104 (32.6%)	48 (48.5%)	46 (25.7%)	
>30	42 (13.2%)	10 (10.1%)	27 (15.1%)	
SBP target	173 (54.7%)	42 (42.9%)	110 (60.1%)	<0.02
DBP target	235 (74.4%)	67 (68.4%)	139 (75.9%)	NS
BP target	161 (50.9%)	39 (39.8%)	101 (55.2%)	<0.03

P is the difference between men and women.
[a]Self-reported condition.