CASE REPORT

Ethambutol and isoniazid induced severe neurotoxicity in a patient undergoing continuous ambulatory peritoneal dialysis

Meijun Si, Huiqun Li, Yanru Chen, Hui Peng

Nephrology Division, The Third Affiliated Hospital of Sun Yat-sen University, Guangzhou, Guangdong, China

Correspondence to
Dr Hui Peng,
pengh@mail.sysu.edu.cn

Accepted 27 April 2018

SUMMARY

Ethambutol (EMB) and isoniazid (INH) are the first-line antituberculosis (anti-TB) drugs. However, their neurotoxicity could cause adverse effect and the patients with end-stage renal disease are especially vulnerable due to the reduction in renal drug clearance. Here, we report a 36-year-old man receiving peritoneal dialysis developed progressive paralysis in lower extremities, vision loss and hoarseness 4 months after anti-TB treatment with INH, EMB and rifapentine because of concomitant pulmonary tuberculosis. A diagnosis of EMB/INH-induced peripheral neuropathy, retrobulbar neuritis and laryngoparalysis was made. The patient's neuropathy gradually improved 2 years after discontinuation of EMB/INH. Since EMB and INH may cause simultaneously severe and complex multineuropathy in dialysis patients, their adverse effects should be closely supervised in dialysis patients.

BACKGROUND

Patients with ESRD are at increased risk for *Mycobacterium tuberculosis* infection due to immunodeficiency resulting from functional abnormalities within the neutrophils, reduced T and B cell function and compromised monocyte and monocyte-derived dendritic cell function.^{1–3} Meanwhile, they are vulnerable to the toxic effects of antituberculosis (TB) drugs due to an impaired ability to excrete toxic substances. An increased incidence of side effects is induced by isoniazid (INH) and ethambutol (EMB).^{4,5} A study from Taiwan indicated that renal disease is a risk factor for optic neuropathy induced by EMB.⁶ However, the concurrence of peripheral neuropathy and retrobulbar neuritis resulting from antituberculosis therapy has seldom been reported in the general population or in patients with comorbid kidney disease. Here, for the first time we report a case of a patient undergoing continuous ambulatory peritoneal dialysis (CAPD) that sequentially manifested peripheral neuropathy, optic neuritis and laryngoparalysis induced by INH and EMB.

CASE PRESENTATION

A 36-year-old Chinese male was hospitalised due to general weakness and poor appetite. He had no history of diabetes mellitus, hypertension or infectious diseases including tuberculosis and viral hepatitis. He denied any exposure to heavy metal

toxicities. On physical examination, the body weight was 49 kg, body mass index 19.38 kg/m² and body temperature 98°F. Neurological examination revealed normal myodynamia and muscular tension, and pathological reflexes were negative. Laboratory findings revealed haematuria and massive proteinuria (urine protein +++, red blood cell count: 464/μL), severe acidosis (CO₂ levels: 7.3 mmol/L), azotaemia (blood urea nitrogen levels: 243.5 mg/dL, creatinine levels: 33.9 mg/dL), hyperphosphataemia (phosphorus levels: 13.2 mg/dL), severe anaemia (red blood cell count: 1.93×10¹² /L, haemoglobin levels: 54 g/dL), hyperparathyroidism (intact parathyroid hormone levels: 810.98 pg/mL) and low levels of serum albumin (35.5 g/L). Ultrasonography revealed shrinkage of the bilateral kidneys (right kidney: 71 mm×30 mm, left kidney: 61 mm×31 mm). The patient was diagnosed with chronic kidney disease (CKD) stage 5, chronic glomerulonephritis (most probably), renal anaemia and secondary hyperparathyroidism. Then haemodialysis was initiated.

One week after hospitalisation, the patient developed a cough with occasional sputum and an intermittent fever that primarily manifested during the night with temperatures fluctuating around 100.4°F and a highest recorded temperature of 102.6°F. Bilateral fine crackles were found on upper lungs. Chest X-rays and chest CT scan showed bilateral infiltrating lesions indicative of an acute TB infection. Sputum-smear microscopy and culture were performed for three times on separate days and the bacteriological results were negative. There was no evidence of extrapulmonary TB. The patient was diagnosed with infiltrating pulmonary TB and anti-TB treatment was initiated with INH (200 mg/day), EMB (15 mg/kg body weight on alternate days) and rifapentine (600 mg/week). On the day of haemodialysis, all the medications were administered after the treatment of haemodialysis. Two months later, haemodialysis treatment was switched to CAPD (2 L×4/day) by request of the patient.

The patient maintained regular follow-up visits at the outpatient clinic after discharge from the hospital. Because his chest X-ray did not show significant improvement after 2 months of treatment, the doses of the anti-TB regimen were adjusted in by pulmonologists as follows: INH (300 mg/day), EMB (15 mg/kg body weight/day) and rifampin (450 mg/day). One month after the dose adjustment, a chest

To cite: Si M, Li H, Chen Y, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223187

Figure 1 MRI of the spine reveals a mild degeneration of the cervical vertebra (A), while thoracic (B) and lumbar spine (C) MRI scans are without obvious abnormality.

X-ray revealed amelioration of the bilateral infiltrating TB and absorption of the pleural effusion. However, after 4 months of anti-TB therapy, the patient developed progressive weakness in his lower extremities and was hospitalised again with a chief complaint of weakness in the lower extremities.

DIFFERENTIAL DIAGNOSIS

The patient did not have a history of trauma and denied drug or alcohol abuse, contact with toxic heavy metals or a history of diabetes. Neurological examination showed muscle strength with a grade of 3 in the proximal lower extremities and 2 in the distal lower extremities, hypermyotonia and hyperreflexia in lower extremities, positive patellar clonus and ankle clonus and positive Kerning and Lasegue's signs. Hypoesthesia, hypalgesia and hypoesthesia were found below the knees. The weakness in the lower extremities gradually progressed to paraplegia and at the same time, the patient complained of numbness in his upper extremities along with a decline in vision. On laboratory findings, HIV test and toluidine red unheated serum test were negative; serum albumin level was 25.7 g/L. An electromyogram revealed peripheral nerve injury in upper and lower limbs (both sensory and motor nerve involved). Prolonged F wave latency in the bilateral tibial nerves was also detected. MRI of the cervical spine revealed mild degeneration of the cervical vertebra, while the thoracic and lumbosacral spine MRI was negative ([figure 1](#)). Besides, brain MRI scan was negative ([figure 2](#)) as was a cerebrospinal fluid (CSF) examination ([table 1](#)). All laboratory results ruled out an infection of the central nervous system, TB-associated transverse myelitis, Devic's disease and HIV-associated or syphilis-associated complications. The absence of contact with heavy metal ruled out heavy metal poisoning and raised suspicion that anti-TB drugs induced the peripheral neuropathy and optic nerve damage. However, this does not explain the positive

Figure 2 MRI scan of the brain shows centrum ovale (A), basal ganglia (B) and optic (C) nerves are without obvious abnormality.

Table 1 Cerebrospinal fluid tests

Tests	Results	Normal range
Pressure (cm H ₂ O)	12	8–18
Colour	Colourless	Colourless
Appearance	Transparent	Transparent
Globin	Negative	Negative
Red blood cell	None	None
White blood cell ($\times 10^6/L$)	1	0–8
Total protein (g/L)	0.4	0.15–0.4
Chlorine (mg/dL)	446.0	428.9–457.3
Glucose (mg/dL)	46.9	45.1–70.3
Ziehl-Neelsen stain	Negative	Negative
Tuberculosis antibody	Negative	Negative
Adenosine deaminase (U/L)	2	0–5
Ink stain	Negative	Negative

upper motor neuron findings; thus, other concurrent neurological disorders such as Wernicke syndrome resulting from vitamin B1 deficiency and subacute combined degeneration of spinal cord associated with vitamin B12 deficiency were suspected.

TREATMENT

Subsequently, all anti-TB drugs were discontinued and the supplements vitamin B1 (1 mg/ kgbody weight/day; intramuscular), pyridoxine (1 mg/ kgbody weight/day; intravenous), mecobalamin (2 mg/day), memantine (10 mg/day) and ganglioside (20 mg/day) were prescribed. A new anti-TB regimen of levofloxacin (500 mg, once every other day) and rifapentine (600 mg/week) was initiated 2 weeks after discontinuation of INH and EMB.

OUTCOME AND FOLLOW-UP

There was no immediate improvement of the symptoms in the patient after withdrawal of INH and EMB. His vision deteriorated to the perception of light, and he subsequently experienced intermittent dizziness, hoarseness and laryngoparalysis. Fundoscopic examination revealed blurred borders in both optic disks and a haemorrhage at the superior temporal part of the right optic disk; a visual field examination could not be performed because he was incapable of cooperating. Bilateral visual acuities were demonstrated by finger counting at 0.5 m and a diagnosis of EMB-induced retrobulbar neuritis was considered. Laryngoscopy examination identified glottic dysrhythmism and an association with EMB-induced laryngoparalysis was suspected.

Two months after the discontinuation of INH and EMB, the patient was able to ambulate for a maximum of 20 steps, and his visual symptoms improved to the extent that he was capable of reading the headlines of newspapers. Meanwhile, he no longer complained of hoarseness or any symptoms of laryngoparalysis. The neurological signs also turned negative. Chest X-ray showed the bilateral infiltrating TB was significantly ameliorated. Two years after the discontinuation of INH and EMB, his eyesight and muscle strength had improved to near baseline. The patient still maintains a regular follow-up schedule.

DISCUSSION

Only a few investigations have evaluated the side effects of anti-TB drugs in patients with renal failure.^{4 5 7–9} Cases of irreversible EMB-induced ocular toxicity or INH-associated peripheral neuropathy have been separately reported in chronic dialysis patients.^{8 9} Here, a case of severe neurological side effects induced

by anti-TB drug therapy is reported in a peritoneal dialysis patient with pulmonary TB. This is the first report on the succession of peripheral neuropathy, retrobulbar neuritis and laryngoparalysis induced by INH and EMB in peritoneal dialysis patients.

EMB is a first-line anti-TB drug mainly used to prevent the emergence of rifampin resistance. Nearly 80% of EMB is eliminated unchanged by the kidney,¹⁰ but in patients with compromised renal function, the clearance of EMB significantly decreases and its half-life is prolonged following the usual dosage of 15 mg/kg.¹¹ One recent study reported a 41% decrease in serum ethambutol concentration during dialysis in a patient using contemporary High-Flux Hemodialyzer.¹² A pharmacokinetic study of one CAPD patient found that 35.3% of EMB is eliminated by peritoneal dialysis.¹³

Retrobulbar neuritis is the most severe side effect associated with EMB administration, but its manifestation varies among individuals and may include progressive blurred vision and decreased colour perception. Patients may originally complain of abnormal colour perception, typically manifesting as red-green colour changes, central scotoma and optic atrophy.^{14 15} It is generally thought that EMB induces optic neuropathy in a dose-dependent manner. According to a study by Leibold,¹⁶ the incidence of ocular toxicity is significantly greater at higher dosages (>35 mg/(kg.d)) relative to lower dosages (<30 mg/(kg.d)). Guidelines from British Thoracic Society recommend a dosage regimen of 15–25 mg/kg three times a week (max of 2.5 g) for EMB in patients with stages 4 and 5 CKD who are not on dialysis,¹⁷ while WHO recommends a dose of 15 mg/kg three times per week for patients with renal failure or severe renal insufficiency.¹⁸ Recommendations for EMB regimen in peritoneal dialysis patients are not suggested by these guidelines. In the present case, the residual renal function was 1.45 mL/min and a dosage regimen prescribed by the nephrologist of 750 mg EMB on alternate days was adjusted to 750 mg/day by the pulmonologist 2 months after the beginning of anti-TB therapy. This particular regimen may contribute to the accumulation of EMB even though EMB can be partially eliminated by peritoneal dialysis.¹³ An increased dosing interval, rather than reducing EMB dosage, appears to be the best method with which to attain a balance between efficacy and safety. Alternative treatments such as moxifloxacin or gatifloxacin may be considered when there are concerns about EMB toxicity because the sterilising activity of these agents is proven to be at least equivalent to EMB.¹⁹

Some studies have found EMB-associated visual impairment to be reversible¹⁶ but other studies have obtained controversial results.^{15 20 21} Permanent visual losses have been reported in chronic dialysis patients^{4 7 9} and age, hypertension and renal diseases are considered to be risk factors for EMB-induced optical neuropathy.⁶ Following the initiation of EMB treatment, the regular monitoring and early detection of visual impairments are cost-effective. Most guidelines recommend a routine visual acuity assessment before the administration of EMB but this assessment is no longer recommended during follow-up visits.^{22 23} Other guidelines suggest baseline vision evaluation, including visual acuity and red-green colour perception tests, as well as regular visual testing during the course of EMB treatment in patients with renal failure.^{24 25} As with the present patient who exhibited a high risk for EMB-induced adverse effects, routine ophthalmic examination regarding optic neuropathy and the proper education of patients regarding potential side effects may assist with the early detection and prevention of irreversible visual impairments. In addition, optic neuropathy was reported as a rare complication of vitamin B12 deficiency.²⁶ Therefore,

we should also consider the possible effect of metabolic disorder on patient's optic neuropathy.

INH is an important component of anti-TB drug regimens. This agent is metabolised into a less active compound by n-acetyltransferase in the liver and then eliminated by the kidneys. Pharmacokinetic studies on haemodialysis patients have found that only 9.2% of INH is recovered in dialysate, which suggests that hepatic metabolism is the primary mechanism for the clearance of INH.¹² Peritoneal dialysis is responsible for 1.3% of all INH clearance; the concentration of INH in the peritoneal fluid after 6 hour of dwell time reaches 53.3% of its simultaneous serum concentration.²⁷

INH causes axonal degeneration by having antagonistic effects on pyridoxine, which results in neurotoxicity most frequently manifesting as peripheral neuropathy with paresthesia and burning in the lower extremities; patients rarely complain of weakness or upper extremity involvement. Neurological examination identified an impaired sensation to light touch, pain and temperature, as well as reduced Achilles tendon reflexes.²⁸ However, in the present case, the patient initially complained of weakness in the lower extremities while the upper extremities gradually became affected. Further neurological findings such as hypermyotonia and hyperreflexia of both lower extremities and positive patellar clonus and ankle clonus suggested the involvement of upper motor neurons which cannot be explained by the complication of peripheral neuropathy. His upper motor neuron signs turned negative after the withdrawal of anti-TB agents as well as the adding of neurotrophic treatment including supplements of vitamin B1 and B12. The deficiency of vitamin B12 could result in sensory abnormalities and ataxia associated with loss of sensation and in some cases even progress to severe myasthenia, spasticity, clonus, paraplegia.²⁹ Thus, the possible existence of concurrent neurological disorders resulting from metabolic abnormalities, especially vitamin B12 deficiency and subacute combined degeneration of spinal cord should be carefully inspected.

INH-associated neuropathy is thought to be dose dependent. Guidelines for the management of TB in patients with CKD suggested a usual daily dose for patients with stages 4 and 5 CKD and those on haemodialysis while the recommendation for peritoneal dialysis patients is not available.^{17 18 30} Only one study has suggested that no adjustment is necessary for peritoneal dialysis patients.²⁷

Pyridoxine deficiency is well documented in patients with chronic renal failure (CRF) primarily due to impairments in pyridoxine metabolism by uraemic toxins. Thus, to prevent pyridoxine deficiency-induced neuropathy, 8.2 mg/day supplemental pyridoxine is recommended for haemodialysis patients and 4.1 mg/day is recommended for peritoneal dialysis patients and patients with CRF.³¹ In patients with CRF who are taking INH, a pyridoxine antagonist, several guidelines for pulmonary tuberculosis recommend preventive treatment with pyridoxine along with INH,^{31–33} and the dosage of pyridoxine should be higher. However, because a megadose of pyridoxine can lead to severe and irreversible demyelinating polyneuropathy, excessive doses should not be administered as a preventive therapy.²⁸ However, there is no recommended dosage for pyridoxine in peritoneal dialysis patients who are treated with INH. Siskind *et al*⁸ recommended 100 mg/day pyridoxine supplementation as a routine prophylaxis against INH toxicity in patients undergoing haemodialysis. The clearance of pyridoxine during peritoneal dialysis is very low, being only 8.8% of urea clearance.³⁴ In the present case, the intravenous supplementation of pyridoxine (1 mg/kg/day) was sufficient to reverse INH-induced peripheral neuropathy, which was likely due to slower pyridoxine removal during peritoneal dialysis

than during haemodialysis. Besides, malnutrition increases the risk of INH neuropathy and is common among the dialysis population.²⁸ The present case manifested malnutrition (albumin levels: 25.7 g/L) on his second hospitalisation, which may have contributed to the onset of neuropathy associated with INH administration.

In conclusion, patients with CKD have a high risk of developing active TB and are susceptible to the toxic effects of anti-TB drugs.^{8 9 35–37} Clinicians should be aware of the side effects of anti-TB treatment, especially neurological complications. An appropriate dosage regimen based on the pharmacokinetic and metabolic characteristics of drugs for dialysis patients is important for efficacious treatment and the prevention of adverse events. The regular monitoring of potential side effects, the prompt discontinuation of anti-TB drugs and intensified neurotrophic therapy are usually effective for addressing neurotoxicity in peritoneal dialysis patients during anti-TB treatment.

Learning points

- Patients undergoing peritoneal dialysis (PD) are at increased risk of developing ethambutol and isoniazid-induced neurotoxicity, including peripheral neuropathy, retrobulbar neuritis and laryngoparalysis, and even the concurrence of these neuropathies.
- The recommendations for management of tuberculosis (TB) in PD patients are lacked based on the current established guidelines. Dosage regimen of anti-TB drugs should be adjusted based on the pharmacokinetic and metabolic characteristic of medication in dialysis patients.
- For PD patients receiving ethambutol, routinely monitoring of symptoms related to ethambutol-induced ocular toxicity should be warranted to avoid severe or even irreversible vision impairment.
- Concurrent metabolic abnormalities could impact neurological disorders in PD patients and thus needed to be evaluated and corrected promptly.

Contributors HP revised the article to the submitted version; responsible for the authenticity and accuracy of the article and supervised the diagnosis and treatment of the patient. MS drafted and assisted in revising the article and involved in the patient's care. HL summarised the examinations and treatment of the case report and followed up the patient and recorded the prognosis of the patient. YC participated in the patient's care and assisted in the literature review.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- Girndt M, Sester U, Sester M, et al. Impaired cellular immune function in patients with end-stage renal failure. *Nephrol Dial Transplant* 1999;14:2807–10.
- Gibbons RA, Martinez OM, Garovoy MR. Altered monocyte function in uremia. *Clin Immunol Immunopathol* 1990;56:66–80.
- Lim WH, Kireta S, Leedham E, et al. Uremia impairs monocyte and monocyte-derived dendritic cell function in hemodialysis patients. *Kidney Int* 2007;72:1138–48.
- Sen N, Turunc T, Karatasli M, et al. Tuberculosis in patients with end-stage renal disease undergoing dialysis in an endemic region of Turkey. *Transplant Proc* 2008;40:81–4.
- Quantrill SJ, Woodhead MA, Bell CE, et al. Side-effects of antituberculosis drug treatment in patients with chronic renal failure. *Eur Respir J* 2002;20:440–3.
- Chen HY, Lai SW, Muo CH, et al. Ethambutol-induced optic neuropathy: a nationwide population-based study from Taiwan. *Br J Ophthalmol* 2012;96:1368–71.
- el-Shahawy MA, Gadallah MF, Campese VM. Tuberculosis of the spine (Pott's disease) in patients with end-stage renal disease. *Am J Nephrol* 1994;14:55–9.
- Siskind MS, Thienemann D, Kirilin L. Isoniazid-induced neurotoxicity in chronic dialysis patients: report of three cases and a review of the literature. *Nephron* 1993;64:303–6.
- Fang JT, Chen YC, Chang MY. Ethambutol-induced optic neuritis in patients with end stage renal disease on hemodialysis: two case reports and literature review. *Ren Fail* 2004;26:189–93.
- Lee CS, Brater DC, Gambertoglio JG, et al. Disposition kinetics of ethambutol in man. *J Pharmacokinetic Biopharm* 1980;8:335–46.
- Varughese A, Brater DC, Benet LZ, et al. Ethambutol kinetics in patients with impaired renal function. *Am Rev Respir Dis* 1986;134:34–8.
- Chew R, Jongbloed S, Jegatheesan D, et al. Ethambutol is cleared by a contemporary high-flux hemodialyzer, and drug monitoring ensures safety and therapeutic effect. *Antimicrob Agents Chemother* 2017;61:e01988–16–16.
- Dume T, Wagner C, Wetzels E. [Pharmacokinetics of ethambutol in healthy individuals and in patients with terminal renal failure]. *Dtsch Med Wochenschr* 1971;96:1430–4.
- Trusiewicz D, Farnsworth 100-hue test in diagnosis of ethambutol-induced damage to optic nerve. *Ophthalmologica* 1975;171:425–31.
- Chan RY, Kwok AK. Ocular toxicity of ethambutol. *Hong Kong Med J* 2006;12:56–60.
- Leibold JE. The ocular toxicity of ethambutol and its relation to dose. *Ann NY Acad Sci* 1966;135:904–9.
- Milburn H, Ashman N, Davies P, et al. Guidelines for the prevention and management of *Mycobacterium tuberculosis* infection and disease in adult patients with chronic kidney disease. *Thorax* 2010;65:557–70.
- World Health Organization. Guidelines for treatment of tuberculosis. 4th edn. <http://www.who.int/tb/publications/2010/9789241547833/en/>
- Rustomjee R, Lienhardt C, Kanyok T, et al. A Phase II study of the sterilising activities of ofloxacin, gatifloxacin and moxifloxacin in pulmonary tuberculosis. *Int J Tuberc Lung Dis* 2008;12:128–38.
- Tsai RK, Lee YH. Reversibility of ethambutol optic neuropathy. *J Ocul Pharmacol Ther* 1997;13:473–7.
- Kumar A, Sandramouli S, Verma L, et al. Ocular ethambutol toxicity: is it reversible? *J Clin Neuroophthalmol* 1993;13:15–17.
- Joint Tuberculosis Committee of the British Thoracic Society. Chemotherapy and management of tuberculosis in the United Kingdom: recommendations 1998. Joint Tuberculosis Committee of the British Thoracic Society. *Thorax* 1998;53:536–48.
- Bass JB, Farer LS, Hopewell PC, et al. Treatment of tuberculosis and tuberculosis infection in adults and children. American Thoracic Society and The Centers for Disease Control and Prevention. *Am J Respir Crit Care Med* 1994;149:1359–74.
- Blumberg HM, Burman WJ, Chaisson RE, et al. American thoracic society/centers for disease control and prevention/infectious diseases society of America: treatment of tuberculosis. *Am J Respir Crit Care Med* 2003;167:603–62.
- Hong Kong Department of Health. Preventive measures against drug-induced ocular toxicity during anti-tuberculous treatment (general recommendations). *Annual Report* 2002.
- Larner AJ. Visual failure caused by vitamin B12 deficiency optic neuropathy. *Int J Clin Pract* 2004;58:977–8.
- Ahn C, Oh KH, Kim K, et al. Effect of peritoneal dialysis on plasma and peritoneal fluid concentrations of isoniazid, pyrazinamide, and rifampin. *Perit Dial Int* 2003;23:362–7.
- Kass JS, Shandera WX. Nervous system effects of antituberculosis therapy. *CNS Drugs* 2010;24:655–67.
- Green R, Kinsella LJ. Current concepts in the diagnosis of cobalamin deficiency. *Neurology* 1995;45:1435–1440.
- Nahid P, Dorman SE, Alipanah N, et al. Official American thoracic society/centers for disease control and prevention/infectious diseases society of america clinical practice guidelines: treatment of drug-susceptible tuberculosis. *Clin Infect Dis* 2016;63:e147–e195.
- Kopple JD, Mercurio K, Blumenkrantz MJ, et al. Daily requirement for pyridoxine supplements in chronic renal failure. *Kidney Int* 1981;19:694–704.
- Turnbull L, Bell C, Child F. Tuberculosis (NICE clinical guideline 33). *Arch Dis Child Educ Pract Ed* 2017;102:136–42.
- American Thoracic Society, CDC, Infectious Diseases Society of America. Treatment of tuberculosis. *MMWR Recomm Rep* 2003;52:1–77.
- Mydlik M, Derzsiová K, Svác J, et al. Peritoneal clearance and peritoneal transfer of oxalic acid, vitamin C, and vitamin B6 during continuous ambulatory peritoneal dialysis. *Artif Organs* 1998;22:784–8.
- Shen TC, Huang KY, Chao CH, et al. The risk of chronic kidney disease in tuberculosis: a population-based cohort study. *QJM* 2015;108:397–403.
- Launay-Vacher V, Izzedine H, Deray G. Pharmacokinetic considerations in the treatment of tuberculosis in patients with renal failure. *Clin Pharmacokinet* 2005;44:221–35.
- Baghaei P, Marjani M, Tabarsi P, et al. Impact of chronic renal failure on anti-tuberculosis treatment outcomes. *Int J Tuberc Lung Dis* 2014;18:352–6.

Copyright 2018 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow