Factors for anemia after kidney transplantation

A data analysis of 826 patients from one institute during 10 years★

Cao Zhi-qiang, Liu Long, Yang Hong-wei, Fan Lian-hui, Li Xin

Abstract

BACKGROUND: Anemia after kidney transplantation has a clinical incidence rate of 30%–40%, is the important risk factor for cardiovascular diseases and kidney failure after kidney transplantation and is also the independent prediction index of patient's death.

OBJECTIVE: To analyze the factors related to anemia after kidney transplantation.

METHODS: A total of 826 patients, including 541 males and 285 females, aged 18–71 years, who received kidney transplantation in the General Hospital of Shenyang Military Area Command of Chinese PLA from January 2000 to December 2009 were included in this study. Altogether 805 patients received cadaver donor kidney transplantation and 21 patients received living-donor kidney transplantation. All included patients were assigned to two groups: anemia and non-anemia. The possible factors for anemia after kidney transplantation were recorded. t test and chi-square test were used for one-way analysis of variance.

RESULTS AND CONCLUSION: In the anemia group (n = 225, 27.2%, aged 26–65 years), the incidence rate of anemia in female and male patients was 23% and 37%, respectively (P < 0.05), 46 patients had hypertension and used angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist and 16 patients had chronic erosive gastritis or upper gastrointestinal tract ulcer, with the human survival rate of 85.3% and kidney failure rate of 25.3%. In the non-anemia group (n = 601, 72.8%, 405 males, 196 females, aged 18–71 years), 35 patients had hypertension and used angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist and 14 patients had chronic erosive gastritis or upper gastrointestinal tract ulcer, with the human survival rate of 92.1% and kidney failure rate of 12.6%. There was significant difference in above-mentioned indices between anemia and non-anemia groups (P < 0.05). These results suggest that gender, age, kidney function, digestive tract disease history, and drug application are closely related to anemia after kidney transplantation.

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INTRODUCTION

In end-stage renal disease-caused anemia patients who received kidney transplantation, endogenous erythropoietin secretion is increased and hemoglobin level will recover to normal within 8–12 weeks along with the functional recovery of transplanted kidney. For this reason, clinical physicians often ignore the occurrence of anemia after kidney transplantation [1]. However, the clinical incidence of anemia after kidney transplantation is 30%–40%[2]. Anemia is the most important risk factor for cardiovascular disease and kidney failure after kidney transplantation[3] and the independent prediction index of patient's death[4], and therefore is paid increasing attention. This study retrospectively analyzed the factors related to anemia after kidney transplantation in 826 patients who received kidney transplantation at the Department of Urology, the General Hospital of Shenyang Military Area Command of Chinese PLA between January 2000 and December 2009 and summarized therapeutic methods based on references.

SUBJECTS AND METHODS

Design

A retrospective investigation on clinical case data.

Time and setting

This study was performed at the Follow-up Center, Department of Urology, the General Hospital of Shenyang Military Area Command of Chinese PLA between January 2010 and January 2011.

Subjects

Inclusion criteria: patients who received renal allograft transplantation at the Kidney Transplantation Center, the General Hospital of Shenyang Military Area Command of Chinese PLA between January 2000 and December 2009 and those patients who received kidney transplantation in other hospitals but were followed up in our hospital were excluded because of incomplete data. Written informed consent regarding data registration and analysis was obtained from each patient.

A total of 826 patients, including 541 males and 285 females, aged 18–71 years (average 42.8 years) were included in this analysis. Altogether 805 patients received cadaver donor kidney transplantation and 21 patients received living-donor kidney transplantation. A total of 730 patients had chronic glomerulonephritis, 24 patients had polycystic kidney, 64 patients had diabetic nephropathy, and eight patients had drug-induced renal damage. Anti-rejection regimen: Altogether 635 patients received an anti-rejection regimen consisting of ciclosporin A/tacrolimus, mycophenolate mofetil and prednisone, 127 patients received an anti-rejection regimen consisting of ciclosporin A/tacrolimus and mycophenolate mofetil, 45 patients received an anti-rejection regimen consisting of ciclosporin A/tacrolimus and azathioprine, and 27 patients had received sirolimus for over 6 months. Diagnostic criteria of anemia: For adult males: ⩾18 years old, hemoglobin < 120 g/L, total number of red cells < 4.5×10^{12}/L or erythrocrit < 0.42; for adult

females: ⩾18 years old, hemoglobin < 110 g/L, total number of red cells < 4.0×10^{12}/L or erythrocrit < 0.37.

Main outcome measures

Patients's age, gender, combined diseases, immunosuppressive drugs or drugs for other diseases, blood routine, and blood pressure.

Statistical analysis

Statistical analysis was performed by the first author using SPSS 13.0 software. One-way analysis of variance (*t* test) was used for evaluation of measurement data and chi-square test was used for evaluation of numeration data. A level of $P < 0.01$ was considered statistically highly significant and $P < 0.05$ was considered statistically significant.

RESULTS

Quantitative analysis of participants

All 826 patients were included in the final analysis.

Data of anemia after kidney transplantation and related risk factors

The 826 kidney transplantation patients were assigned to two groups: anemia and non-anemia. In the anemia group (n = 225, 27.2%), there were 136 males and 89 females, with an average age of 41 (26-65) years; the incidence of anemia in females and males was 23% and 37%, respectively, this suggests that anemia after kidney transplantation easily occurs in males ($P < 0.05$); 112 patients (50% of anemia patients) had poor function of transplanted kidney; 46 patients (20% of anemia patients) had hypertension and used angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist and 16 patients (7.1% of anemia patients) had chronic erosive gastritis or upper gastrointestinal tract ulcer, with the human survival rate of 85.3% and kidney failure rate of 25.3%. In the non-anemia group (n = 601, 72.8%), there were 405 males and 196 females, with an average age of 43 (18–71) years, and there was no significant difference in these data between anemia and non-anemia groups; 74 patients (12.3% of non-anemia patients) had poor function of transplanted kidney, and comparison results showed that poor function of kidney transplantation is an independent risk for anemia after kidney transplantation ($P < 0.01$); 35 patients (5.8% of non-anemia patients) had hypertension and used angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist and 14 patients (2.3% of non-anemia patients) had chronic erosive gastritis or upper gastrointestinal tract ulcer, and this suggests that angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist is closely related to complicated digestive tract disease and anemia after kidney ($P < 0.05$). The human survival rate was 92.1% and kidney failure rate was 12.6%, suggesting that anemia after kidney transplantation significantly influences human/kidney survival rate ($P < 0.01$). In the past, not too many patients received kidney transplantation because of poor knowledge of anemia after kidney transplantation, so a simple comparison was not made between the anemia and non-anemia groups. Detailed data are shown in Table 1.

Table 1 Data of anemia after kidney transplantation in the General Hospital of Shenyang Military Area Command of Chinese PLA in 2000-2009

Item	Anemia group	Non-anemia group	*P*
Gender (male /female)	136/89	405/196	< 0.05*
Age (yr)	41(26–65)	43(18–71)	> 0.05
Blood type (*n*)			
A	34	153	
B	25	222	< 0.05*
O	22	174	
AB	44	52	
Complicated digestive tract diseases (*n*)	16	14	< 0.05*
Drugs (*n*)			
Azathioprine	23	46	< 0.05*
Mycophenolate mofetil	134	457	< 0.05*
Ciclosporin A	156	503	> 0.05*
FK506	67	74	> 0.05*
ACEI	24	37	< 0.05*
ARB	35	23	> 0.05*
Prednisone	213	564	> 0.05*
Human/kidney death (*n*)	33/57	47/76	< 0.05*

Note: * indicates the *P* value corresponding to percentage value in the same group.

DISCUSSION

The main reasons for anemia after kidney transplantation include the followings.

Poor function of transplanted kidney: renal anemia is the common reason for anemia after kidney transplantation. The clinical incidence of anemia in patients with > 20 mg/L serum creatinine is 3 times higher than that in patients with ⩽ 20 mg/L serum creatinine[5]. The characteristics of anemia in chronic kidney diseases are hemopoietin deficiency and resistance to hemopoietin. The causes for hemopoietin deficiency after kidney transplantation are complex. Renal fibroblast destruction or phenotype transformation can reduce the cells generating hemopoietin; inflammatory transmitter in the local diseased region can inhibit the formation of hemopoietin; cell injury surrounding the renal tubule that secretes hemopoietin can cause deficient secretion of hemopoietin, and kidney compensation causes hemopoietin deficiency. During kidney transplantation, ischemia/reperfusion injury, acute tubular necrosis, delayed recovery of kidney function, and acute rejection can cause hemopoietin deficiency, which cannot improve anemia. In a few patients with normal kidney function, non-recovered anemia may result from hemopoietin deficiency or relative resistance to hemopoietin[6]. Hemopoietin resistance after kidney transplantation is mainly attributed to two following factors: one is dialysis-related sequelae, iron deficiency, which is the most important reason, aluminium poisoning, secondary hyperparathyroidism and chronic inflammation; the other one is adverse reactions of drugs used including trimethoprim + sulfamethoxazole, acyclovir, azathioprine, and sirolimus. Chronic infection can lead to hemopoietin resistance. In addition, vitamin deficiency (vitamin C, B12, and folic acid), chronic blood loss, hemoglobinopathy, myelofibrosis, malignant

tumor, and hemolysis can also lead to hemopoietin resistance[7]. A comparison of anemia incidence between normal controls and kidney transplantation patients showed that kidney function was normal in the two groups. The incidence of anemia was higher, and the hemoglobin level was also lower, in the kidney transplantation patients than in the normal controls, indicating that poor function of transplanted kidney is the only one cause of anemia[8].

Some drugs may lead to myelosuppression: Most of immunosuppressive agents used after kidney transplantation may produce bone marrow toxicity and bone marrow suppression. Different drug combinations and drug interaction play important role in anemia after kidney transplantation. Hormone promotes the formation of red cells. Hormone withdrawal or decrease after kidney transplantation would increase the incidence of anemia[9]. Bone marrow suppression is the most common adverse reaction of immunosuppressive agent azathioprine and it is complicated by reduced white cells and blood platelets. Long-term use of azathioprine can lead to megalocytosis. Whether mycophenolate mofetil can induce anemia remains disputed[10]. Inosine monophosphate dehydrogenase can inhibit the initial synthesis of T, B cells. Because salvage pathway is involved in the formation of cell cells, so theoretically mycophenolate mofetil can not influence the formation of red cells. But in the clinic, mycophenolate mofetil is found to inhibit, to some an extent, the existence of bone marrow cells. Evidence exists that mycophenolate mofetil's effects on anemia is related to its dose, and proper drug dose can avoid the occurrence of anemia complications[11]. In the early stage of kidney transplantation, mycophenolate mofetil alone or its combination with Tacrolimus can increase anemia incidence and blood transfusion rate[12]. Sirolimus, as the macrolides, can produce immunosuppressive effects via the activity of transplanted T and B cells. Theoretically, Sirolimus has no effects on inhibiting red cells formation and reduced white cells. Clinical studies have shown that Sirolimus effects on red cells formation are closely related to drug dose and concentration. Evidence exists that Sirolimus induces anemia by influencing iron metabolism[13]. Sirolimus can mediate the autoregulation of inflammation and secondary infection-induced anemia by influencing interleukin 10[14]. Sirolimus effects on generation of red cells are related to drug dose and concentration[15]. Sirolumus combined with mycophenolate mofetil easily results in enhanced bone marrow suppression, so this combination should be avoided as possible. Calcineurin inhibitors including cyclosporine and tacrolimus exhibit slight effects on bone marrow suppression. In the clinic, cyclosporine and Tacrolimus are even used for treatment of aplastic anemia and acquire marked curative effects. Angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist can decrease cardiovascular disease death rate, alleviate renal tissue fibrosis, reduce proteinuria and protect the function of transplanted kidney. Therefore, they are widely used after kidney transplantation.

In normal population, angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist can to some extent reduce hematocrit and decrease erythropoietin level *in vivo*, and at present, they are the main drugs for treatment of erythrocytosis after kidney transplantation. Theoretically, angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist has the potential to aggravate the anemia, but in the clinic, this trend is not obvious. In this study, there was no obvious increasing incidence of anemia after use of these two drugs ($P < 0.05$). Compared with the advantages of these two drugs, the adverse reaction that these two drugs can aggravate anemia can be ignored, therefore, these two drugs should be considered after kidney transplantation.

Nutritional problems lead to anemia: malnutrition, iron deficiency and dysmetabolism, folic acid deficiency, and vitamin B12 deficiency can lead to anemia.

Gastrointestinal tract blood loss leads to anemia: anemia during the early stage of kidney transplantation is caused by intraoperative blood loss, hemodilution, and surgery-related inflammatory state. Hemodialysis and frequent blood taking for tests can cause blood loss. Chronic blood loss during the late stage of kidney transplantation mostly results from gastrointestinal tract chronic blood loss, and various factors-caused gastrointestinal dysfunction can also lead to malnutritional anemia[16].

Infection leads to anemia: bacterial infection (including Bacillus tuberculosis infection), cytomegalovirus infection, minute virus B19 (PvB19) infection, EB virus and Epstein-Barr virus, and human immunodeficiency virus infection can lead to anemia[16].

In vivo iron metabolism abnormality: iron is the important integral of red cell formation, and iron deficiency anemia has a high incidence in common population. Reduced iron content *in vivo* in patients with functional failure of transplanted kidney is a primary factor of anemia[17]. The recovery speed of anemia after kidney transplantation is parallel to decrease in serum ferritin *in vivo*. Serum ferritin is influenced by various factors, such as systemic inflammatory state and liver disease[18]. Iron deficiency anemia is often accompanied by increased serum transferrin *in vivo*, while serum transferrin content is an indicator of acute inflammatory reaction in patients who underwent kidney transplantation. Transferrin can be used to evaluate the iron content used *in vivo*. The degree of saturation of transferrin can reflect the content of iron that combines with transferrin in serum. However, transferrin is limited in clinical application due to its great variance in degree of saturation. Because there are many factors influencing the degree of saturation of ferritin and transferrin, so for patients who received kidney transplantation, it is necessary to select proper indictors to guide chalybeate supplementation and judge bone marrow change[18]. For this reason, diagnosing iron deficiency only depending on the criteria of serum ferritin in patients who received kidney transplantation has a high rate of missed diagnosis.

Age and gender factors: the present data show that anemia more easily occurs in male elderly patients who received kidney transplantation than in female elderly patients ($P < 0.05$), which is similar to a previous report[19].

Treatment: Anemia is always ignored by physicians[20]. A study regarding anemia after kidney transplantation showed that only 5.2% of anemia patients received erythropoietin treatment, and only 17.8% of 343 patients with severe anemia received erythropoietin treatment[21].

To treat anemia after kidney transplantation, the causes of anemia should be identified under the guidance of physicians. The precise process should include removal of inflammatory state *in vivo*, treatment of infection, acute and chronic rejection, selection of proper immunosuppressive scheme, and

improvement in kidney transplantation. Erythropoietin is preferred to treat anemia after kidney transplantation. Abbud-Filho *et al*[22] suggested that erythropoietin treatment is used when hematin is < 11 g/L. For poor curative effects of erythropoietin treatment, bone marrow suppression and erythropoietin resistance should be excluded and possible pathological factors should be identified and managed as soon as possible. For example, immunoglobulin should be selected for PVB19 infection; if anemia is caused by antihypertensive drugs, such as angiotensin-converting enzyme inhibitor or angiotensin Ⅱ receptor antagonist, and by antimetabolites, such as azathioprine or mycophenolate mofetil, drugs should be reduced in amount or changed. For treatment of digestive tract hemorrhage, acid-inhibitory drugs and stomach mucous membrane protectant should be used, digestive tract irritant drugs should be limited in use, and simultaneously, digestive tract tumor and concretion should be excluded. For patients with abnormal iron metabolism, sufficient amount of chalybeate should be supplemented. Oral administration of chalybeate has drawbacks including poor absorbance, great stimulation to gastrointestinal tract, slow effect, and influences on absorbance of other drugs. Intravenous administration of iron sucrose in combination with supplementation of folic acid and vitamin C as well as hypoproteinemia treatment would produce better curative effects.

It remains disputed regarding selection of treatment time for anemia after kidney transplantation and determination of hemoglobin level. Evidence exists that erythrocrit is negatively correlated with mortality in anemia after kidney transplantation, and the optimal hematocrit is 38.2% at which the mortality is lowest[23]. Other studies show that complete correction of anemia in patients with grades 3–4 chronic nephropathy did not reduce cardiovascular complications and even led to an increasing incidence in some patients[24-25], but the fact that increasing hematocrit increases mortality was not found[26]. A study shows that at 12 months after kidney transplantation, human/kidney survival rate would be greatly decreased in patients who received kidney transplantation[27]. A 4-year follow-up study showed that patient mortality rate and organ loss rate were greatly increased in patients who received anemia after kidney transplantation[28], which were basically consistent with the present data.

Taken together, anemia after kidney transplantation is very common. Correction of anemia can decrease the incidence of cardiovascular events, postpone the progression of chronic renal insufficiency, retain good function of transplanted kidney and maintain long-term survival rate of human/transplanted kidney. Therefore, anemia after kidney transplantation should be paid sufficient attention and given timely diagnosis and treatment, which can rapidly increase hemoglobin level and erythrocrit to normal and further improve the long-term prognosis of transplanted kidney.

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肾移植后贫血因素分析：同一机构 10 年 826 例移植者资料回顾★

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摘要

背景：临床上肾移植后贫血发生率为 30%~40%，是肾移植后心血管疾病和移植肾失功的重要危险因素，并且是患者死亡的独立预测指标。

目的：分析肾移植后贫血发生的相关因素。

方法：分析 2000-01/2009-12 解放军沈阳军区总医院肾移植 826 例患者资料，其中男 541 例，女 285 例，年龄 17~71 岁；尸体供肾 805 例，亲体活体供肾 21 例。根据移植后是否发生贫血将患者分成贫血组与非贫血组，记录可能引起移植后贫血的各项参数，分别用 *t* 检验和卡方检验进行单因素分析。

结果与结论：826 例肾移植患者中发生贫血 225 例，发生率为 27.2%。女性和男性患者贫血发生率分别为 23%和 37%($P < 0.05$)；年龄 26~65 岁，其中伴有高血压应用血管紧张素转化酶抑制剂或血管紧张素Ⅱ受体拮抗剂类降压药物者 46 例，伴有慢性糜烂性胃炎或上消化道溃疡者 16 例，人存活率为 85.3%，肾失功率为 25.3%；非贫血组 601 例，占总例数的 72.8%，其中男 405 例，女 196 例，年龄 17~71 岁，伴有高血压应用血管紧张素转化酶抑制剂或血管紧张素Ⅱ受体拮抗剂降压药物者 35 例，伴有慢性糜烂性胃炎或上消化道溃疡者 14 例，人存活率为 92.1%，肾失功率为 12.6%，与贫血组对比差异有显著性意义($P < 0.05$)。分析表明，性别、年龄、肾功能、消化道疾病史、药物因素等与肾移植后贫血的发生密切相关。

关键词：肾移植；贫血；病因；治疗；器官移植

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外国专家修饰的医学英语句型：本刊英文部

中文	修改前	修改后
应用 RNA 干扰(RNA interference，RNAi)技术干扰发育期大鼠大脑皮质神经元 $GABA_AR$ α1 基因的合成，应用膜片钳技术检测神经元电活动中外向钾电流的变化。	**In this study, we used** RNA interference (RNAi) to **interfere** synthesis of the cortical neuronal γ-aminobutyric acid A receptor (GABAAR) α1 in rats **at** development, **phase and detected** outward K^+ currents during neuronal electrical activity using whole-cell patch-clamp techniques.	**We used** RNA interference (RNAi) to **disrupt** synthesis of the cortical neuronal γ-aminobutyric acid A receptor ($GABA_AR$) α1 in rats **during** development, and **measured** outward K^+ currents during neuronal electrical activity using whole-cell patch-clamp techniques.
$GABA_AR$ 由 2 个 α、2 个 β 与 1 个 γ_2 组成五聚体，在成熟脑中 α1 亚单位广泛表达，随着脑的发育表达逐渐增加，与脑成熟、神经元迁移、分化以及突触形成有着密切的联系。	The GABAAR is a pentamer **comprising** two α, two β and one γ_2. α1 subunit is extensively expressed in **mature** brain, **and highly** correlated with brain maturation, neuronal migration and differentiation, and synapsetic formation.	The $GABA_AR$ is a pentamer **consisting of five subunits**: two α, two β and one γ_2. **The** α1 subunit is extensively expressed in **the adult** brain, **and its expression is highly** correlated with brain maturation, neuronal migration and differentiation, and synapse formation.