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# Reversibility of Grade 4 Hypertensive Retinopathy One Month After Living Donor Kidney Transplant in A 26-Year-Old Woman with IGA Nephropathy Induced End Stage Renal Disease: A Rare Case Report

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**ABSTRACT**

The patient was a 26-year-old woman; she developed end stage renal disease (ESRD) due to IgA nephropathy. She was on maintenance hemodialysis for one year. Her blood pressure was 160/110 mmHg; barely controlled with 4 different anti-hypertensive drugs. It occasionally rose to 220/130 mmHg at the end of hemodialysis. Fundoscopy revealed Grade 4 Hypertensive Retinopathy and carotid intima media thickness was increased (1.0 mm). Her blood pressure decreased to 140/90 mmHg 3 days after living donor kidney transplant, requiring monotherapy. Fundoscopy one month after transplant showed Grade 2 Hypertensive Retinopathy.

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## INTRODUCTION

Hypertension is one of the leading cause of cardiovascular morbidity and mortality. Severe hypertension has higher risks; coronary artery disease, heart failure, stroke and chronic kidney disease (CKD) (Park et al., 2023) (Callus et al., 2017) (Seeman et al., 2009) (Gago Fraile et al., 2009). They emphasized timely diagnosis and effective treatment to reduce complications.

Hypertension and CKD have a cause-and-effect relationship. A falling kidney function with advanced CKD can lead to increased blood pressure, whereas sustained elevations in blood pressure can deteriorate kidney function (Verdalles et al., 2016) (Gu et al., 2013). Hypertension in patients with chronic kidney disease (CKD) was very common and often poorly controlled (Buhnerkempe et al., 2021)(Fay & Cohen, 2021) (Maasila et al., 2018) (Kim et al., n.d.). They recommended to follow a stepwise, evidence-based approach in controlling blood pressure including renal denervation (Acelajado et al., 2019) (Seeman et al, 2020) (Filippone et al., 2024) (Balahura et al., 2022) and carotid baroreceptor stimulation (Doumas et al., 2011).

Several studies mentioned the effect of hypertension on retina; and Grade 3 Hypertensive Retinopathy was commonest among patients with CKD stage 5/ESRD and renal transplant recipients (Jain, Jain, et al., 2024) (Arriozola-Rodríguez et al., 2015)(Ooi et al., 2015). Moreover, they reported the association between retinal changes among hypertensive patients and impaired renal function (Omotoso et al., 2016) (Ślizień et al., 2019).

The blood pressure in post-transplant period was found to be higher than their pre-transplant level in majority of kidney transplant recipients (Seeman, 2009) (Kuźmiuk-Glembin et al., 2018) (Opelz & Döhler, 2005) (Jaques et al., 2021) (Agarwal et al., 2023) (Pagonas et al., 2019). In addition, they found that higher blood pressure had great impact on both graft and patient survival in them (Callus et al., 2017). This

case report described the difficulties in managing severe hypertension in patient on maintenance haemodialysis; and, the changes after living kidney transplantation and retinopathy were illustrated.

## CASE PRESENTATION

The patient was 26 years old; a single from middle part of Myanmar. She had history of acute nephritis in 2021; and she was apparently healthy. She was found to have raised serum creatinine in June 2023 when she did medical check-up. And, renal biopsy was done. It showed mesangial proliferation in glomeruli; interstitial fibrosis; and, tubular atrophy in 40% of tubules. There was no evidence of arteriolosclerosis. Immunofluorescence studies revealed dominant IgA depositions in mesangial region consistent with IgA nephropathy. Blood urea was 59.7 mg/dl; serum creatinine was 6.08 mg/dl; electrolytes were within normal limits); liver function tests were normal; total cholesterol was 227.8 mg/dl; LDL was 174.6 mg/dl; triglycerides was 102.6 mg/dl; HDL was 48.5 mg/dl; uric acid was 535.0 mg/dl; phosphorus was 3.75 mg/dl; calcium was 9.10 mg/dl; corrected calcium was 4.3 g/dl; urine albumin was 92.67 mg/dl; and urine albumin creatinine ratio (UACR) was high (2,093 mg/g).

Therefore, she was diagnosed as ESRD (eGFR = 7.8 ml/min/1.73 $m^2$) with underlying chronic glomerulonephritis IgA nephropathy. She was planned for living-donor kidney transplantation; and, maintenance hemodialysis initiated. There was no history of blood transfusion. The residual urine output was approximately 500 ml/day. Her blood pressure was initially controlled with 2 drugs (Nifedipine 20 mg BD & Frusemide 40 mg OD cm). Two months later, blood pressure was increasing and not well controlled with 4 anti-hypertensive medications (Nifedipine 40 mg TDS, carvedilol 1.5 mg BD, Aldactone 25 mg OD, Frusemide 40 mg BD). It rose up to 180/130 mmHg and 220/110 mmHg at the end of each dialysis. She suffered from gradual blurring of vision and started to wear eye-glasses for her vision.

In April, 2024, she was planned for living-donor renal transplantation. Donor was her sister, 36 years old (1$^{st}$ degree relative). Prior to transplant, her biochemistry revealed blood urea 47.4mg/dl; serum creatinine 6.94 mg/dl; serum phosphate 3.75 mg/dl; serum calcium 9.1 mg/dl; hemoglobin 11.0 gm%; Total WBC 8.3X$10^9$ /L; platelet count 289X$10^9$ /L; urine ACR 2093 mg/g. In screening for atherosclerosis, carotid doppler was performed. The carotid intima media thickness was 0.7 mm on right common carotid artery and right bulb; and 0.8 mm on left common carotid artery and 0.9 mm on left bulb. There was neither soft plaque nor hard plaque. On fundoscopy, papilledema, flame shape hemorrhages and arterio-venous nipping suggestive of Grade 4 Hypertensive Retinopathy were found. There was no feature suggestive of raised intracranial pressure clinically, the treating team withhold doing NECT scan of head.

Living-donor renal transplantation was successfully done on September 2024. In post-operative period, the average blood pressure was 150/90 mmHg. One week later, her blood pressure became stable at 125/75 mmHg; she did not require anti-hypertensive therapy. Fundoscopy done one month after renal transplantation revealed resolution of Grade 4 Hypertensive Retinopathy. Her serum creatinine decreased (1.1 mg/dl) and urine output was 4,000 cc daily. She was on 3 immunosuppressants; corticosteroids, mycophenolate mofetil and tacrolimus.

## DISCUSSION

Severe hypertension causes higher rates of major adverse cardiovascular events such as coronary artery disease, heart failure, stroke and chronic kidney disease (CKD). Therefore, timely diagnosis and treatment of severe hypertension improved short and long-term prognosis (Park et al., 2023) (Callus et al., 2017) (Seeman et al., 2009) (Gago Fraile et al., 2009). On the other hand, the prevalence of severe hypertension increases with age, the degree of CKD and albuminuria (Verdalles et al., 2016) (Gu et al., 2013). Hypertension and CKD have a cause-and-effect relationship. A falling kidney function with advanced CKD can lead to increased blood pressure, whereas sustained elevations in blood pressure can deteriorate kidney function. This patient had late stage of CKD (end stage renal disease ESRD); and, the aetiology was IgA nephropathy. Therefore, she had increased risks for adverse cardiovascular outcome (Seeman et al, 2020). The aim of controlling blood pressure in this case was to prevent adverse cardiovascular events.

Hypertension in patients with chronic kidney disease (CKD) was very common and often difficult to control (Buhnerkempe et al., 2021)(Fay & Cohen, 2021) (Maasila et al., 2018) (Kim et al., n.d.). They recommended to follow a stepwise, evidence-based approach in controlling blood pressure. The steps in management of severe hypertension in patients with CKD/ESRD were as follows: (1) dietary sodium restriction; (2) an angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker as the antihypertensive agent of first choice; (3) long-acting dihydropyridine calcium channel blockers as second-line therapeutic options; (4) diuretics as third-line therapeutic options; (5) the addition of spironolactone to the baseline antihypertensive regimen; (6) the non-steroidal mineralocorticoid receptor antagonist ocedurenone; (7) dual endothelin receptor antagonist aprocitentan; (8) the aldosterone synthase inhibitor baxdrostat (Georgianos & Agarwal, 2023); (9) sympathetic inhibition (alpha blockade, centrally acting sympathoinhibitors, or both); (10) renal denervation through radiofrequency, ultrasound, or alcohol infusion, directly targeting on sympathetic overactivity (Acelajado et al., 2019) (Fan et al., 2011) (Dobrowolski et al., 2015) (Seeman et al, 2020) (Filippone et al., 2024) (Acelajado et al., 2019)

(Balahura et al., 2022); and, (11) carotid baroreceptor stimulation (Doumas et al., 2011).

And, her blood pressure was very difficult to control; it did not drop below 160/120 mmHg with 4 different drugs (Nifedipine retard 40 mg TDS, Carvedilol 12.5 mg BD, Aldactone 25 mg OD, Frusemide 40 mg BD). It became sky high at the end of some haemodialysis requiring intravenous nitrate infusion and hydralazine. It even rose to 200/140 mmHg at the end of haemodialysis for several episodes. We did exclude the "white-coat effect" by monitoring her home blood pressure; it was found to be the same as hospital setting. As one of the contributing factors for hypertension in cases with ESRD was fluid overload, we increased haemodialysis session to three times a week. We checked compliance to drugs and diet because adherence to antihypertensive medication was a common factor in severe hypertension. The dialysate sodium content was related with intra-dialysis hypertension; thus, we used low sodium dialysate. However, it did not work. Finally, the dosage of long-acting calcium blocker was maximized to 40 mg three times a day; it was titrated with heart rate and blood pressure. Her body weight was 110 lb and total dose of nifedipine was 120 mg per day. It was maximally tolerated dosage by this patient. Nonetheless, she was tolerating well to medications. According to Acelajado et al, severe hypertension was associated with renin angiotensin aldosterone system hyperactivity and excess fluid retention. The residual urine was 500 cc per day with diuretics; frusemide 40 mg twice a day. They also mentioned that increased sympathetic activity was recorded in patients with severe hypertension (Acelajado et al., 2019). Then, beta blocker was prescribed (Carvedilol 12.5 mg BD); it was titrated with heart rate. A mineralocorticoid receptor antagonist- spironolactone (Aldactone 25 mg OD) was added; it was monitored with serum potassium. A blocker of the renin-angiotensin system (angiotensin-converting enzyme inhibitor or angiotensin receptor blocker (ACEI/ARB) was not tried in this case because of danger of hyperkalemia. One reason for reporting this case was to share the challenges in controlling blood pressure prior to renal transplant.

This patient did not have effect of hypertension on heart as heart size was normal in chest radiograph. She did not have ECG evidence of ischemic heart disease. It might be due to relatively young age and being a non-smoker. However, she had evidence of atherosclerosis in carotids; raised carotid intima media thickness in both carotids. Having premature atherosclerosis in this patient was due to severe hypertension and chronic kidney disease. Among the causes of premature atherosclerosis (systemic lupus erythematosus, diabetes mellitus), CKD/ESRD is one of them. It is one reason for case reporting.

In the study done by Jain, in patients with ESRD, the highest percentage of eyes affected were with Grade 3 Hypertensive Retinopathy (Jain, Jain, et al., 2024). Additionally, hypertension related microvascular disease was found to be severe in renal transplant recipients and subjects with CKD stage 5/ESRD (Arriozola-Rodríguez et al., 2015)(Ooi et al., 2015). Omotoso et al studied retinal changes among Nigerian hypertensive patients and they found that hypertensive retinopathy changes were associated with impaired renal function (Omotoso et al., 2016) (Ślizień et al., 2019). This patient had Grade 4 Hypertensive Retinopathy; papilledema, flame shaped hemorrhages, arterio-venous nipping and silver wiring. Having Grade 4 Hypertensive Retinopathy reflected severe hypertension with target organ involvement. It took few months to develop retinal changes. Therefore, having Grade 4 Hypertensive Retinopathy in this case was in accordance with previous findings (Jain, Sen, et al., 2024) (Berindán et al., 2017) (Ginu et al., 2021) (Ślizień et al., 2019). It again pointed out the need for interdisciplinary partnership between nephrologists and ophthalmologists.

The severity of hypertensive retinopathy may improve following good control of blood pressure. The timing to take such changes in fundoscopy was rarely mentioned. This patient had clinical evidence of effect of severe hypertension on retina (Grade 4 Hypertensive Retinopathy) prior to renal transplant. The effect of reduction of blood pressure after transplant was well recognised in follow up fundoscopy; Grade 2 Hypertensive Retinopathy. In this case, retinopathy improve over one month. This is the main reason for case writing. It also highlighted the need for monitoring fundoscopy in renal transplant recipient; collaboration between ophthalmologist and renal transplant team. Blood pressure reduction in post-transplant period reflected reversibility of severe hypertension after living donor renal transplant; it would be a rescue-therapy for severe hypertension in patients on maintenance haemodialysis. And, fundoscopic changes pointed out the reversibility of effect of severe hypertension on target organ- retina.

Balagobi et al reported bilateral nephrectomy in 16-year-old boy with severe hypertension; it resulted in satisfactory control of hitherto refractory hypertension. The boy had multiple life-threatening episodes of hypertensive crises (Balagobi et al., 2022). In this case, the blood pressure became 140/90 mmHg with Nifedipine SR 10 mg daily after transplant. There was a significant reduction in blood pressure levels and number of antihypertensive medications used after renal transplantation. This is another reason for case reporting. Most of the reports on kidney transplant illustrated the changes of blood pressure higher than pre-transplant level (Seeman, 2009) (Kuźmiuk-Glembin et al., 2018) (Opelz & Döhler, 2005) (Jaques et al., 2021) (Agarwal et al., 2023) (Pagonas et al., 2019). They emphasized the importance of controlling blood pressure to improve graft survival and mortality. However, one report revealed changing blood pressure to lower than pre-transplant level (Kubo et al., 2016). Therefore, this case was a rare form.

## CONCLUSION

Severe hypertension may be a complication or the cause of CKD/ESRD. It is frequently seen in patients on maintenance haemodialysis; it is not easy to control with both pharmacological and non-pharmacological measures. Hypertensive retinopathy reflects the complication and severity of hypertension on target organ in addition to its effect on heart, brain and kidney particularly in patients on maintenance haemodialysis. The reversibility of severe hypertension after living donor renal transplant is evidenced by not only changes in blood pressure but also target organ involvement in retina.

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Our institution does not require ethical approval for reporting cases.

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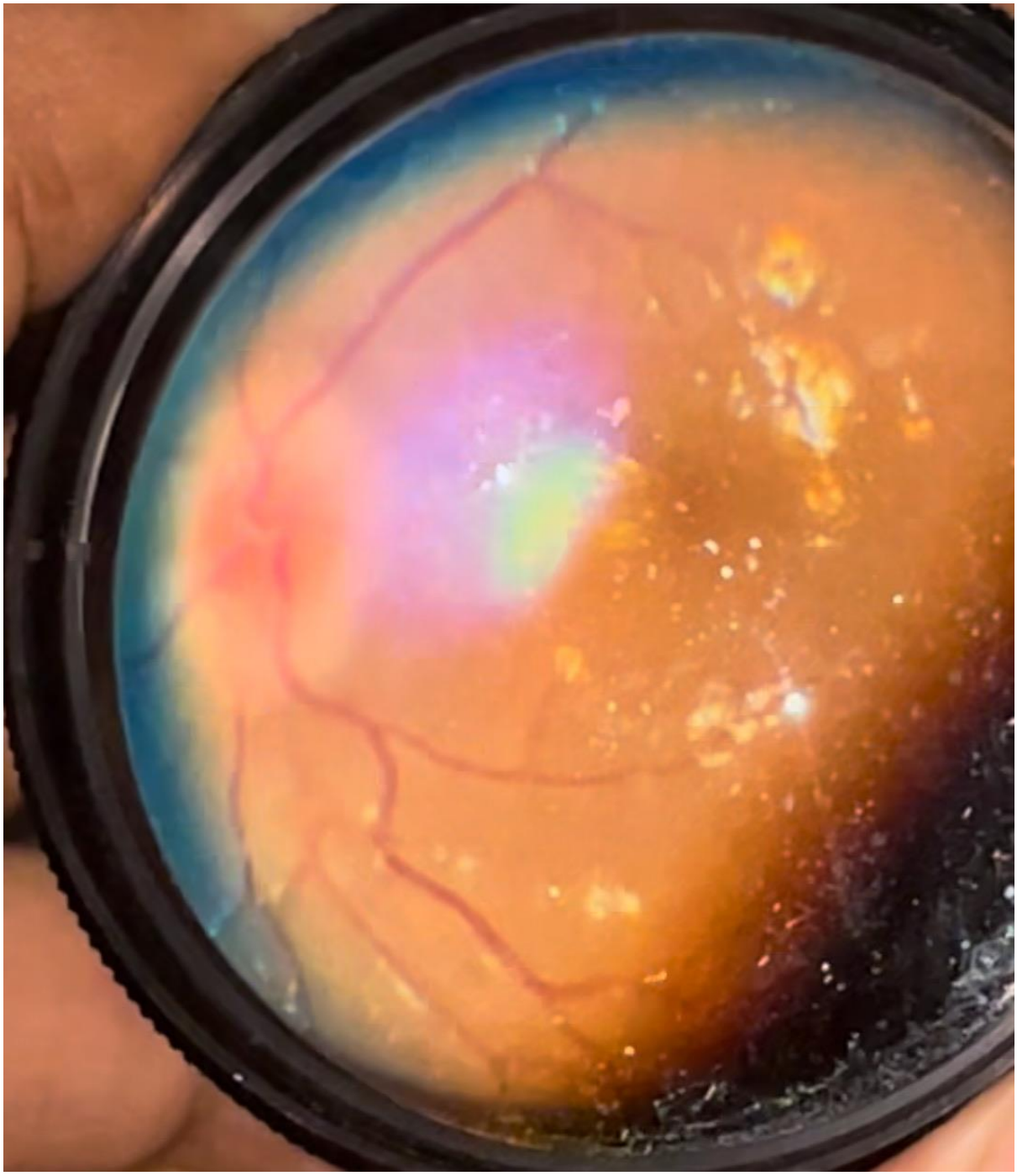

**Photo (1) Papilledema, AV nipping and multiple hard exudates in left eye prior to transplant**

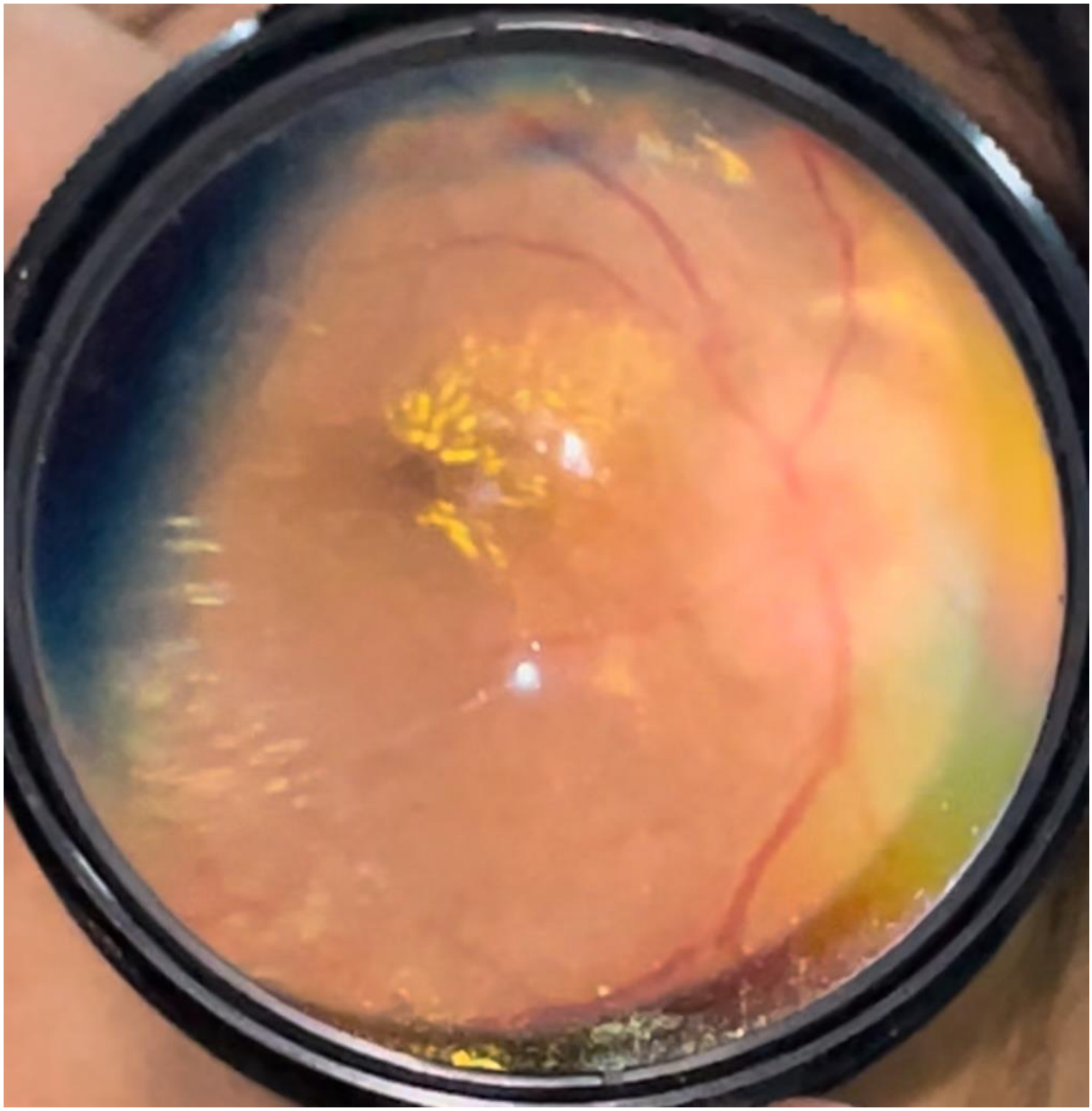

**Photo (2) Papilledema, AV nipping and multiple hard exudates near macula having macular star in right eye taken prior to transplant**

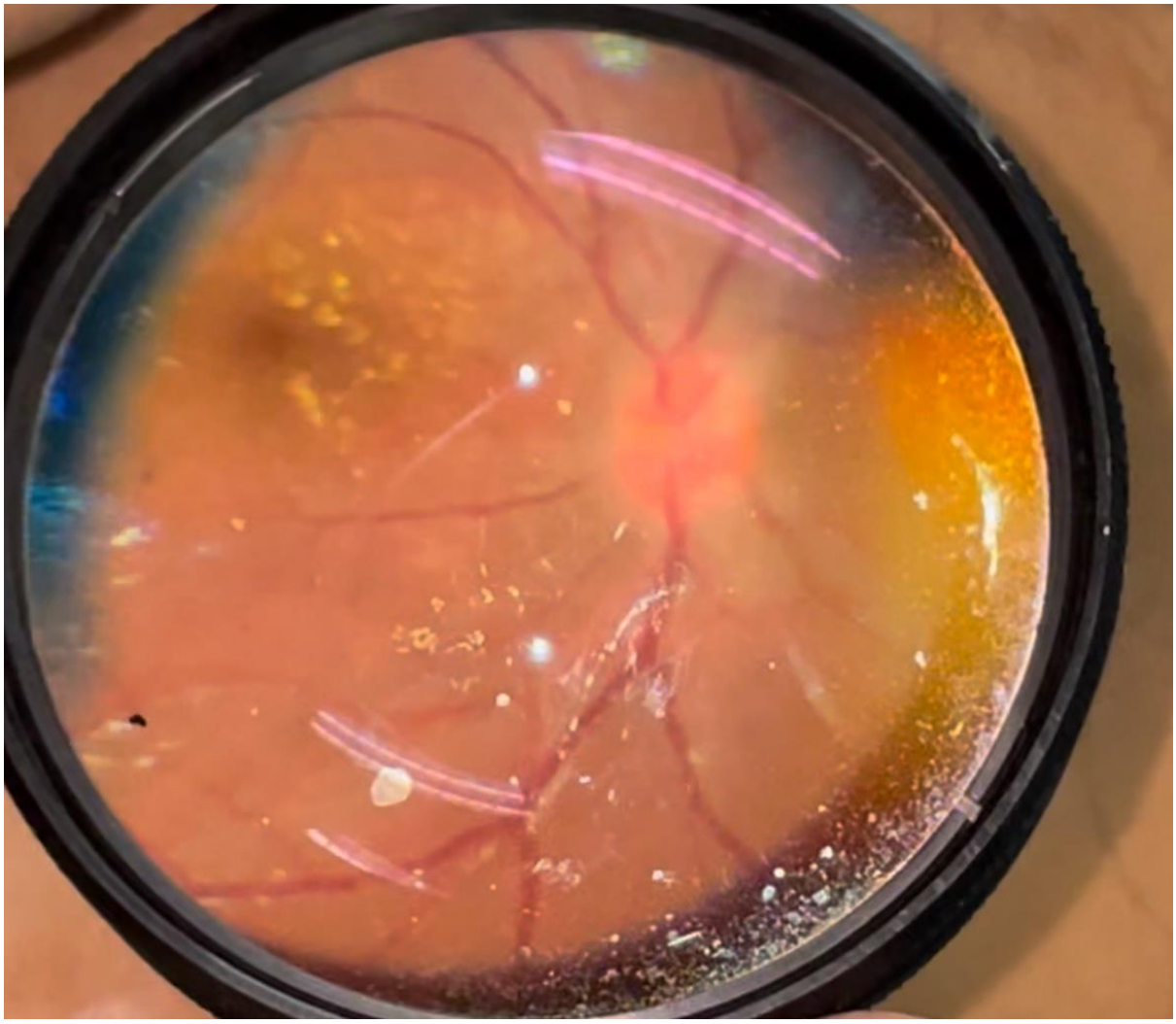

**Photo (3) Normal optic disc of left eye taken 2 months after transplant**

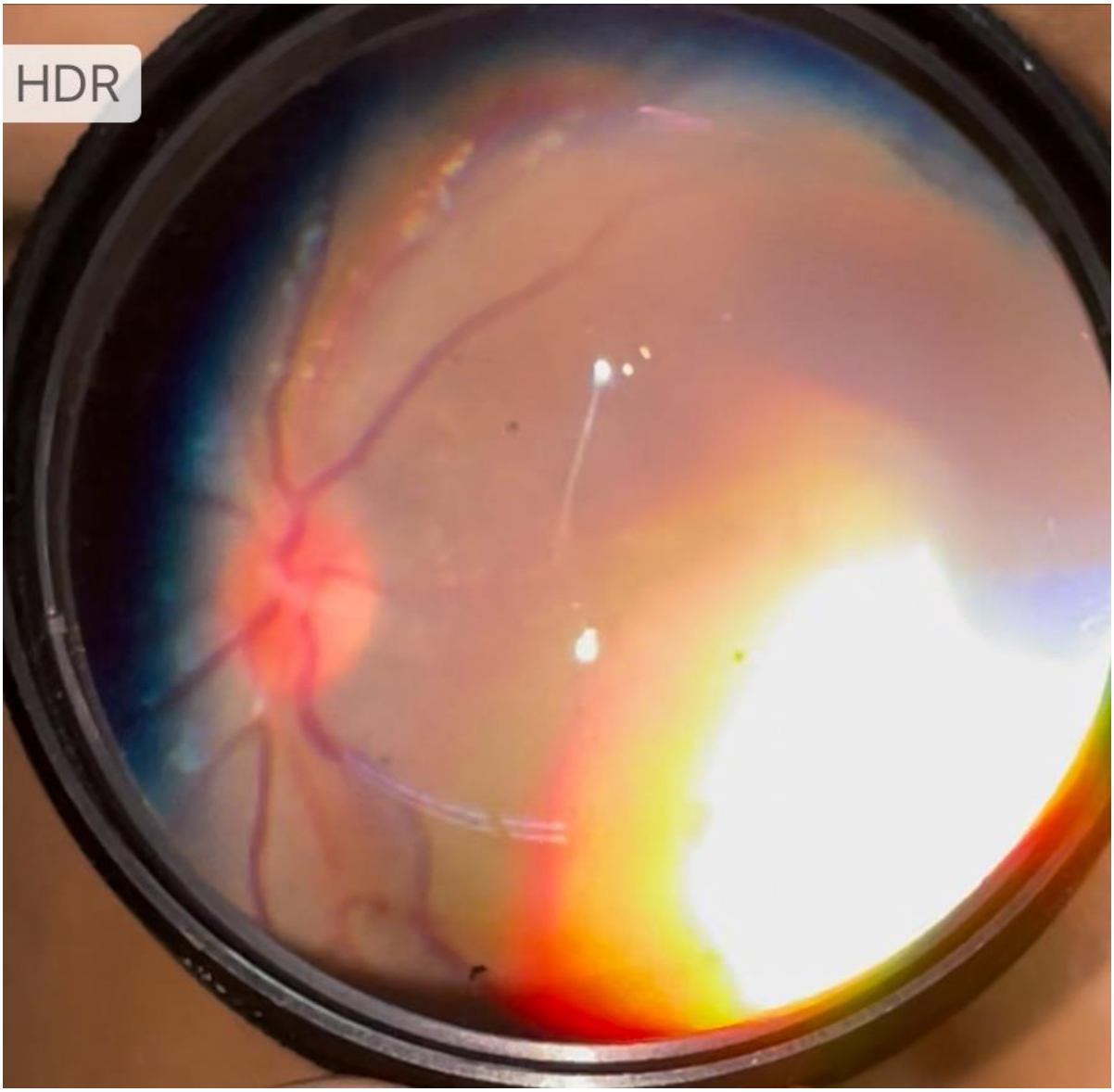


**Photo (4) Normal optic disc of right eye taken 2 months after transplant**

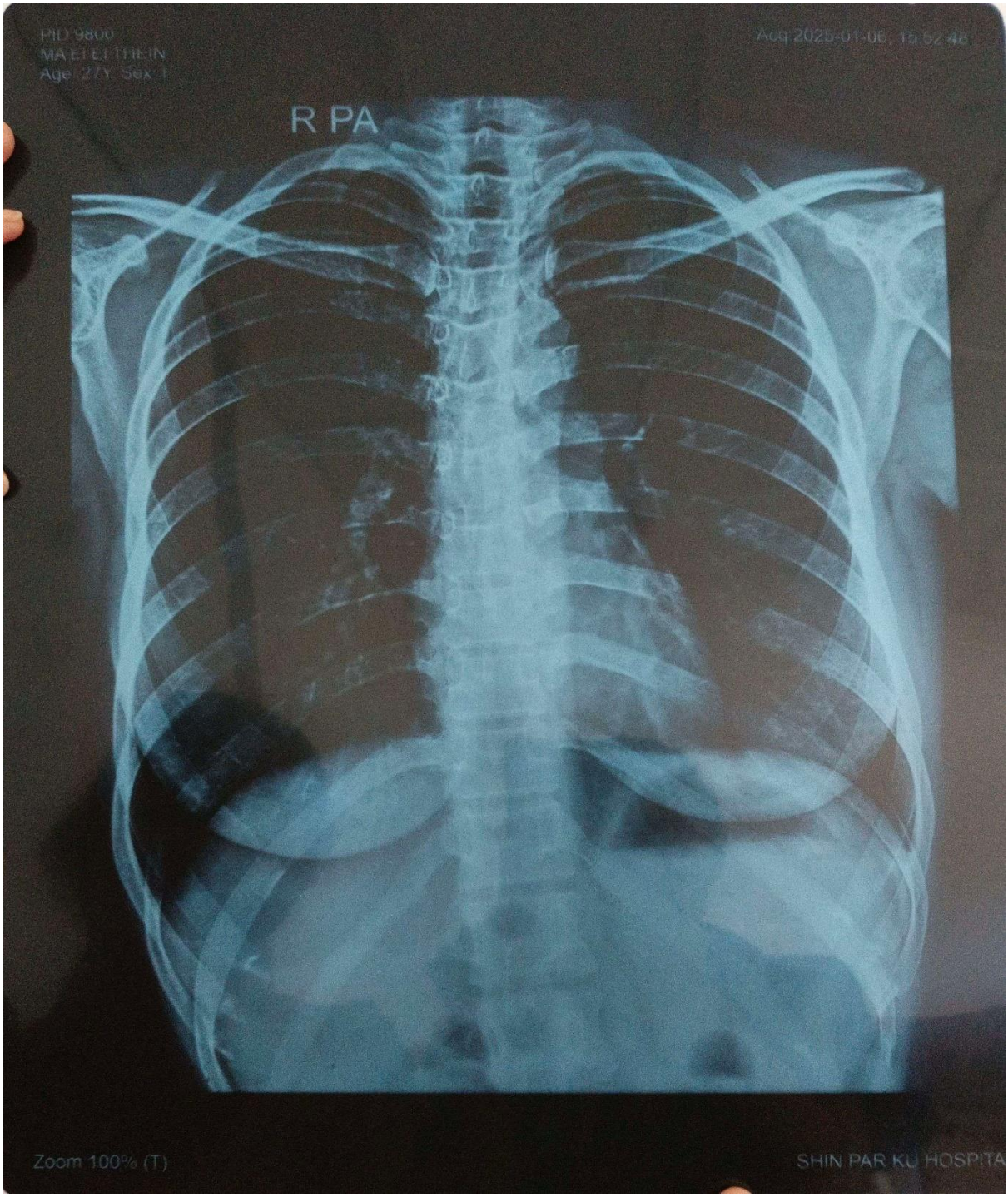


**Photo (5) Chest radiograph taken before renal transplant**

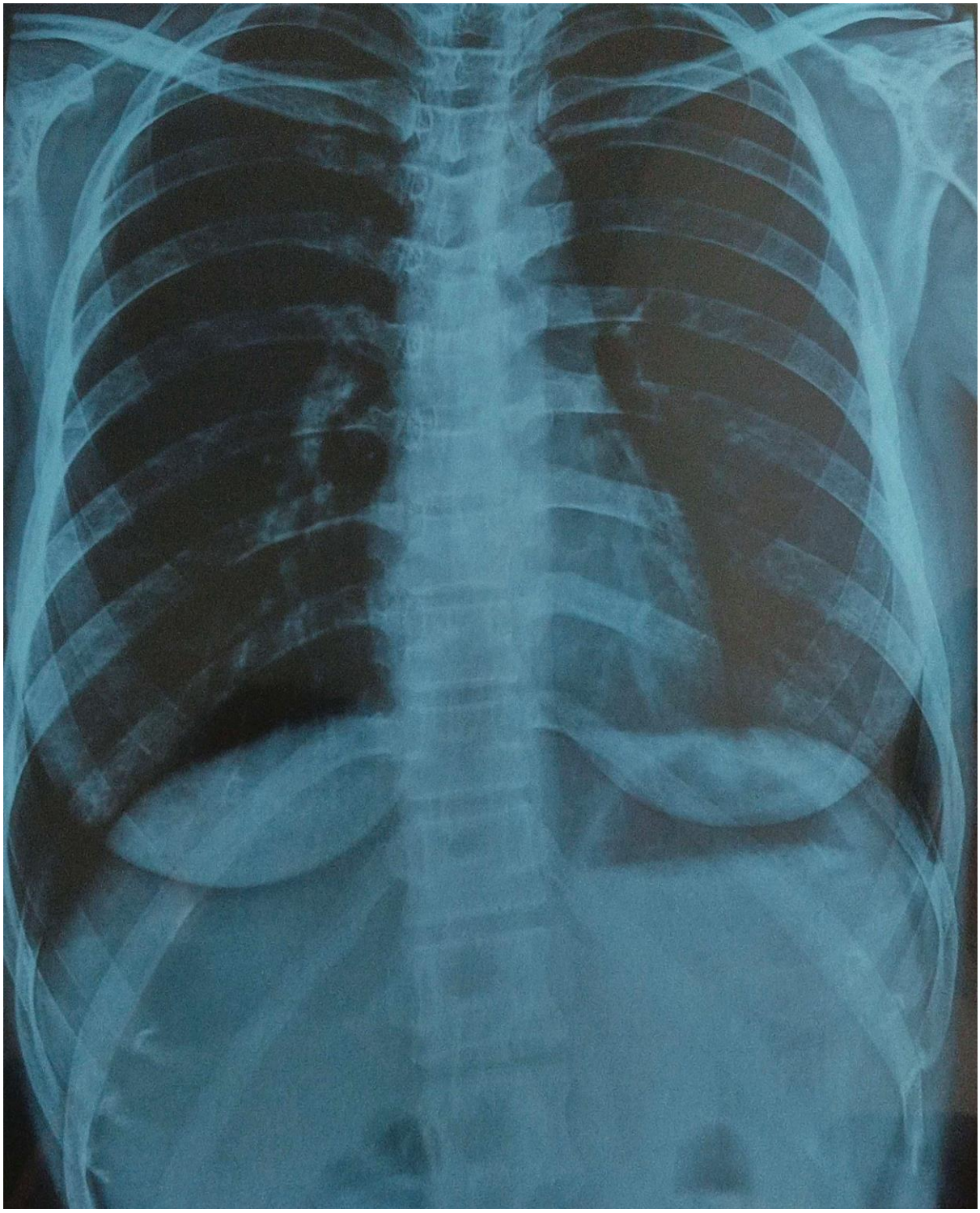

**Photo (6) Chest radiograph taken one week after renal transplant**