



Case report

Breathlessness worsened by haemodialysis

Rizna Abdul Cader*, Kanagerajan Thoriappa, Rozita Mohd, Wei Yen Kong, Ruslinda Mustafar, Lydia Kamaruzaman

Department of Medicine, Universiti Kebangsaan Malaysia Medical Centre, Kuala Lumpur, Malaysia



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ABSTRACT

A 54 year old lady with underlying chronic lung disease on long term oxygen therapy and end stage renal disease of unknown aetiology on regular haemodialysis for two years started developing progressive shortness of breath during her routine haemodialysis. She was unable to tolerate her haemodialysis sessions which had to be terminated prematurely in view of her symptoms despite adjustment of her dry weight and treatment of anaemia. She was not in chronic fluid overload and her symptoms always worsened after initiation of haemodialysis and improved after termination of haemodialysis. She was admitted to hospital for further investigations and initially treated for a lung infection but her symptoms did not improve. A computed tomography pulmonary angiography did not reveal any evidence of pulmonary embolism, and was consistent with chronic fibrotic changes. Her hypoxemia was concluded to be due to her underlying chronic lung disease, worsened by alveolar hypoventilation during haemodialysis. Her symptoms improved slightly with supplemental oxygen during her routine haemodialysis but we had to shorten her haemodialysis duration to 3 hours.

1. Introduction

Hypoxemia during haemodialysis has been well recognized since the early days of haemodialysis with studies reporting it to be as common as 10% in end stage renal disease patients on haemodialysis [1–3]. During haemodialysis, the PaO₂ may fall by 10–20 mmHg and this drop can be detrimental to patients with underlying chronic lung disease or chronic heart failure [4,5]. There have been several mechanisms proposed, however the causes remains unclear. Some studies have attributed hypoxemia during haemodialysis to the type of dialysate used, type of membrane used or a combination of both [6,7]. Other factors contributing to this include anaemia, chronic volume overload and compromised pulmonary function [5–7].

2. Case report

A 54-year-old woman with end stage renal disease of unknown aetiology on regular haemodialysis for the last two years via right internal jugular vein permanent catheter was noted to be short of breath during haemodialysis.

She also has a background history of chronic lung disease which was diagnosed at another hospital after having had three admissions during that year with shortness of breath come reduced effort tolerance and cough with purulent sputum. Investigations for pulmonary tuberculosis

were negative. She had never smoked and had no pets. There was no history of exposure to dust. Prior to the diagnosis of ‘chronic lung disease’, the patient was tolerating haemodialysis well. However, she started developing progressively shortness of breath during haemodialysis. During the initial presentation, patient was still able to tolerate the mild shortness of breath and able to complete 4 hours of haemodialysis. Since three months ago her symptoms became more evident requiring hospital admission. She was noted to be tachypneic and hypoxic by her dialysis nurse with oxygen saturation of 85% under room air during her haemodialysis sessions. Her dry weight was accurate and she had no anemia that could contribute to this. She was therefore admitted and investigated further for her breathlessness. She lives with her son and never had smoked.

On examination, patient was petite weighing 45kg with a blood pressure 140/80 mm Hg, pulse rate of 95 beats/min and oxygen saturation of 96% on nasal prong 3L/min. She was comfortable and neither short of breath nor in distress while being able to complete full sentences. There was no peripheral or central cyanosis. She had grade 2 clubbing on her fingers. Auscultation of her lungs revealed bilateral lower zone inspiratory crepitations. Cardiovascular and abdominal examination was unremarkable. Her full blood count and liver function tests were normal except for serum albumin of 24 g/L.

As this was her first presentation to our hospital, she was investigated with a computed tomography scan which excluded

* Corresponding author.

E-mail address: rizna_c@hotmail.com (R.A. Cader).

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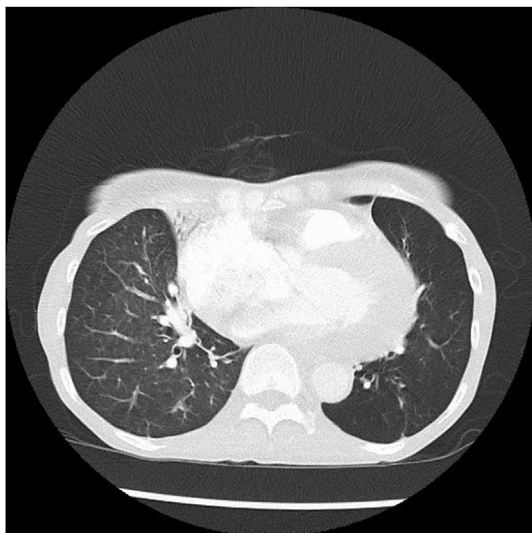


Fig. 1. CT scan demonstrating bronchiectasis changes.

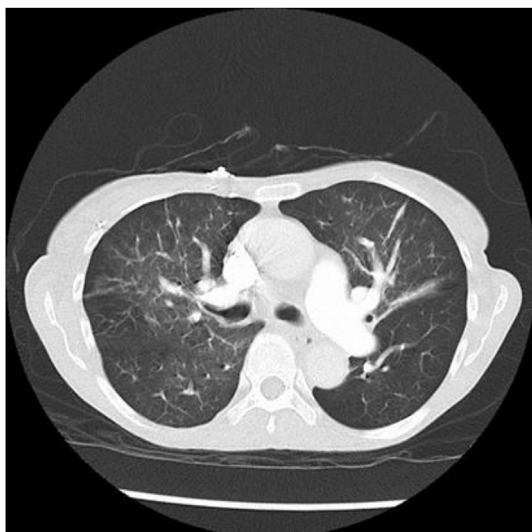


Fig. 2. CT scan demonstrating bronchiectasis changes.

pulmonary embolism; however, both her lung fields were hyper inflated with pleural thickening, bronchiectasis and fibrotic changes. Hence the diagnosis of chronic bronchiectasis was entertained with evidence of an acute infective exacerbation. The computed tomography findings are shown below (Figs. 1 and 2). Lung function test were not performed during this admission as she had an infective exacerbation of bronchiectasis. A resting ECG was normal as was an ECG during haemodialysis. An echocardiogram revealed preserved ejection fraction of 57% with good left ventricular function. There was no regional wall motion abnormalities noted and all heart chambers and valves appeared normal.

She was treated for an infective exacerbation of bronchiectasis with antibiotics and after completion of antibiotics, arterial blood gases (ABG) were performed during and after haemodialysis. ABG during haemodialysis revealed a PH 7.414, P02 58.4 mm Hg, PCO2 31.3 mm Hg, bicarbonate 21.2 mmol/L and oxygen saturations of 85% on 3L/min oxygen. ABG thirty minutes post haemodialysis demonstrated a PH 7.356, P02 75.4 mm Hg, PCO2 34.8 mm Hg, bicarbonate 20.1 mmol/L and oxygen saturations of 93% on 3L/min oxygen.

Upon discharge, she received pneumococcal and influenza vaccination and scheduled for full lung function tests during her follow up appointment. Unfortunately, the patient was readmitted several times

to our institution over the next four months with infective exacerbation of bronchiectasis.

3. Discussion

The prevalence of hypoxemia in haemodialysis patients can be as high as 10% and this mild degree of hypoxemia can last up to 60 minutes during haemodialysis [8–10]. Studies have demonstrated a drop in oxygen saturation and partial pressure (PaO₂) at the beginning of haemodialysis treatment that usually reaches a nadir after 30–60 min and then returns to pre-dialysis levels towards the end of haemodialysis [10,11]. The oxygen levels in the arterial blood can drop by 5–23% during haemodialysis and although this may go unnoticed in most patients, it can be detrimental in those with heart or lung problems [5]. The hypoxemia may be due to a variety of factors including anaemia, chronic fluid volume overload, compromised pulmonary function, complement activation due to type of haemodialysis dialysate and dialyzer membrane [5,12]. Meyring et al. found those patients who had hypoxemia were older, had longer dialysis duration and had higher prevalence of congestive heart failure and chronic obstructive pulmonary disease [8]. In our patient, we excluded congestive cardiac failure as her echocardiogram showed a preserved ejection fraction. Furthermore, our patient had no episodes of chest pain or intradialytic hypotension to suggest cardiac ischaemia that compromised oxygen delivery.

Acetate based dialysate was used in the early days of haemodialysis which can lead to hypoxemia. The cause of this is due to small gaseous carbon dioxide losses across the dialyzer, as acetate metabolism causes a decrease in carbon dioxide production and cause hypoventilation which can cause hypoxemia [8]. Acetate buffer solutions were eventually replaced by bicarbonate solutions to reduce this phenomenon [5]. As most haemodialysis centres now use bicarbonate buffer solutions, our patient was already on a bicarbonate based buffer, and this was not the cause for her hypoxemia.

In 3–5% of haemodialysis treatments, a Type B dialyzer reaction mediated by the complement cascade, with neutrophil activation and sequestration in the pulmonary circulation can occur resulting in hypoxemia [13]. The degree of complement activation depends on the type of dialyzer membrane used and is reportedly less with biocompatible membranes. Our patient was dialyzing using a biocompatible dialyzer membrane which could not explain the cause of her hypoxemia.

We have demonstrated that she was significantly hypoxic on haemodialysis based on the arterial blood gases taken during and after haemodialysis. She also got symptomatic relief with the oxygen but she still couldn't tolerate the full 4 hour haemodialysis treatment and therefore we had to shorten the duration of her haemodialysis. A comparison study carried out to compare pulmonary gas exchange during haemodialysis with patients with and without chronic obstructive pulmonary disease showed hypoxemia in both patient groups during haemodialysis to almost the same degree [11]. This is in keeping with our arterial blood gas findings. However, the lower baseline oxygen in the chronic obstructive pulmonary disease patients made them to be more symptomatic.

4. Conclusion

Several studies have demonstrated that hypoxemia during haemodialysis occurs in both patients with and without underlying chronic lung disease irrespective of the dialysate buffer used. However, patients with chronic lung disease have lower partial pressure oxygen to begin with compared to normal patients thus they were more symptomatic when there was a drop in P0₂. In the effort of reducing her symptoms, our patient received supplemental oxygen during haemodialysis.

Conflicts of interest

All authors declare no conflict of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.rmcr.2018.10.022>.

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