Kidney **T**ransplantation in Patients with **P**lasm**a** Cell **D**yscracias (KITPAD)

A Nordic consensus treatment protocol for kidney transplantation in patients with multiple myeloma (MM) and monoclonal gammopathy with renal significance (MGRS)

2026 MM/MGRS Expert group with haematologists, nephrologists and organ transplant specialists from Sweden, Norway, Denmark and Finland.

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The Nordic Group of Kidney Transplant in Plasma cell dyscrasia:
This guideline was developed by a multidisciplinary Nordic working group of senior clinicians and researchers with expertise in haematology, nephrology, kidney transplantation and renal pathology.

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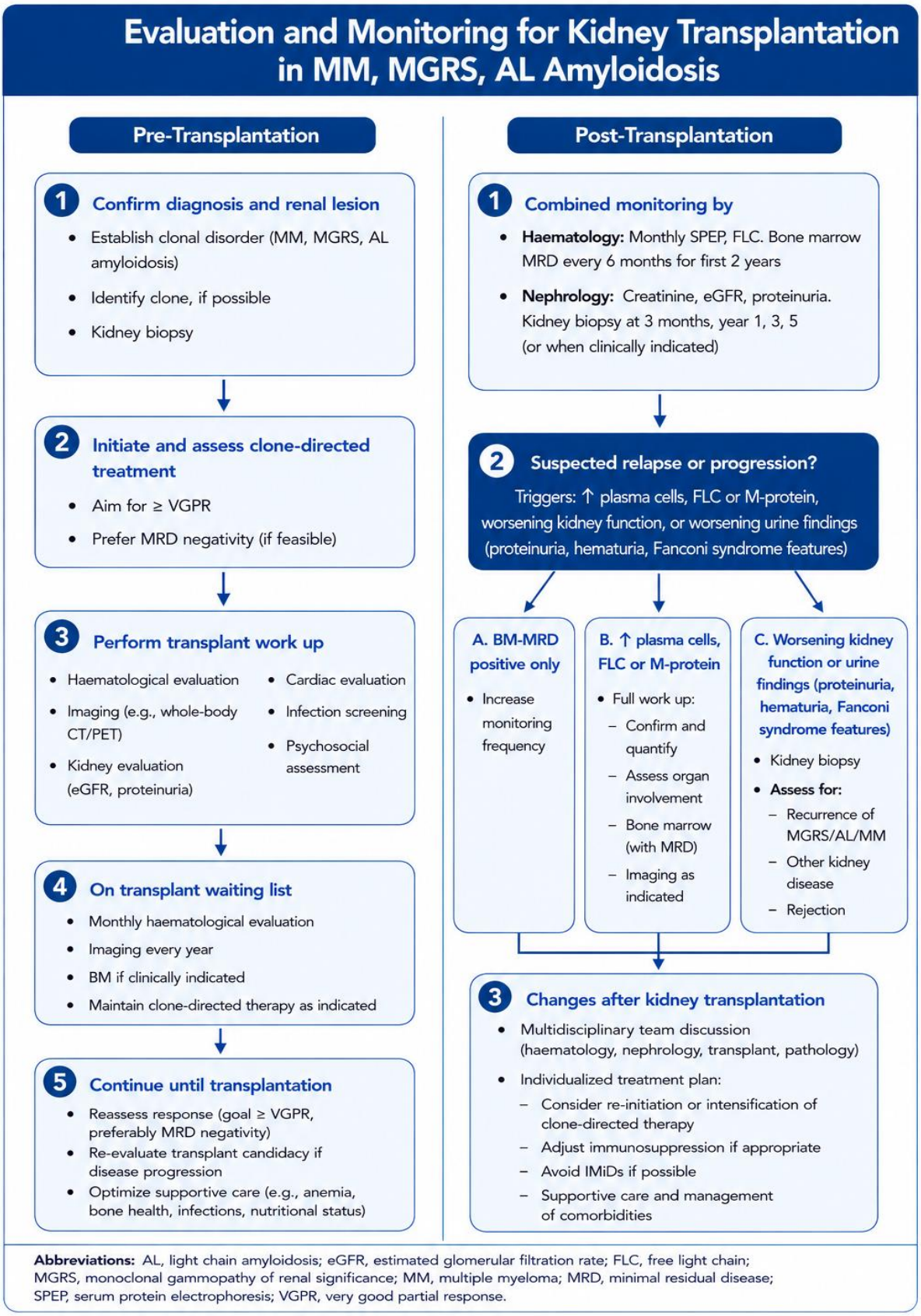
Summary of the KITPAD recommendations



1. Background

Rationale for the Nordic protocol development

There are currently no prospective trials investigating MM and MGRS and kidney transplantation and no Nordic guidelines on the treatment of MGRS outside of AL-amyloidosis. In 2020, the Transplant Centre at Sahlgrenska University Hospital in Gothenburg started the Kidney transplantation program for patients with multiple myeloma and these experiences serve as a template for this Nordic protocol. **The Nordic Group of Kidney Transplant in Plasma cell dyscrasia** was formed of haematologists, nephrologists and kidney transplant experts in Sweden, Norway, Finland and Denmark, and have taken part in writing this protocol in meetings in the time period of 2024-2026.
Having a multidisciplinary approach within the nephrology, the transplant and the haematology units both before and after transplant is crucial in maximizing the chance of successful kidney transplant patients in haematological disease(1-3).
National and Nordic treatment conferences will therefore hopefully be part of the work in this expert group on renal impairment in MM and MGRS to continuously follow the research front and if necessary, update the guidelines based on the current knowledge.
The guidelines are planned to be updated every 2 years as a minimum.

Aims of this protocol:
The guidelines will provide recommendations on pre transplant clone-reducing treatment, indications for transplant and risk factors aimed at Nordic haematologists and nephrologists. Recommendations on monitoring before and after kidney transplant, and maintenance and relapse treatment options are discussed with emphasis on maintaining response and preserving graft function.
We suggest a kidney transplant procedure at the participating centres' discretion. Protocol biopsies from the transplant kidney and bone marrow and blood tests are recommended performed for a standardized monitoring of remission and Minimal Residual Disease (MRD) of the B-cell/plasma cell clone.
With close monitoring of the plasma proteins in the blood, MRD in the bone marrow and standardized kidney transplant biopsies we hope to detect early recurrence of the B-cell/plasma cell disease and have a standardized protocol treatment based on the best expert consensus available and so improve the prognosis of these patients.
We believe that these guidelines will enable us to determine the pre- and post-transplantation disease characteristics in MGRS and MM with renal failure, prognostic factors and outcomes after kidney transplantation and raise awareness of the MGRS disease in the Nordic countries.

2. Introduction

Despite great progress in multiple myeloma (MM) clone-directed treatment, patients with MM with kidney failure at diagnosis have a high morbidity and mortality(4).

Case series and case reports have historically been published reporting kidney transplant in MM and MGRS, but recurrence of the B-cell/plasma cell disease is a huge problem with loss of graft and reduced survival in this group(5-7). Patients with MM and AL amyloidosis now have effective treatment protocols inducing good responses and long-lasting remissions(8), making an increasing number of patients eligible for renal transplant in remission. A multicentre study was performed by the International Kidney and Monoclonal Gammopathy Research Group (IKMG) with 287 AL Amyloidosis patients with renal transplant between 1987 and 2020. The study shows a median survival from renal transplant of 8.6 years and longer in patients with CR or VGPR on hematologic treatment pre transplant(9) indicating that renal transplant should be considered in this group of patients(10). The absolute incidence of MM and MGRS is increasing due to the increasing elderly population and the increase in prevalence is even greater following prolonged MM and MGRS survival thereby expanding the proportion of individuals eligible for renal function restoration.

Kidney transplantation is the best treatment option for advanced chronic kidney disease in MM and MGRS. These patients have, however, increased risk of disease recurrence after kidney transplantation(1, 9, 11). Recurrence poses significant clinical challenges, including the potential for graft dysfunction and loss. It necessitates vigilant post-transplant monitoring and may require tailored therapeutic strategies to manage and mitigate the impact on renal function. Understanding the underlying lesion´s recurrence mechanisms is crucial for developing effective interventions and improving long-term outcomes for transplant recipients. Attaining a full hematologic response could lower the risks of recurrence, graft failure, and mortality(12). Further research is imperative to discern the impact of hematologic response on outcomes specific to each MGRS-associated lesion(11).

3. Multiple myeloma (symptomatic MM) and kidney transplantation

Multiple myeloma (MM) is a haematological disease with clonal expansion of malignant plasma cells in the bone marrow with anaemia, skeletal lesions, hypercalcemia and/or kidney failure as clinical manifestations. In the last decade, with modern treatment strategies, the survival has markedly increased and is now > 10 years for patients < 60 years at diagnosis(13). In newly diagnosed patients who undergo optimal induction therapy, followed by an autologous stem cell transplantation (ASCT) and maintenance treatment, their time to next treatment can be documented to be nearly 5 years from therapy initiation(14).
Renal involvement is common in patients with MM, with about 25% of patients having renal impairment at time of initial presentation and 50% developing kidney disease during the course of their disease(15). These patients can present with several patterns of injury including cast nephropathy (CN), monoclonal immunoglobulin deposition disease (MIDD), or AL-amyloidosis. Cast nephropathy is the most common pattern occurring in 40-63% of myeloma patients with renal involvement. MIDD occurs in 19% to 26% of patients with renal involvement and 7% to 30% will have amyloid kidney involvement(1). Patients with MM and terminal kidney failure have been subjected to chronic dialysis treatment, but in the light of better MM prognosis, kidney transplant can be considered in selected patients. The collective experience is still scarce and the number of published case series is low(1).

Because of the significant advances in the treatment of multiple myeloma and improvement of long-term survival, more individuals with multiple myeloma are being referred for kidney transplantation, especially those with good functional capacity and minimal comorbidities. The current Swedish Myeloma Treatment Guidelines recommend a referral for kidney transplantation in motivated MM patients in good treatment response with severe renal failure and a prognosis of > 5 years life expectancy(16). In the Norwegian guidelines: Kidney transplantation may be performed in advanced renal failure if the kidneys are the dominant or only manifestation of disease and the overall life expectancy is otherwise good.

When to refer patients with multiple myeloma (MM)

- When in best treatment response: see criteria below
- Consider: - the risk of recurrence in the transplanted organ.
  - the risk of prolonged immunosuppressive treatment.
- Timing: a minimum of 6-12 months in remission is needed. This would give time to eliminate patients with functional high-risk disease who relapse in MM within 6-12 months of diagnosis and will not benefit from KTx.

Patient and disease criteria for kidney transplant

- Treatment goal is MRD negativity with immunophenotypic MRD with EUROFLOW in bone marrow **and** PET CT negativity, but ≥ VGPR according to IMWGs criteria can suffice.
- Preferably patients with standard-risk cytogenetics (MM)
- At least 6-12 months of remission following induction treatment.
- Patients with del(17p), multiple high risk cytogenetic aberrations (including triple-hit disease), or extramedullary disease (EMD) should **only** be considered if they have achieved complete remission (CR) following first- line therapy and have maintained MRD neg remission for at least 12 months.
- No serious ASCT- or induction treatment complications

Myeloma Induction treatment

For the management of multiple myeloma (MM) in patients with severe renal impairment, please refer to the relevant national multiple myeloma guidelines(17-20).
Sweden/Norway/Denmark/Finland

MM Maintenance treatment after myeloma induction in patients with renal failure: Lenalidomide often is often poorly tolerated in patients with advanced renal impairment. Therefore, maintenance therapy with daratumumab and/or bortezomib may be preferred. In recipients of a of living-donor kidney transplant, maintenance therapy is to be discontinued at least 2 weeks before transplantation (at least 4 weeks for IMiD-based therapy). In recipients of a deceased-donor kidney transplant, maintenance therapy should be discontinued at the time of transplantation (3).

4. Smouldering multiple myeloma (SMM)

SMM is defined as an asymptomatic plasma cell disorder characterized by ≥10% clonal bone marrow plasma cells and/or serum monoclonal protein ≥30 g/L, in the absence of myeloma-defining events or amyloidosis. SMM represents a biological intermediate stage between monoclonal gammopathy of undetermined significance (MGUS) and MM, where patients fulfil diagnostic criteria for myeloma but do not yet have end-organ damage or other features requiring initiation of therapy.
Patients with SMM should be evaluated in the context of underlying renal disease and potential MGRS. In cases where kidney impairment is directly related to the plasma cell clone, management may align with treatment principles used for multiple myeloma. Conversely, in patients undergoing kidney transplantation for an unrelated renal disease who are found to have concurrent SMM, a dedicated bone marrow biopsy should be performed to not miss a development into MM, followed by careful individual assessment to determine whether clone-directed therapy is appropriate. As access to and reimbursement for treatment of SMM vary across Nordic countries, management decisions should be individualized and made in close dialogue between the treating physicians and the patient.

5. Monoclonal gammopathy with renal significance (MGRS) and kidney transplantation

MGRS is a relatively new diagnostic concept, aimed at improving the classification of patients with kidney diseases caused by monoclonal proteins. The number of MGRS patients is increasing with age. In a report covering various different MGRS diagnoses from United Kingdom, at diagnosis the median age of patients was 63 years while the median time from MGRS diagnosis to kidney failure (defined as need for chronic kidney replacement therapy [dialysis or transplantation], or an eGFR of less than 15 mL/min per 1·73 m² for 4 weeks or more) were 8.2 years(21). The definition of MGRS is provided below, except for AL amyloidosis, which is discussed in detail in chapter 6.

MGRS definition
Any B-cell or plasma-cell clonal disorder that does not meet current criteria for immediate treatment (in the case of cancer) but produces a nephrotoxic monoclonal immunoglobulin that directly or indirectly results in kidney disease or injury (31). The kidney disease is often caused by the nephrotoxic monoclonal immunoglobulin or indirectly via complement activation.

Renal pathology findings in MGRS

The Renal Pathology Society/International Kidney and Monoclonal Gammopathy Research Group has presented a new report upon pathology findings in MGRS(22). They suggest the following six main groups of findings and specify the pathology diagnostic criteria's as well as clinical findings. More details about the findings are found in the article.
1. ***Light chain cast nephropathy (LCCN),*** former myeloma cast nephropathy, LCNN is the typical finding in MM and is a CRAB criteria, but the terminology is changed since the findings are not exclusively in MM. The main microscopic finding is ≥1 cast monotypic Ig light chain staining.
2. ***Immunoglobulin amyloidosis*** includes both light- and heavy chain amyloidosis or a combination of them, all Congo-positive deposits. The group points out that it is important to rule out AA-amyloidosis. However, this terminology has yet to be accepted by the International Society of Amyloidosis.

3. ***Monoclonal Ig deposition disease*** **(MIDD)** is characterised by a diffuse linear monotypic Ig chain depositions in tubular basement membranes but sometimes also in other basement membranes or in the mesangium of the glomeruli. This diagnose includes former light chain and heavy chain depositions diseases or a combination of both.
4. ***Monoclonal associated glomerulonephritis***. Six different glomerulopathies have been identified by the working group with respective diagnostic criteria. They are:

1. Proliferative glomerulonephritis with monoclonal Ig deposits (PGNMID),
2. Monotypic membranous nephropathy,
3. Monoclonal immunotactoid glomerulopathy (ITG/GOMID),
4. Cryoglobulinemic glomerulonephritis type I and II (CryoGN),
5. Intracapillary monoclonal IgM glomerulopathy (former Waldenström Macroglobulinemia glomerulopathy)
6. C3 glomerulopathy in the setting of monoclonal gammopathy (both C3GN and dense deposit disease.

IgG-subclass staining is mandatory to diagnose PGNMID and membranous nephropathy and is helpful in distinguishing other renal monoclonal gammopathies. Rare glomerulopathies such as heavy chain fibrillary GN and monoclonal gammopathy associated thrombotic microangiopathy have not been included in the report.

5. ***Monoclonal associated crystalline nephropathies*** are crystals found in renal structures but often needs electron microscope to be seen. Several diagnoses are included such as light chain Fanconi syndrome, crystal storing histiocytosis, light chain crystalline podocytopathy and more.
6. ***Non-crystalline light chain tubulopathy*** is a diagnose of some controversy in the working group. Non-crystalline inclusions must be found by both light microscopy and electron microscopy and be accompanied by tubular injury.

MGRS without detectable circulating monoclonal protein or detectable B cell clone

It is important to highlight that monoclonal gammopathy should be considered as a cause of renal disease also in cases of immune complex mediated membranoproliferative glomerulonephritis (IC-MPGN) with or without circulating light chain or M-component or detectable B cell clone as effective treatment is available(23-25). The synonym for the disease in this case is proliferative glomerulonephritis with monoclonal deposits (PGNMID) in which the light microscopy pattern is that of IC-MPGN. The diagnosis requires IgG subtyping and demonstration of heavy chain IgG restriction and light chain restriction or in cases related to IgA or IgM heavy classes only light chain restriction. Light chain-only variant has also been described. It must, however, to be taken into consideration that if the patient has two heavy chains (one monoclonal and one polyclonal), that may mask the restriction of light chains as the other heavy chain is polyclonal (26). With more sensitive techniques this diagnostic pitfall can be surpassed (IF staining for Ig heavy chain/light chain and for Ig VK and VL subgroups)(27). Nasr et al retrospectively examined 37 patients with PGNMID. In this study, a corresponding serum M protein was documented in only 11 patients (30%), which suggests that PGNMID can be caused by lesser amounts of monoclonal immunoglobulin that are well below the level of detection in the systemic circulation through serum immunofixation. In fact, 1 of the 11 patients (9%) had no serum M protein in the initial evaluation; the systemic monoclonal gammopathy became detectable only 3 years later follow-up testing(28).

Monoclonal protein cannot be demonstrated in the kidney biopsy in certain MGRS entities, supporting an indirect role in the pathogenesis of renal diseases. This view is strengthened by the observation that clone-directed therapy results in improved renal outcome. Despite missing evidence of clonality from kidney biopsy, monoclonal gammopathy is evident in haematological investigations in these cases. C3G is characterized by dominant C3 renal deposits by IF and often a membranoproliferative pattern of glomerular injury and has an accepted status as MGRS diagnosis(23). C3G and a subset of thrombotic microangiopathies (TMA) result from the dysregulation of the alternative complement pathway and sometimes these entities result from monoclonal protein behaving as an autoantibody affecting the complement system and causing the renal disease. TMA has currently a provisional status among MGRS diagnoses(29, 30). In all MGRS entities mediated through indirect pathogenic mechanisms, an extensive differential diagnostic work-up ir required. The diagnosis of MGRS should only be established after exclusion of alternative causes and when the renal lesion is considered more likely to be MGRS-related than a coincidental coexistence of MGUS and TMA or C3 glomerulopathy (C3G). Thus , the diagnosis is largely one of exclusion. Advanced and often locally developed analyses of complement activation may be performed in research laboratories to support the diagnostic evaluation; however, such investigations are not mandatory for the diagnosis of MGRS.

When to suspect MGRS

All patients with a monoclonal gammopathy with unexplained kidney function impairment and/or proteinuria and patients presenting with kidney dysfunction and/or proteinuria, where further investigation reveal a monoclonal gammopathy should be suspected of MGRS(16).

Diagnostic work-up in suspected MGRS.

1. **Standard renal evaluation,** including immunological testing, complement factors (C3, C4), and hepatitis serology.
2. **Kidney biopsy (gold standard):** Histopathological evaluation should include light microscopy, immunofluorescence/ immunohistochemistry, and, where available electron microscopy (EM), which is strongly recommended. To establish clonality, light -and heavy-chain subtyping should be performed when available. Paraffin-embedded immunofluorescence (PIF) may be required to unmask immunoglobulins or light-chain deposits. In cases with amyloid-positive staining, amyloid typing by immunogold electron microscopy, immunohistochemistry or mass spectrometry is required. Also, IgG subtyping may be necessary in selected cases.
3. If MGRS is confirmed on kidney biopsy; the patient should be referred for comprehensive haematological evaluation to identify the underlying pathogenic clone. (31).

Haematological work-up in MGRS

1. **Detection of the monoclonal protein, including** serum and urine electrophoresis with immunofixation, as well as measurements of serum free light chains.
2. **Identification of the underlying clonal B-cell or plasma cell population** through bone marrow aspirate and biopsy, with characterization of the underlying clonal population including immunophenotyping and immunohistochemistry, cytogenetics and molecular studies where appropriate. .
3. **Assessment for extrarenal organ involvement and exclusion of overt haematological malignancy**, including MM, Waldenström macroglobulinemia, lymphoma or other lymphoproliferative disorders. Extrarenal manifestations are most commonly observed in AL amyloidosis and may involve the heart, liver, GI-tract, peripheral nervous system, and other organs, but may occur in monoclonal immunoglobulin deposition disease (MIDD) as well. In patients with C3 glomerulopathy (C3G) or dense deposit disease (DDD), ophtalmologicmanifestations such as drusen and retinopathy should be considered.

Treatment strategies and clinical outcomes in patients with MGRS patients in whom no detectable clones can be identified remain heterogenous, highlighting the need for more sensitive methods for clone detection(25). Emerging technologies, such as RNA-based immunoglobulin repertoire sequencing (RACE-RepSeq) are being developed to identify low-level clones and can be performed on peripheral blood, bone marrow samples or potentially kidney biopsy tissue (32). However, the technique is still under development and not in clinical use but may prove to be important in cases where clonality is not evident from routine investigations. Thus far, it has revealed that some PGNMID IgG3 kappa cases are likely not monoclonal but polyclonal in nature. Other techniques like Mass spectrometry in blood sample is under development in the Nordic countries.

Treatment strategies in MGRS

The 2019 IKMG guidelines recommend treatment of MGRS based on the underlying clone driving the pathophysiological process, advising consideration of age, clinical signs, patient comorbidities, renal metabolism, and nephrotoxicity of putative agents(31). Treatment aims at reducing the non-malignant clone to reduce the toxic load on the kidneys and improve renal function.

The treatment strategy for MGRS

Clone-directed treatment is indicated for MGRS and it has been shown to preserve kidney function and prevent recurrent disease after kidney transplantation (16).

1. Establish the characteristics of the clone (plasma cell, B cell, or lymphoplasmacytic) that is producing the nephrotoxic monoclonal immunoglobulin, the type of kidney injury and the ability to reverse or halt further kidney damage (44).
2. Since the nephrotoxicity derives from the monoclonal protein aim at the best possible response expressed as elimination of M-proteins in the blood, BM, and MRD (minimal residual disease).

Plasma cell clone directed treatment in MGRS except AL amyloidosis: There are no randomized controlled trials, however real-world evidence suggests novel myeloma drugs are effective in MGRS. Modern induction treatment include bortezomib and cortisone, cyclophosphamide and CD38-antibodies (12, 44).
B-cell or lymphoplasmacytic cell clone directed therapy: Includes CD20 antibodies (e.g., Rituximab).
No clone detected: Both the above strategies can be tried until response is achieved.

Induction treatment before kidney transplant

In non-IgM related plasma cells dyscrasias therapy should be like myeloma or AL amyloidosis therapy containing CD38 monoclonal antibody (daratumumab), proteasome inhibitor (bortezomib), alkylating agent (cyclophosphamide), and cortisone. The decision for autologous stem cell transplantation should be evaluated considering the patients' therapeutic response, comorbidities and performance status. Most patients reach a very good remission (VGPR or CR) after modern induction treatment, also without stem cell transplantation (ASCT)(31, 33). In these patients stem cell harvest can be performed, but ASCT deferred to second line treatment or omitted. Retrospective evidence on MGRS patients (MIDD, LCPT, PGNMID) have implied that ASCT may offer better haematological as well as renal response, but information from prospective trials is lacking(34).

Proteasome inhibitor has the highest efficacy in M-protein-associated renal disorders since it rapidly reduces tumor load and toxic M-proteins. In addition, bortezomib clearance is independent of renal function(35, 36). MIDD should be treated as AL-amyloidosis and the renal transplantation should be evaluated in the same way(37) .

We recommend 6 cycles of Daratumumab-VCD28 since reports show better outcome than VCD therapy. (ANDROMEDA-study) See national treatment guidelines.

IgM-related disease should be treated in a similar fashion as CLL/B-cells lymphoma with a regimen containing CD20 monoclonal antibody (rituximab)(35, 36). For further information on diagnosis and induction treatment, we refer to the current national guidelines(38-41):
Sweden / Denmark / Norway / Finland

6. AL amyloidosis and kidney transplantation

AL-Amyloidosis with renal involvement

70-80% of AL amyloidosis patients have renal involvement, 19-24 % will require kidney replacement therapy during the course of their disease(42). The main manifestation of renal involvement in AL amyloidosis is excessive proteinuria (≥500 mg/24 hour), predominantly albuminuria, often leading to overt nephrotic syndrome.
The diagnostic gold standard is kidney biopsy with subsequently light microscopy on Congo-red (or thioflavin T) stained biopsy. Electron microscopy can reveal fibrils, and immunostaining with known amyloidogenic proteins makes the diagnosis of the amyloid subtype along with mass spectrometry(43). An alternative diagnostic approach is Congo-red staining of bone marrow biopsy and/or fat tissue aspiration. The combination of these will be able to demonstrate amyloid deposits in approximately 85%. Both procedures are readily available in haematology departments and have low complication rates.
Approximately 2/3 of the patients have involvement of more than one organ. The most frequently involved organs are the heart, kidneys, nerves (both peripheral and autonomic neuropathy) and gastrointestinal tract. For further decisions on whether to proceed to kidney transplant, careful assessment on involvement of other organs is pivotal.
In recent years new treatment protocols with Cyclophosphamide, Bortezomib and dexamethasone (VCD) + daratumumab (D-VCD) have compared favourably to all previous regimes (33). The Andromeda study demonstrated a significantly higher response rate, with 70% achieving ≥ VGPR in the Daratumumab group compared to 50,3% in the VCD group. This translated into a significant difference in renal response rates at 6 months being 53% versus 24%(44). Thus Daratumumab-based regimens, including Dara-VCD represent a new standard of care. However, this combination is not reimbursed in all the Nordic countries. An increasing number of patients achieve VGPR or CR with modern induction treatment without ASCT, therefore ASCT may be deferred or omitted in patients with very good remission/MRD negativity after induction therapy.

For further information on diagnosis and induction treatment, we refer to the current national guidelines(45-48):
Sweden / Denmark / Norway / Finland

Indication for kidney transplantation in AL- Amyloidosis

Despite achieving profound haematological (CR or VGPR) response, including long lasting responses, amyloid fibrils in the kidneys can persist and lead to continuously decline in renal

function. These patients have a favourable survival outcome compared to MM, but the risk of ongoing decline in renal function can ultimately result in end stage kidney disease (ESKD) and the need of renal replacement therapy or kidney transplant.
Case series from the past decade indicate that kidney transplantation outcomes in AL amyloidosis are comparable to those for transplantation due to diabetes mellitus, in terms of both patient and graft survival(10). Patients with concurrent cardiac involvement face a bleaker prognosis, and significant cardiac involvement should be seen as a contraindication. Favourable indicators for transplantation include a robust response to initial treatment, ideally achieving haematological response at least. CR and VGPR translate into significantly better PFS and OS(49). Moreover, kidney transplantation may be considered in cases of stable disease not requiring treatment, despite ongoing deterioration in kidney function necessitating dialysis(42). Furthermore, kidney transplantation should also be a strategy to consider in later lines of treatment, as 2nd or later lines of therapy in AL amyloidosis can similarly translate into deep and durable responses.

When to refer to kidney transplantation:

Kidney transplantation may be considered in patients with AL amyloidosis after completion of clone-directed therapy and achievement of a satisfactory haematological response, typically at least VGPR. There is no requirement for a predefined duration of remission prior to referral(42). -The timing of referral should balance the benefit of deeper haematological response against the risk of ongoing renal deterioration. Careful reassessment of cardiac involvement and other extra-renal organ manifestations is essential, as these significantly influence transplant eligibility and outcomes. Kidney transplantation may also be considered in selected patients with stable disease not requiring ongoing therapy but with progressive renal failure(2, 3). Detailed response criteria, eligibility assessment, and pre-transplantation work-up are described in Chapter 7-8.

7. Kidney transplantation, general considerations

General criteria for referral to kidney transplantation

1. Patient and haematological disease criteria

- All patients with MM or MGRS, including AL amyloidosis, who are candidates for clone-directed therapy may be considered for kidney transplantation.
- The underlying haematological disease should be considered controlled or controllable, with the patient in the best achievable state of remission.
- No serious treatment related complications or significant comorbidity precluding transplantation.
- Estimated life expectancy >5 years.

MM
- At least 6–12 months in haematological remission after induction treatment
- This also applies to patients on maintenance therapy
- Earlier referral may be considered in patients with standard cytogenetic risk and good response.

MGRS/AL amyloidosis
-Earlier referral may be once a good haematological response has been achieved.

2. Kidney transplantation criteria

- The patient should be suitable for kidney transplantation according to respective transplant center's criteria.
- Patients should have an acceptable immunological risk, including assessment of pre-transplant donor-specific antibodies (DSA), as determined by the transplant centre.

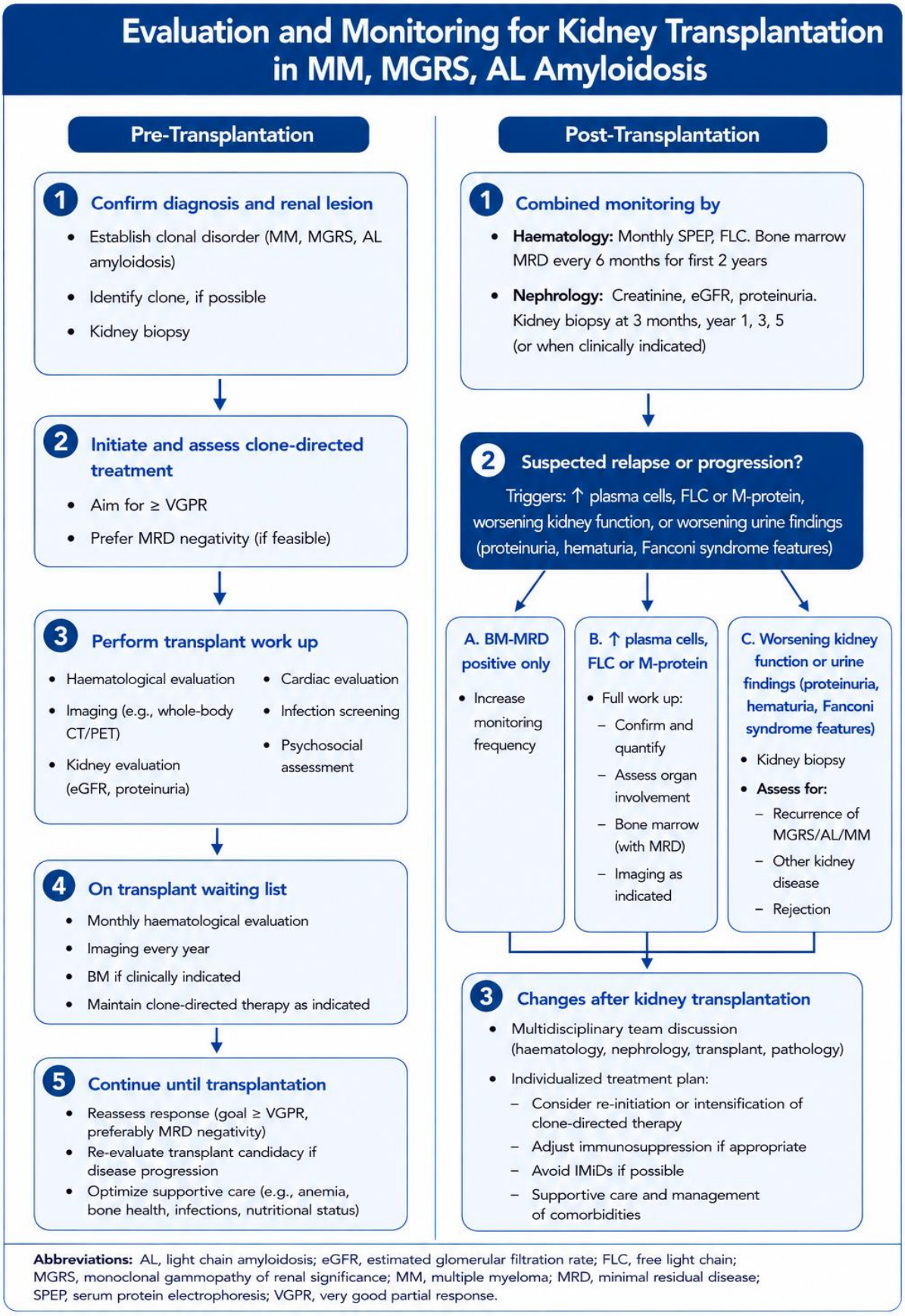
Contraindication, absolute

- Severe general comorbidity
- Other malignancies, except treated and cured or low-grade cancers (individual assessment required
- Cardiac AL-amyloidosis with NYHA >2 and/or EF<35% despite cardiac optimization and haematological induction treatment

Contraindication, relative

- Significant extra renal organ dysfunction.
- Highly HLA-immunized individuals

8. Diagnostic work-up before kidney transplantation and post-transplant monitoring



Decision pathway before referral to kidney transplantation

1. Confirm diagnosis
    i. Identify type of clonal disorder
    ii. Define renal lesion (kidney biopsy where relevant).
2. Initiate and assess clone-directed therapy
    i. Treatment according to national guidelines
    ii. Aim for deepest possible haematological response
3. Assess haematological response, if
    i. Inadequate → continue haematological treatment
    ii. Adequate → proceed to transplant work-up

Response requirements before referral

1. General
    i. A response of ≥ VGPR is recommended.
    ii. Depth of haematological response before kidney transplantation is strongly associated with positive outcome (36, 37).
    iii. MRD negativity is preferred, when available.
2. MM
    i. Sustained remission
    ii. MRD negativity strengthens decision-making
3. MGRS
    i. ≥ VGPR (≥90% reduction in circulating monoclonal protein or involved FLC)
    ii. In non-measurable disease: individualized assessment required
    iii. Consider:
        i. bone marrow findings
        ii. presence or absence of detectable clone
        iii. stability of renal parameters
4. AL-Amyloidosis
    i. Target: ΔFLC <40 mg/L (VGPR)
    ii. No requirement for prolonged remission
    iii. Reassess cardiac and other organ involvement
5. Non-secretory disease

i. Response assessment based on bone marrow evaluation, imaging and standard kidney transplant follow-up

Work-up after clone directed treatment before referral to kidney transplantation (maximum 6 months old)

1. Haematological evaluation
    i. SPEP + immunofixation when in CR
    ii. Serum free light chains
    iii. UPEP when relevant
    iv. Albumin / proteinuria assessment
    v. Bone marrow when in CR:
        i. response assessment, preferably bone marrow biopsy
        ii. MRD (EuroFlow), when available
2. Kidney evaluation at nephrologists´ discretion
3. Cardiac evaluation
4. Infection screening
5. Psychosocial assessment

Monitoring before transplantation (waiting list)
Patients should remain under haematological follow-up while awaiting transplantation to confirm stable response and detect possible progression. Recommended monitoring:

i. M-protein and FLC: monthly
ii. Imaging (MM): at least yearly
iii. Bone marrow: when clinically indicated
iv. Maintain clone-directed therapy as indicated
v. Reassess haematological response
vi. Re-evaluate transplant candidacy if disease progression
vii. Optimize supportive care such as anemia, bone health, infection prophylaxis, nutritional status and assessment of social network and caregiver support.

Monitoring and evaluation after kidney transplantation
During the first 5 years of follow-up, all patients will be managed according to the standardized transplantation treatment protocol after kidney transplantation provided from the 2026 Nordic consensus treatment protocol for treatment (see above) of patients with monoclonal gammopathies and kidney transplantation **and should remain in controls both at nephrology and haematology departments.**

Haematological monitoring

i. SPEP + FLC: Monthly 1st year. Thereafter regularly per local guidelines at each centre but no less than every 3 months

ii. Immunofixation: When M-protein becomes undetectable to confirm CR

iii. Bone marrow: **Only in CR**: Every 6 months first 2 years then annually with EURO-FLOW MRD

Renal monitoring
Standard kidney transplant follow-up at transplant centre's discretion.
Protocol kidney allograft biopsies are recommended after KTx at 3, 12, 36 and 72 months.
Unexplained deterioration in eGFR/Creatinine or increasing proteinuria prompt for transplant biopsy. See follow-up flow chart above.

Management suspected relapse after kidney transplantation

1) **MRD positive in BM sample, no other changes**
    a. Increase monitoring
    b. Repeat haematological evaluation
    c. No immediate treatment however multidisciplinary team discussion regarding eventual change in maintenance (when applicable)
2) **Significant rise in FLC, M-protein or plasma cells, stable kidney function**
    a. Repeat laboratory testing monthly
    b. Perform full haematological work-up (bone marrow and imaging included)
    c. Multidisciplinary team discussion
    d. Consider kidney biopsy
    e. Individualized treatment decision
3) **Worsening kidney function (rise in creatinine) or urine findings (proteinuria, haematuria, Fanconi syndrome features)**
    a. Perform transplant kidney biopsy
        i. If recurrence of MGRS, AL Amyloidosis or MM is confirmed:
            1. Classify as disease relapse and initiate clone-directed treatment according to disease type
            2. Multidisciplinary management involving haematology and nephrology is recommended
    b. If other kidney disease is identified:
        i. Management and further diagnostic work-up should be performed in collaboration with nephrology at their discretion
    c. If rejection is identified
        i. Management should be guided by the treating nephrologist/transplant nephrology team at their discretion
4) **Positive biopsy without clinical deterioration**

a. Individualized assessment

b. Consider closer monitoring and multidisciplinary team discussion

c. Treatment decisions should be individualized based on disease type, biopsy findings and risk of progression

Suggested post-transplant monitoring strategy

The purpose of the monitoring strategy is to enable early detection of haematological relapse, recurrent renal disease, transplant-related complications and infectious events after kidney transplantation in patients with plasma cell dyscrasias. Monitoring intensity should be highest during the first year after transplantation, when the risk of recurrence, rejection and infectious complications is greatest.

The protocol below represents a suggested minimum monitoring framework. Additional investigations should always be performed when clinically indicated and local transplant centre practices may vary. Haematological monitoring should preferably be performed in collaboration between nephrology, transplant medicine and haematology.

Investigations marked with parenthesis are considered optional, centre dependent or primarily intended for specialized or research-oriented follow-up protocols.

Investigation	Before KTx (waiting list)	KTx Day -1 - 0	Monthly 1st year	Every 3 months 1st year	Yearly	By indication
Haematological monitoring						
Routine haematology lab [1]		x	x		x	x
Clonal disease monitoring [2]		x	x		x	x
Extended clonal assessment [3]		(x)		(x)	(x)	(x)
Blood mass spectrometry	(x)*	(x)	(x)		(x)	x
BM ± MRD (EuroFlow) in CR [4]	(x)*				(x)	x
Renal monitoring						
Routine renal lab [5]		x	x		x	x
mGFR (iohexol clearance				(x)	(x)	(x)
Protocol transplant biopsy [6]				x*	x*	x
Transplant and infectious monitoring						
Viral PCR monitoring [7]		x		x	x	x
DSA/cPRA assessment [8]		x	(x)		(x)	x

Footnotes

* Blood mass spectrometry and MRD in BM (EuroFlow)requires a pre-treatment/pre-transplant serum sample with a detectable intact monoclonal protein to establish the patient-specific mass spectrometric signature. Without such a baseline sample, longitudinal monitoring may not be feasible.

[1]**Routine haematology lab:** Haemoglobin, complete and differential white blood counts, platelet count.

[2]**Clonal disease monitoring**: Serum protein electrophoresis (SPEP), serum free light chains (FLC), M-protein assessment and, when relevant, urine electrophoresis.

[3]**Extended clonal assessment**: Serum and urine immunofixation, uninvolved immunoglobulins and mass spectrometry according to local center's practice

[4] **BM ± MRD (EuroFlow) in CR**: Suggested intervals for bone marrow (BM) and MRD assessment in complete remission (CR) are every 6 months during the first 2 years after transplantation and annually thereafter.

[5]**Routine renal lab**: Creatinine, eGFR (if not on dialysis), Na+, K+, liver transaminases, HbA1c, C-reactive protein, UACR (urine albumin-creatinine-ratio), serum/plasma albumin protein UPCR

[6]**Protocol transplant biopsy**: Suggested protocol biopsy time points include approximately 3 months, 12 months, 3 years and 5 years after transplantation depending on local center's practice. Additional biopsies are recommended in graft dysfunction, increasing proteinuria or suspected recurrent disease. Analyze biopsy with light microscopy, immunohistochemistry and electron microscopy. Always at time of protocol biopsy, clonal disease monitoring should be done.

[7]**Viral PCR monitoring**: CMV, EBV and BK virus minimum every 3 months the 1st year, thereafter by indication or according to local transplant protocols and intensity of immunosuppression.

[8]**DSA / cPRA assessment:** Calculated panel reactive antibody (cPRA) and donor specific antibodies (DSA) are assessed at KTx day 0, thereafter at centers' discretion.

9. Maintenance treatment after kidney transplantation

There are no validated studies. Proven experience recommends considering Daratumumab 1 year or Bortezomib every two weeks or carfilzomib if neuropathy is a concern. We emphasize caution when considering IMiDs (thalidomide, lenalidomide, pomalidomide) due to the risk of transplant rejection(33). Monitoring strategies are described in Chapter 8.

10. Treatment in recurrence of the disease

Definition of relapse/recurrence of disease

- Bone marrow: MRD positivity or reoccurrence of malignant plasma cells in immunophenotyping or cytology.
- Blood: FLC positivity, reoccurrence of M-protein in SPEP in the blood
- Kidney biopsy: Histological recurrence in kidney (both in protocol or indication biopsies) with Light microscopy, immunofluorescence, and if possible, EM
- Renal progressive disease

Recurrence in MGRS is a frequent problem and in PGNMID and C3G-Mig and it may come fast. Currently there are no validated progression criteria. Palladini 2018 recommends progression criteria similar to AL Amyloidosis response criteria (International society of Amyloidosis – ISA), meaning ΔFLC >20mg/L, ΔFLC at least 20% from dg, ΔFLC elevation >50% from best response.
In earlier guidelines (2014), therapy was to be initiated only when symptomatic disease (35). Current data also suggest that progression of amyloidosis can be managed without harming the transplanted organ (50) and the patient can be treated with the first signs of recurrence. Relapse in MM should be treated with the patients' survival and symptom reduction as priority and kidney transplant as 2nd priority.
Repeat of first line therapy can be considered if prior good and long-term response. In other cases, the patient should be discussed among colleagues with special interest in MGRS. According to national guidelines informed decision must be made prior to immunomodulatory drugs due to the risk of rejection(51).
Bortezomib - Proteasome inhibitors are well tolerated in kidney transplant (KTx) recipients. They have also been suggested as a potential therapeutic option in antibody-mediated rejection (ABMR), given their ability to lower donor-specific anti–human leukocyte antigen (HLA) antibody levels(52-54).
Lenalidomide - Use of lenalidomide has been associated with an increased risk of renal allograft rejection, likely mediated through enhanced interleukin-2 signaling and disruption of immune tolerance mechanisms. In addition, lenalidomide therapy carries a well-documented risk of thromboembolic events, necessitating prophylactic anticoagulant treatment. An elevated incidence of second primary malignancies has been reported when lenalidomide is administered in combination with oral melphalan, whereas this risk has not been observed when combined with dexamethasone alone(55, 56).
Carfilzomib - It remains unclear whether the risk of thrombotic microangiopathy (TMA) is increased when carfilzomib is used concomitantly with calcineurin inhibitor–based maintenance immunosuppression. In reported cases, TMA may be complement-mediated, and there is emerging evidence suggesting potential benefit from complement inhibition in this setting(57).
Daratumumab - Histopathological improvement of chronic ABMR has been described in a patient with smoldering multiple myeloma (SMM) treated with daratumumab. False-positive crossmatch results have also been reported, attributed to interference from daratumumab binding to CD38 on donor lymphocytes rather than true donor-specific antibodies; this effect was mitigated by treatment with dithiothreitol(58).

11. Planned evaluations of the protocol

All Nordic transplant centres are welcome to participate in the follow up of these guidelines. A CRF will be available in REDCAP given from the guideline steering group. Data will be annotated in an electronic database at the Institute of Medicine, Sahlgrenska Academy and in the Transplantation registers in respective countries**.**

In a retrospective chart review we later plan to study the following questions:

1. To investigate a more standardized monitoring of MM and MGRS after kidney transplant including blood tests of plasma proteins and find prognostic factors for KTx success rate.
2. To evaluate the role of protocol BM biopsy including MRD with immunophenotyping (EUROFLOW) at different time points after kidney transplant to detect early (subclinical) relapse (will be performed at certain centres in the Nordic countries).
3. To evaluate the role of a protocol kidney biopsy in different time points after kidney transplant (3 and 12 months) at certain centres in the Nordic countries.
4. To find the rate of recurrence of MM and MGRS after kidney transplant expressed as positivity in MRD in the bone marrow and plasma proteins as well as in kidney biopsy.
5. To describe the given treatment on the disease recurrence even expressed as MRD positivity in the bone marrow, positive plasma proteins and kidney biopsy
6. To investigate the prevalence of different toxicities such as opportunistic infections (CMV, BK, PC-pneumonia) following haematological induction treatment and kidney transplant
7. To study the prevalence of BK neuropathy, CMV and EBV infections post-transplant
8. To assess the rate of rejection after kidney transplant in MM and MGRS
9. To investigate the varied reasons for loss of transplanted organ
10. To assess the kidney function, kidney survival and patient survival 1, -3 and -5 years after kidney transplantation
11. To establish a national or Nordic register of all kidneys transplanted MM and MGRS patients

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