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Does basiliximab induction trigger life-threatening ARDS and shock in young patients after kidney transplantation?

A case report and literature review

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Key words

renal transplantation
– basiliximab – shock –
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(ARDS) – induction

Abstract. Background. Kidney disease Improving Global Outcomes (KDIGO) guidelines strongly recommend administering an anti-IL-2R mAb (i.e., basiliximab) for induction in all kidney transplant recipients. We describe a life-threatening episode of shock following basiliximab injection and review the literature. Methods and Results: A 20-year-old male was given tacrolimus, methylprednisolone, mycophenolate, and basiliximab, 20 mg in the context of living-related kidney transplantation. On post-operative Day 1 (POD 1), he developed acute respiratory distress syndrome (ARDS), shock, multiple organ failure, and had a cardiac arrest. After effective resuscitation, he received rescue therapies (NO inhalation, extra-corporeal membrane oxygenation, and CVVHD) but lost the graft as the result of cortical necrosis. We conducted PubMed searches that yielded 7 similar cases; 6 required invasive ventilation. Three patients developed cardiac arrest, 3 required major inotropic support, and 2 developed MOF and myocardial depression. All but 1 patient recovered rapidly within a few days. There was no evidence for infectious, allergic, or over-hydration concerns. Although the direct causal role of basiliximab cannot be formally proven, the fact that ARDS at the time of induction therapy with other immunosuppressive agents is otherwise extremely rare suggests a direct role for basiliximab. Conclusions: Basiliximab could be associated with shock and ARDS.

nist (IL-2RA) should be administered to all adult kidney transplant recipients [1]. Indeed, the most recent meta-analysis shows that, in comparison to placebo, IL-2RA reduces the incidence of acute rejection and improves renal graft survival [2]. Importantly, IL-2RA has proven to be as well tolerated in large randomized controlled trial (RCT) as placebo in both adults [3, 4, 5] and children [6, 7]. Basiliximab (Simulect®, Novartis Pharma AG, Basel, Switzerland) is the most widely used IL-2RA today and has been administered to more than 4,000 patients as induction therapy [8]. It is a chimeric monoclonal antibody, with the entire Fab variable region coming from the parental mouse antibody, while the constant regions are made of human IgG1 molecules. Rare cases of IgE-sensitization, responsible for anaphylactic reactions, have occurred, particularly on re-exposure after a previously failed graft [9, 10, 11, 12].

We describe an unusual, non anaphylactic, life-threatening episode of shock following basiliximab injection. We also reviewed the published literature about the possible association between shock and daclizumab (a humanized anti-IL-2RA), ATG preparations (depleting anti-lymphocyte polyclonal antibodies), OKT3 (a murine monoclonal antibody directed against T-cells), alemtuzumab (a monoclonal anti-CD52 antibody that causes profound lymphocyte depletion), as well as other immunosuppressive agents.

Introduction

KDIGO guidelines recommend that induction with an anti-IL-2 receptor antago-

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Subject and methods

Patient description

At 2 1/2-year-old, this male Caucasian patient suffered from severe hemolytic uremic syndrome following salmonella B enteritis. He finally recovered after a total of 13 days of anuria but developed progressive kidney failure in the subsequent years. At the age of 20, he underwent pre-emptive paternal kidney transplantation. His peak and current panel reactive anti-HLA antibodies were null. He had 0/1/1 HLA A/B/DR incompatibilities with his father. Donor and recipient CMV IgG status were respectively negative and positive. An inaugural hemodialysis session was performed the day before transplantation because of azotemia at 320 mg/dl along with metabolic acidosis with HCO_3^- at 15 mEq/l. On the day of transplant, he was given 4 hours pre-operatively cyclosporin (7 mg/kg, 350 mg po), methylprednisolone (250 mg i.v.), mycophenolate (720 mg po) together with acetylsalicylic acid (100 mg po). Anesthetic drugs consisted of remifentanyl, cisatracurium, and propofol. Two grams of cefazoline were given just after anesthesia induction. Intravenous basiliximab (20 mg) was administered for 30 minutes just before the vascular clamps were removed. The transplantation surgery was uneventful. Post-operative analgesic treatment consisted of piritramide and paracetamol at recommended doses. At this stage, he did not require any blood transfusion. Cold and warm ischemia times were 2 hours, 40 minutes, and 31 minutes. In the post-transplant period, he was given ranitidine, acetaminophen, and again 1 g of cefazoline. In total, the patient passed 500 ml urine in the operating room but shortly after became oliguric with an average urine output of 25 ml/h during 6 hours. He was prescribed 3 times 500 ml hydroxyethylstarch 130/0.4 (Voluven, Fresenius Kabi, Sèvres, France) and one red blood cell unit 8 hours after surgery. Nine hours after the end of surgery, his central venous pressure (CVP) increased from 8 to 16 mmHg. He rapidly developed respiratory failure, with drop in oxygen saturation from 100% to 79%. The patient was admitted to the ICU, the trachea was intubated, and mechanical ventilation was initiated. Chest X-rays showed bilateral

infiltrates. A continuous infusion of furosemide was started. Renal echography showed permeable renal artery and vein. The patient developed hemodynamic instability. He then immediately received 2×1 mg ephedrine with i.v. fluids, followed by a continuous infusion of 200 g/min norepinephrine. Arterial blood gases showed a pH of 6.96; a PaCO_2 of 53 mmHg and a PaO_2 of 46 mmHg under controlled volume ventilation at tidal volume of 300 ml, respiratory rate 36/min, positive end-expiratory pressure (PEEP) 5 cm H_2O and F_iO_2 100%. Laboratory tests demonstrated multiple organ failure. Despite rapid escalation of ventilatory support (PEEP up to 15 cm H_2O) he eventually underwent a cardiac arrest. After return of spontaneous circulation, a trans-esophageal echocardiography showed a dilated right ventricle, a global hypokinesia and an inhomogenous contraction, which were not present at an echocardiography done 15 days before. There was no foramen ovale. A pulmonary artery catheter was inserted. It showed a cardiac output of 5.9 l/min (cardiac index 3.7 l/min/ m^2), a pulmonary artery pressure of 21/10 mmHg, and a wedge pressure of 4 mmHg. The mixed venous saturation in oxygen was 46%. This was compatible with general hypovolemia and pulmonary hypertension, suggesting a distributive shock with subsequent acute respiratory distress syndrome (ARDS), acute cor pulmonale, and myocardial dysfunction.

As the patient failed to respond to usual therapies (high doses of vasopressor and inotropic agents combined with protective lung ventilation), we were forced to institute rescue therapies such as nitric oxide (NO) inhalation, rapidly followed by veno-arterial Extra-Corporeal Membrane Oxygenation (ECMO). Continuous renal replacement therapy (CRRT) was also initiated. After ECMO was started, the patient's condition stabilized. The ECMO F_iO_2 was set at 100% but was rapidly weaned after 24 hours. ECMO was maintained for hemodynamic support and was weaned off completely 30 hours after insertion. The patient had fully recovered at POD 3 and was extubated. He was discharged from ICU at POD 6. CRRT was continued until and after discharge from ICU. Two weeks after his transplantation, a control trans-thoracic echocardiography showed a normal biventricular function with

Table 1. History and demographic features of the patients having suffered from ARDS following basiliximab injection*.

	Age, gender	Nephropathy	Ethnic group	Prior renal replacement therapy (dialysis vintage)	Rank of the graft, history	Allergy history
1	17, F	Renal hypodysplasia	Hispanic	No	– First	NA
2	21, F	Focal segmental glomerulosclerosis	African-American	No	– Third – Second deceased donor renal graft lost as a result chronic rejection and disease recurrence; complicated by OKT-3 induced non-cardiogenic pulmonary edema and dilated cardiomyopathy – First transplantation with mother kidney lost from accelerate acute rejection	NA
3	19, M	Juvenile nephronophthisis	African-American	PD (NA)	– Second – First living related renal transplant lost within 24 h to graft thrombosis (subsequent protein C deficiency discovery → low molecular weight heparin)	NA
4	15, M	NA	NA	HD (NA)	– First	NA
5	19, F	Ectodermal dysplasia syndrome	NA	PD (60 months)	– First	NA
6	6, M	Posterior urethral valves and renal dysplasia	Caucasian	PD (20 months)	– First	No
7	11, M	Posterior urethral valves and renal dysplasia.	Caucasian	HD (< 36 months)	– First. History: had two episodes of pulmonary embolism due to central venous line thrombosis	No
8	20, M	Hemolytic uremic syndrome	Caucasian	First HD pre-operatively	– First. Pulmonary edema at 3 years in the context of acute renal failure pancreatitis	No

*References: Bamgbola et al. [13] (Patients 1 – 3), Skorokhod et al. [15] (Patients 4 and 5), Dolan et al. [14] (Patients 6 and 7), Massart et al. [this case] (Patient 8). HD = hemodialysis, NA = data not available, PD = peritoneal dialysis.

a persisting mild pulmonary hypertension. Unfortunately, graft function was lost due to cortical necrosis. The patient was subsequently explanted. At this occasion and also years later after a second kidney transplantation the patient received the same anesthetic, antalgic, and antibiotic drugs without complications. We retained the diagnosis of idiosyncratic reaction to basiliximab associated with severe acute respiratory failure (ARDS) and circulatory failure.

Review of the literature

We carried out PubMed searches using the following MESH terms: “antibodies monoclonal, basiliximab, daclizumab, anti CD25 antibody, alemtuzumab, thymoglobulin, OKT3 and kidney transplantation” combined, successively, with either, “pulmonary edema, acute lung injury, respiratory distress syndrome, adults, or shock”. An extra search about “pulmonary capillary leak syndrome and kidney transplantation” was also conducted.

We retrieved reports on severe, unusual, respiratory, or shock manifestations and excluded the papers providing evidences of septic, anaphylactic, or hypervolemic contexts.

All these searches were obtained for the last time on August 26, 2012.

Results

Basiliximab induction

We found 6 similar cases [13, 14]. Novartis® informed us of one paper not found otherwise [15].

Demographic and clinical features (Table 1). The 8 patients ranged from 6 to 21 years of age, were from various ethnicity and were well-balanced regarding gender repartition. Five were suffering from renal dysplasia. No one had a history of allergies. It was the first transplantation for 6 of the patients and the first basiliximab injection for all of them. The rest of their treatment was usual in the context of transplantation and anesthesia. Two

Table 2. Immunosuppression of the patients having suffered from ARDS following basiliximab injection*.

	Type of donor	Total ischemia times	Immunosuppressors given peri-operatively
1	DD	23 h	– Basiliximab, 12 mg/m² over 30 min 2 – 4 hours before surgery – tacrolimus 0.15 mg/kg 6 – 12 hours pre-transplant – methylprednisolone given i.v. intraoperatively
2	LD, familial	1.25 h	Id
3	DD	24 h	Id
4	LD	NA	– Basiliximab, 20 mg i.v. over 30 min following the induction of anesthesia – tacrolimus – mycophenolate – methylprednisolone
5	LD	NA	– Basiliximab, 20 mg i.v. over 30 min following the induction of anesthesia – tacrolimus – mycophenolate – methylprednisolone
6	LD	NA	– Basiliximab, 2 hours prior to surgery, 10 mg if BW < 40 kg, 20 mg if BW > 40 kg, in 50 ml 0.9% NaCl over 30 min – tacrolimus, 0.1 mg/kg – methylprednisolone – mycophenolate mofetil commenced post-operatively
7	DD	NA	Id
8	LD, familial	3.5 h	– Basiliximab 20 mg, i.v., intraoperatively – cyclosporine (7 mg/kg, 350 mg) – mycophenolate – methylprednisolone

References: Bamgbola et al. [13] (Patients 1 to 3), Skorokhod et al. [15] (Patients 4 and 5), Dolan et al. [14] (Patients 6 and 7), Massart et al. [this case] (Patient 8). BW = body weight, DD = deceased donor, LD = living donor, NA = data not available, PRA = panel reactive antibodies.

patients had a previous history of thrombosis (graft thrombosis, pulmonary embolism).

Immunosuppression data (Table 2): There were 4 living donors and 4 deceased ones. Basiliximab was administrated before (n = 5) or during the surgery. The rest of the immunosuppressive treatment consisted of calcineurin inhibitors (tacrolimus in 7 cases, cyclosporin in 1 case), methylprednisolone (n = 8), and mycophenolate (n = 5). No patient was HLA-sensitized (data known for 4 patients). Average ischemia time was 13 hours (n = 4). No patients underwent previous or further basiliximab injection.

Volume assessment (Table 3): No patients had previous history of cardiac dysfunction (7 echocardiograms done/8). After operation, no patients had evidence of hypervolemia according to chest X-rays, albumin or hematocrit levels, fluid balance, or central venous pressure. Seven patients passed urine abundantly just after the transplantation.

Clinical features and outcomes (Table 4): After initially being well for some hours (max 48 hours), all patients developed ARDS. Six

patients required invasive ventilation in a volume-control mode, one required non-invasive ventilation, and the last was only given oxygen through a face-mask. Extra-pulmonary manifestations developed in most patients, including: arrhythmias (n = 1), need for massive inotropic support (n = 3), multiple organ failure with myocardial depression (n = 2), acute renal failure (n = 4), and disseminated intravascular coagulation (n = 2). There was no evidence of infectious context. Three patients developed cardiac arrest and one died. Overall, four grafts were lost (death, n = 1), acute antibody-mediated rejection with post-transplant de novo microangiopathy (n = 1), graft necrosis (n = 2). The other 4 patients recovered fully in a few days and had functioning grafts at one year.

Other induction agents

Daclizumab was not suspected to trigger any case of pulmonary edema or shock. Of note, in a trial studying reperfusion edema fol-

Table 3. Volume assessment of the patients having suffered from ARDS following basiliximab injection*.

	Pre-transplant echocardiogram data	Pre-transplant diuresis	Cardiothoracic ratio	Post-operative diuresis	Post-operative central venous pressure (cm H ₂ O)
1	NA	Normal urine output	< 0.6	Prompt	5 – 10
2	SF 30,5%	Normal urine output	< 0.6	Prompt	NA
3	SF 35%	NA	< 0.6	Prompt then delayed graft function	6 – 11
4	NA	NA	NA	Prompt and negative fluid balance post-operatively	5 – 11
5	Normal LVEF	NA	Enlarged cardiac shadow. Tamponade ruled out by pericardial window.	NA	NA
6	Normal LVEF	1.5 l/day	NA	Negative fluid balance with furosemide	1
7	Normal LVEF	2.5 l/day	NA	2 l urine intraoperatively	1,3
8	Normal LVEF	Normal urine output	0.39	500 ml intraoperatively then 25 ml/h	4

*References: Bamgbola et al. [13] (Patients 1 to 3), Skorokhod et al. [15] (Patients 4 and 5), Dolan et al. [14] (Patients 6 and 7), Massart et al. [this case] (Patient 8). LVEF = left ventricular ejection fraction, NA = data not available, SF = left ventricular shortening fraction.

Table 4. Clinical features and outcomes of the patients having suffered from ARDS following basiliximab injection*.

	Day of onset	Pulmonary and extra-pulmonary manifestations	Cardiac evaluation	Duration (days)	Treatment	Issue
1	POD 2	ARDS	Normal	7	Non-invasive ventilation	Screat. 0,8 mg/dl at one year
2	POD 1	ARDS, lethargy, central cyanosis.	Normal	3	Invasive ventilation	Screat 1 mg/dl at one year
3	POD 1	ARDS, cardiorespiratory arrest at POD 3. Retroperitoneal hemorrhage at autopsy	Normal	Not applicable	Invasive ventilation. Hemodialysis	Death on the POD 3
4	POD 1 (+ 9 h)	ARDS	Normal	3	Oxygen without ventilation	Unremarkable
5	POD 1	Minutes of bradycardia and decreased blood pressure just after graft reperfusion; bradycardia, cardiogenic shock, ventricular fibrillation, cardiorespiratory arrest ; multiple organ failure with ARDS (+ 48 h)	Abnormal	Several	Intubation for hemodynamic instability, intraortic balloon pump, inotropic support.	Graft loss; otherwise banal, discharge from the hospital 2 weeks later
6	POD 2	ARDS	Normal	10	Mechanical ventilation	Screat 0,53 mg/dl
7	POD 1 (+ 5 h)	ARDS, shock	Normal	5	Mechanical ventilation Inotropic support	Graft loss within 3 PO weeks due to thrombotic microangiopathy and severe acute antibody-mediated rejection
8	POD 1 (+ 9 h)	ARDS, shock, multiple organ failure, disseminated intravascular coagulation, myocardial depression, cardiorespiratory arrest	Abnormal	3	PRVC ventilation, inotropic support, CVVHD inhalation of NO, ECMO, empirical antibiotherapy	Immediate graft loss by cortical necrosis

*References: Bamgbola et al. [13] (Patients 1 to 3), Skorokhod et al. [15] (Patients 4 and 5), Dolan et al. [14] (Patients 6 and 7), Massart et al. [this case] (Patient 8). CVVHD = continuous veno-venous hemodiafiltration, ECMO = extracorporeal membrane oxygenation, NA = data not available, POD = post-operative day, PRVC = pressure regulated volume control.

lowing lung transplantation [16], daclizumab was found to be equivalent to placebo.

Thymoglobulin was associated with two reports describing the occurrence of ARDS in 2 patients. The first one [17] was a 21-year-old man who developed ARDS 5 hours after the completion of a second Minnesota horse ALG (850 mg), given for induction after renal transplantation. He required mechanical ventilation for up to 2 weeks but the outcome was favorable. In a second case report [18], a 57-year-old woman received ATG (20 mg/kg, without premedication) for aplastic anemia. Her respiratory status started to decline 3 hours after the infusion of a second dose of ATG until she finally died from respiratory failure. In both cases, a hemodynamically driven edema could be reasonably ruled out by clinical evaluation and fluid removal treatments. No multisystemic involvement was reported.

OKT3: Dyspnea and wheezing are commonly observed (1 out of 5 cases) after the first or, to a lesser extent, the second OKT3 injection [19]. However, pulmonary edema remains quite a rare adverse event reported to occur in less than 2% [19, 20] of kidney recipients and up to 10% of heart recipients [21]. Several risk factors were identified, such as pre-existing fluid overload greater than 3% above the ideal weight [19, 22], pancreas transplantation [23], and the absence of uremia in preemptive transplantations [23]. This complication was described as moderate in the context of heart transplantation as it did not result in death or permanent damage [21]. However, in the early experience of treatment of acute renal allograft rejection, 2 fatalities were reported among 5 patients after the initial OKT3 injection (reported unpublished data from [19]). In retrospect, these 5 subjects were all suffering from marginally compensated cardiac failure [22]. Below, we detail three unusual and life-threatening reactions arising in the context of OKT3 injection. First, Lee et al. [24] described a 53 year-old renal transplanted man who received OKT3 for steroid resistant rejection on Day 19 post-transplantation. He developed seizure and cardiac arrest 1 minute following 5 mg OKT3 injection, and a similar phenomenon after the second OKT3 dose. Of note, he had a past medical history of mild concentric left ventricular hypertro-

phy before the transplantation. Then, Greenfield [25] described a 28-year-old man with end stage renal disease following Type 1 membranoproliferative glomerulonephritis under OKT3 induction for his first renal transplantation. He had no past cardiac medical history. After the third 5 mg OKT3 dose in 3 days, he suffered from progressive respiratory failure and then ARDS so severe that he eventually required extra-corporeal membrane oxygenation. He could be weaned off it after only 68 hours and came back home 1 month later with only slightly impaired graft function. Finally, Thillainathan et al. [26] reported on a 55-year-old diabetic renal transplant recipient who developed hypoxemia, shock, and cardiac arrest ~ 12 hours after receiving a first OKT3 injection for kidney rejection. He died on the day following the infusion.

Alemtuzumab was incriminated in two acute chest syndrome presentations. First, Sachdeva et al. [27] reported a diffuse alveolar hemorrhage complicating alemtuzumab injection in the context of renal transplantation for a X-linked Alport syndrome. Diagnoses of infectious pneumonia or anti-GBM disease were ruled out. The second case consisted of peri-operative pulmonary edema [28] with progressive hypotension requiring dopamine infusion in a 22-year-old man undergoing a second kidney transplantation for polycystic kidney disease.

Other immunosuppressive drugs

While 16 cases of systemic capillary leak syndrome [29, 30] have been reported after bone-marrow transplantation, cyclosporine related-ARDS in the context of liver, kidney, or combined kidney-pancreas transplantation is distinctly rare (11 patients [31, 32, 33, 34, 35]). Most ARDS cases (n = 9) occurred after the i.v. use of cyclosporine and could have been due to cremaphor, the solvent of the i.v. formulation. Only 2 cases occurred after induction with oral cyclosporine. For solid organ transplantation, clinical presentations were those of pulmonary capillary leak syndrome, sometimes requiring mechanical ventilation, occurring either hours or days after induction and sometimes associated with hypotensive episodes [31]. We found one

case attributed to sirolimus after exclusion of other causes [36]. It occurred 1 month after the treatment had been initiated.

Discussion

We report on a life-threatening episode of ARDS, non cardiogenic shock, and multiple organ failure in a 20-year-old patient following basiliximab injection. Besides the well-described basiliximab-associated immediate anaphylactic shock, our literature review suggests that a distinct type of complication consisting of delayed ARDS and shock could also be associated with basiliximab in young patients. Patients with anaphylactic reactions developed hypotension and allergic signs (such as pruritic rash, tongue swelling, wheezing, urticaria, skin flushing, or sneezing) within minutes or hours following basiliximab injection [9, 10, 11, 12]. This occurred either after a second or a first [12] injection of the drug. In the former case, the most common scenario was the immediate failure, within days, of a first graft when the patient received basiliximab and had his or her immunosuppressive drugs rapidly discontinued. Anaphylactic shock then occurred when basiliximab was given again at the time of re-transplantation. Anaphylactic reaction after the first or subsequent basiliximab injection is consistent, respectively, with anti-xenogenic and with either anti-xenogenic and/or anti-idiotypic anti-basiliximab IgE antibodies [9, 37].

The second group of patients ($n = 8$) reviewed here suffered from wide-ranging manifestations: from ARDS requiring only non-invasive ventilation, to shock complicated by multiple organ failure or even death after a basiliximab injection. Underlying infectious or hemodynamic processes were convincingly ruled out by the authors. These patients differ in many aspects from the former subset: they had no allergy history; they did not display allergic signs and/or symptoms; there was a several hour-delay between basiliximab injection and the onset of symptoms; they also differed in the occurrence of arrhythmias and the length of the episodes.

The occurrence of pulmonary oedema in the context of antibody induction must evoke

cytokine release syndrome resulting in pulmonary vascular leak syndrome, as reported with Thymoglobulin or OKT3 injections [38]. However, although hundreds of thousands of patients have been treated with basiliximab, there is no other evidence to date to suggest that this monoclonal antibody triggers any cytokine release syndrome.

In order to better understanding whether if the ARDS/shock syndrome associated with basiliximab was purely coincidental, or whether there was more ground for a causal relationship, we searched for the reported prevalence of similar ARDS and/or non-cardiogenic shock after kidney-transplantation, irrespective of the timing post-transplant. Our systematic search yielded a total number of 20 events for 6 different drugs. When we further restrict these events to those associated with the immediate post-operative period, this figure comes down to 7 patients, among whom 4 received cyclosporine i.v. Our impression about the extreme rarity of ARDS/non-cardiogenic shock among renal recipients receiving induction is largely supported by a study of Shorr et al. [44] reporting on the incidence of ARDS during the first 3 years after kidney transplantation with various immunosuppressive regimens, from 1994 to 1998. No patients received anti-IL2R Abs. Among 42,190 transplantations, ARDS occurred in only 1 patient within the first post-transplant month.

Other diagnostic possibilities must also be consider. Serum sickness was reported after renal transplantation with both monoclonal (rituximab) and polyclonal (ATGAM, ATG) antibody preparations [39, 40, 41]. However, antibody formation requires several days before symptoms, which consist of arthritis, persistent fever, rash, and sometimes acute renal failure, appear. In our report, both early occurrence after basiliximab administration and the clinical manifestation were not in favor of this diagnosis. Such reaction is also unlikely when the protein administered is combined with other immunosuppressive drugs [42]. Finally, serum sickness generally implies polyclonal molecules in order to give rise to immune complexes big enough to damage small vessels inside skins or kidneys [43].

Finally, the fact that daclizumab was not incriminated in similar reports raises the pos-

sibility that the severe reaction we observed with basiliximab, which is a chimeric and not a humanized anti-IL2R, was driven by anti-xenogenic antibodies. Further mechanistic investigations would be needed to better understand this issue.

To find predictive risk factors of developing such adverse events after basiliximab administration would be of great interest but is limited by our small number of observations. However, we noticed that all concerned patients were young (the oldest was 21 year-old). We do not know if this could reflect immunological reactivity or the particular primary disease encountered at these ages (mainly CAKUT), or whether this observation is purely incidental. Our observations, in addition to the failure of multiple trials to show any clinical benefit from the use of basiliximab in low-immunological risk pediatric populations [6, 7, 45, 46], cast doubts on the systematic administration of basiliximab in young kidney recipients. Clinicians should be aware of this possible life-threatening adverse reaction, and the risk-benefit ratio of the administration of basiliximab in young recipients should be carefully weighted on an individual basis.

In summary, this case and the literature review suggest that basiliximab may trigger ARDS and shock in the early post-operative course of renal transplant recipients, even when they are young and free of comorbid conditions. The mechanism behind this complication remains unknown. We suggest that the first administration of basiliximab should be performed in the setting of appropriate medical care.

Conflict of interest

The authors have no competing interests to declare.

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