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## A Case Report On Difficulty In Improving Anemia In Patient With CKD-End Stage Renal Disease Undergoing Hemodialysis

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### ABSTRACT

Chronic kidney disease (CKD) is defined as any abnormality in kidney structure or function present for three months or longer, with implication for health. Anemia, which affects most patients with CKD, is caused by a decreased production of erythropoietin (EPO), a glycoprotein that stimulates red blood cell production in the bone marrow and is released in response to hypoxia. A 68 years old female came with complaints of generalized tiredness, left side chest pain during inspiration, pedal oedema and breathing difficulty. Her past medication history include CKD, diabetes mellitus, hypertension, recurrent UTI, gastritis. A diagnosis of CKD-end stage renal disease associated with anemia was made following the review of her clinical examination and laboratory findings. She was subsequently managed for anemia with recombinant human erythropoietin and intravenous iron, monitored, and expected result was not found. In this study we conclude that the patient is resistant to erythropoietin so packed RBC is given for the management.

**Keywords:** Chronic kidney disease, Anemia, human erythropoietin, packed RBC

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## INTRODUCTION

Chronic kidney disease is defined as the condition in which decreased kidney function shown by glomerular filtration rate (GFR) of less than 60 mL/min per 1.73 m<sup>2</sup>, or markers of kidney damage, or both, of at least 3 months duration, regardless of the underlying cause. Anemia commonly occurs in people with chronic kidney disease (CKD) the permanent, partial loss of kidney function. Anemia might begin to develop in the early stages of CKD, when someone has 20 to 50 percent of normal kidney function. Anemia tends to worsen as CKD progresses. Most people, who have total loss of kidney function, or kidney failure, have anemia. When kidneys are diseased or damaged, they do not make enough erythropoietin. As a result, the bone marrow makes fewer red blood cells, causing anemia. When blood has fewer red blood cells, it deprives the body of the oxygen it needs, so patient becomes anemic. Recombinant human erythropoietin is given for the management of anemia in CKD. Here the patient fails to respond to EPO therapy. Infection and inflammation have been shown to influence responsiveness to rh-Epo by disrupting iron metabolism and eliciting the release of cytokines that inhibit erythropoiesis. Hyperparathyroidism can lead to reduction in the number of responsive erythroid progenitor cells<sup>1</sup>. The hyperparathyroid state induces anaemia in patients with normal kidney function<sup>2</sup>. In secondary hyperparathyroidism of CKD patients, the high levels of circulating PTH have multiple biological effects, including an unfavourable influence on the anaemia of CKD patients. Possible pathogenic mechanisms of this relationship may be PTH-direct effects on inhibition of early erythroid progenitors, endogenous EPO synthesis and RBC survival and loss.

## CASE STUDY REPORT

A 68 years old female came with complaints of generalized tiredness, left side chest pain during inspiration, pedal oedema and breathing difficulty. Her past medication history was CKD, diabetes mellitus (26 years), hypertension (8 years) recurrent UTI, gastritis. On examination she was found to have generalized tiredness, conscious and oriented. Further laboratory evaluation revealed that she had elevated creatinine, urea, phosphorous, parathyroid hormone, CPK, ESR, TC and decrease in Hb, Tsat, TIBC and presence of proteinuria. A diagnosis of CKD-end stage renal disease associated with anemia was made following the review of her clinical examination and laboratory findings. Her treatment schedule include T.Cilacar 10mg, T. Arkamin 0.1mg, T Minipress XL 2.5mg, T. Isolazine, T.Pramipex 0.25 mg., T.Thyronorm 125mcg, T. Storvas 20mg, T.Ecospirin 150mg, T.Pantocid DSR, T. Revlamer 400mg, T- Bact ointment, Seroflo 250 mcg, K Bind 15ml with Sorbiline, Inj. Nucarnit 1 amp, Inj RPO 10000 IU, Inj Overcom 100mg. She was

subsequently managed for anemia with recombinant human erythropoietin and intravenous iron, monitored and the expected result was not found.

### **Laboratory Findings**

<b>Parameters</b>	<b>Observed value</b>	<b>Normal value</b>
Hb	8.5	12-15gm/dl
K	5.86	3.5-5mEq/l
Urea	134	15-45 mg/dl
Serum Creatinine	7	0.7-1.2 mg/dl
PTH	766	10-65ng/l
TIBC	214	45-85 micromol/l
Phosphorous	7.4	2.5-4.5mg/dl
Sodium	140	135-145mEq/l
Iron	54	65-180mcg/dl
CPK	177	10-120mcg/l
Ferritin	979	12-300ng/ml
TSAT	25.2	60-170 mcg/dl

## **MATERIALS AND METHOD**

Searches of English language publications were conducted in both MEDLINE and EMBASE. The search used previously developed strategies combining inclusive terms for chronic kidney disease, anemia, and observational studies, clinical review. A hand search of the bibliographies of the retrieved articles were analyzed.

### **Addressing the Issue**

This case raised an important question that is why it is difficult in improving anemia in this patient even after the treatment was given. Is there any abnormality in iron stores or is it due to gastritis? Is there any evidence for justifying this question?

## **RESULTS AND DISCUSSION**

A 68 year old female was diagnosed to have CKD-ESRD with anemia undergoing hemodialysis. Laboratory findings reveal that her Hb is low but iron stores are maintained and she is having hyperparathyroidism. She was subsequently managed for anemia with recombinant human erythropoietin and intravenous iron. After the treatment was given the patient is monitored and Hb was tested. The result shows no improvement in Hb level and patient had generalized tiredness. Here the patient may be resistant to erythropoietin. Resistance to recombinant human erythropoietin is a common condition in dialyzed patients with chronic kidney disease and is associated with more hospitalizations, increased mortality and frequent blood transfusions. However, a high proportion of patients do not respond to treatment, even to the use of intravenous iron, which indicates the presence of other important causes of resistance. The most common

causes of resistance include inflammation, infection, malnutrition, inadequate dialysis, and hyperparathyroidism, although other factors may be associated. In the presence of adequate iron stores, other causes should be investigated and treated appropriately<sup>3</sup>. For this patient packed RBC can be given for the management.

## CONCLUSION

The major cause of anemia is inadequate renal production of erythropoietin, and treatment with erythropoiesis stimulating agents is highly effective in improving the level of haemoglobin. In most of the studies the authors concluded that in long-term hemodialysis patients, hyperparathyroidism, inflammation, infection, malnutrition, and are associated with significant erythropoietin stimulating agent hypo responsiveness. In this study the patient is having adequate iron stores but her haemoglobin is not improving even after the erythropoietin is given. The possible reason is erythropoietin resistance which is due to hyperparathyroidism and gastritis. Here the patient is at CKD-ESRD and to maintain her Hb, can be managed with packed RBC transfusion for the correction of anemia.

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