Formulário de Recurso

ANULAÇÃO DE QUESTÃO

RECURSOS CONTRA O EDITAL DE RESULTADO DAS PROVAS OBJETIVAS

TRIBUNAL DE JUSTIÇA
ESTADO DO RIO GRANDE DO SUL

CONCURSO PÚBLICO PARA PROVIMENTO DOS CARGOS DE MÉDICO PSIQUIATRA PJ-J, PSICÓLOGO JUDICIÁRIO PJ-J, PEDAGOGO JUDICIÁRIO PJ-I; MÉDICO JUDICIÁRIO CLASSE "R", ASSESSOR JUDICIÁRIO CLASSE "P" (TJM) E CONTADOR CLASSE "R" (TJM EDITAL 03/2016)

FORMULÁRIO ESPECÍFICO DE RECURSO CONTRA O RESULTADO PRELIMINAR DAS PROVAS OBJETIVAS, APLICADAS NO DIA 22/05/2016, CONFORME EDITAL DE ABERTURA N.º 03/2016 – DRH-SELAP-RECSEL (DIRIGIDOS À COMISSÃO DO CONCURSO)

RAZÕES DE RECURSO

MÉDICO JUDICIÁRIO CLASSE "R" – ESPECIALIDADE: CLÍNICA MÉDICA OU MEDICINA INTERNA

Nº DA QUESTÃO: 52

O gabarito traz como correta a alternativa C (I - citomegalovírus; II - sífilis; III - botulismo). Contudo o botulismo, doença provocada pelo Clostridium botulinum, não é relacionado à síndrome nefrótica. Além disso, a alternativa "IV - Malária" é verdadeira, pois está relacionada à síndrome nefrótica.
Como não há alternativa com as opções "Apenas I, II e IV", esta questão deve ser anulada.

Em anexo a referência extraída do guideline KDIGO (Kidney Disease Improving Global Outcomes), da Sociedade Internacional de Nefrologia, capitulo 9 – Infection-related glomerulonephritis –, com a tabela (página 201, "table 21: infectons associated with glomerulonephritis") que cita infecções sistêmicas que estão relacionadas à glomerulopatias, que são causas de síndrome nefrótica; além de outra página que cita a malária como causa de síndrome nefrótica.

Chapter 9: Infection-related glomerulonephritis

Kidney International Supplements (2012) **2,** 200–208; doi:10.1038/kisup.2012.22

9.1: For the following infection-related GN, we suggest appropriate treatment of the infectious disease and standard approaches to management of the kidney manifestations: (*2D*)
- **poststreptococcal GN;**
- **infective endocarditis-related GN;**
- **shunt nephritis.**

INTRODUCTION

This chapter provides recommendations for the treatment of infection-associated GN, which may occur in association with bacterial, viral, fungal, protozoal, and helminthic infection (Table 21). The cost implications for global application of this guideline are addressed in Chapter 2.

BACTERIAL INFECTION–RELATED GN

BACKGROUND AND RATIONALE

The prototype for bacterial infection-related GN (also called postinfectious GN) is poststreptococcal GN, which most often occurs in children following a pharyngeal or cutaneous infection (impetigo) caused by a particular nephritogenic strain of *Streptococci*, and usually has a favorable outcome.

However, in the last decades the spectrum of postinfectious GN has changed. The incidence of poststreptococcal GN, particularly in its epidemic form, has progressively declined in industrialized countries. Recent series reported that streptococcal infections accounted for only 28–47% of acute GN, *Staphylococcus aureus* or *Staphylococcus epidermidis* being isolated in 12–24% of cases and Gram-negative bacteria in up to 22% of cases.[330–332] Bacterial endocarditis and shunt infections are also frequently associated with postinfectious GN. Moreover, the atypical postinfectious GN tends to affect mainly adults who are immunocompromised, e.g., in association with alcoholism, diabetes, and drug addiction. While spontaneous recovery within a few weeks is still the rule in children affected by the typical poststreptococcal GN, the prognosis in immunocompromised adults with postinfectious GN is significantly worse, with less than 50% in complete remission after a long follow-up.[333]

POSTSTREPTOCOCCAL GN

BACKGROUND AND RATIONALE

The diagnosis of poststreptococcal GN requires the demonstration of antecedent streptococcal infection in a patient who presents with acute GN. Nephritis may follow 7–15 days after streptococcal tonsillitis and 4–6 weeks after impetigo.[334]

The nature of the nephritogenic streptococcal antigen is still controversial.[334–336] Kidney biopsy is not indicated unless there are characteristics that make the diagnosis doubtful, or to assess prognosis and/or for potential therapeutic reasons. The kidney histology shows acute endocapillary GN with mesangial and capillary granular immune deposition.

The clinical manifestations of acute nephritic syndrome usually last less than 2 weeks. Less than 4% of children with poststreptococcal GN have massive proteinuria, and occasionally a patient develops crescentic GN with rapidly progressive kidney dysfunction. Serum C3 values usually return to normal by 8–10 weeks after recognition of the infection. Persistent hypocomplementemia beyond 3 months may be an indication for a renal biopsy, if one has not already been performed. A lesion of MPGN is commonly found in persistently hypocomplementemic GN.

The short-term prognosis of the acute phase of poststreptococcal GN is excellent in children; however, in elderly patients, mortality in some series is as high as 20%. Although the long-term prognosis of poststreptococcal GN is debated, the incidence of ESRD in studies with 15 years of follow-up is less than 1%, with the exception being that long-term prognosis is poor in elderly patients who develop persistent proteinuria.[333,334]

Well-documented streptococcal infection should be treated with penicillin, or erythromycin if the patient is allergic to penicillin, to resolve streptococcal infection and prevent the spread of the nephritogenic streptococcus among relatives or contacts. However, antibiotics are of little help for reversing GN, as the glomerular lesions induced by immune complexes are already established.

The management of acute nephritic syndrome, mainly in adults, requires hospital admission if features of severe hypertension or congestive heart failure are present. Hypertension and edema usually subside after diuresis is established. Adult patients persisting with urinary abnormalities beyond 6 months, especially if proteinuria >1 g/d, should receive ACE-I or ARBs, as in other proteinuric glomerular diseases (see Chapter 2). The long-term prognosis is worse in patients, mainly adults, who have persistent proteinuria after 6 months.[337]

Pulses of i.v. methylprednisolone can be considered in patients with extensive glomerular crescents and rapidly progressive GN, based on extrapolation from other rapidly progressive and crescentic GNs, although there is no evidence from RCTs.

RESEARCH RECOMMENDATIONS

- An RCT is needed to evaluate the treatment of crescentic poststreptococcal GN with corticosteroids.

Table 21 | Infections associated with glomerulonephritis

Bacterial
Mycobacterium leprae, M. tuberculosis
Treponema pallidum
Salmonella typhi, S. paratyphi, S. typhimurium
Streptococcus pneumoniae, S. virdans, S. pyogenes
Staphyloccoccus aureus, S. epidermidis, S. albus
Leptospira species[a]
Yersinia enterocolitica[a]
Neisseria meningitidis, Neisseria gonorrhoeae[a]
Corynebacterium diphtheriae[a]
Coxiella burnettii[a]
Brucella abortus[a]
Listeria monocytogenes[a]

Fungal
Histoplasma capsulatum[a]
Candida[a]
Coccidiodes immitis[a]

Protozoal
Plasmodium malariae, P. falciparum
Leishmania donovani
Toxoplasma gondii
Trypanosoma cruzi, T. bruci
Toxocara canis[a]
Strongyloides stercoralis[a]

Viral
Hepatitis B and C
Human immunodeficiency virus
Epstein-Barr virus
Coxsackie B
ECHO virus
Cytomegalovirus
Varicella zoster
Mumps
Rubella
Influenza

Helminthic
Schistosoma mansoni, S. japonicum, S. haematobium
Wuchereria bancrofti
Brugia malayi
Loa loa
Onchocerca volvulus
Trichinella spiralis[a]

ECHO, enteric cytopathic human orphan; GN, glomerulonephritis.
[a]Only case reports documented.

- Research is needed to determine the nature of the streptococcal antigen, as a basis for developing immunoprophylactic therapy.

GN ASSOCIATED WITH INFECTIVE ENDOCARDITIS

BACKGROUND AND RATIONALE

The natural history of GN associated with infective endocarditis has been significantly altered with the changing epidemiology of the disorder, and with the use of antibiotics.[337–340]

In USA, infective endocarditis is diagnosed in approximately 40 cases per million every year, and the disease is increasingly frequent in elderly individuals and in patients with no underlying heart disease. i.v. drug usage, prosthetic heart valves, and structural heart disease are risk factors. *Staphylococcus aureus* has replaced *Streptococcus viridans* as the leading cause of infective endocarditis. The incidence of GN associated with *Staphylococcus aureus* endocarditis ranges from 22% to 78%, the highest risk being among i.v. drug users. Focal and segmental proliferative GN, often with focal crescents, is the most typical finding. Some patients may exhibit a more diffuse proliferative endocapillary lesion with or without crescents.[337–340]

The immediate prognosis of the GN is good, and is related to the prompt eradication of the infection, using appropriate antibiotics for 4–6 weeks.

RESEARCH RECOMMENDATION

- Multicenter studies are needed to determine the incidence, prevalence, and long-term prognosis of infective endocarditis–related GN.

SHUNT NEPHRITIS

BACKGROUND AND RATIONALE

Shunt nephritis is an immune complex–mediated GN that develops as a complication of chronic infection on ventriculoatrial or ventriculojugular shunts inserted for the treatment of hydrocephalus.[341]

The diagnosis is based on clinical evidence of kidney disease (most commonly, microscopic hematuria and proteinuria, frequently in the nephrotic range, occasionally elevated SCr and hypertension) with prolonged fever or signs of chronic infection, in a patient with a ventriculovascular shunt implanted for treatment of hydrocephalus. The histologic findings are typically type 1 MPGN, with granular deposits of IgG, IgM, and C3, and electron-dense mesangial and subendothelial deposits.

The renal outcome of shunt nephritis is good if there is early diagnosis and treatment of the infection. Ventriculovascular shunts may become infected in about 30% of cases. GN may develop in 0.7–2% of the infected ventriculovascular shunts in an interval of time ranging from 2 months to many years after insertion. The infecting organisms are usually *Staphylococcus epidermidis* or *Staphylococcus aureus*. In contrast to ventriculovascular shunts, ventriculoperitoneal shunts are rarely complicated with GN.

A late diagnosis, resulting in delays in initiating antibiotic therapy and in removing the shunt, results in a worse renal prognosis.

RESEARCH RECOMMENDATION

- Multicenter observational studies are needed to determine the incidence, prevalence, and long-term prognosis of shunt nephritis.

RESEARCH RECOMMENDATION

- Studies are required to evaluate the precise contribution of *Salmonella* infection to schistosomal nephropathy, and the value of treating these two infections separately or together on the outcome.

FILARIAL NEPHROPATHY

BACKGROUND AND RATIONALE

Filarial worms are nematodes that are transmitted to humans through arthropod bites, and dwell in the subcutaneous tissues and lymphatics. Clinical manifestations depend upon the location of microfilariae and adult worms in the tissues. Of the eight filarial species that infect humans, glomerular disease has been reported in association with *Loa loa*, *Onchocerca volvulus*, *Wuchereria bancrofti*, and *Brugia malayi* infections in Africa and some Asian countries.[452–456]

Glomerular involvement is seen in a small number of cases. Light microscopy reveals a gamut of lesions, including diffuse GN and MPGN, membranoproliferative GN, minimal-change and chronic sclerosing GN, and the collapsing variant of FSGS.[457] Microfilariae may be found in the arterioles, glomerular and peritubular capillary lumina, tubules, and interstitium.[457] Immunofluorescence and electron microscopy show immune deposits along with worm antigens and structural components.[456,458]

Urinary abnormalities have been reported in 11–25% and nephrotic syndrome is seen in 3–5% of patients with loiasis and onchocerciasis, especially those with polyarthritis and chorioretinitis.[456,459] Proteinuria and/or hematuria was detected in over 50% of cases with lymphatic filariasis; 25% showed glomerular proteinuria.[460,461] A good response (diminution of proteinuria) is observed following antifilarial therapy in patients with non-nephrotic proteinuria and/or hematuria. The proteinuria can increase and kidney functions worsen following initiation of diethylcarbamazepine or ivermectin,[461,462] probably because of an exacerbation of the immune process secondary to antigen release into circulation after death of the parasite.[463]

The response is inconsistent in those with nephrotic syndrome, and deterioration of kidney function may continue, despite clearance of microfilariae with treatment. Therapeutic apheresis has been utilized to reduce the microfilarial load before starting diethylcarbamazepine to prevent antigen release.[464]

The incidence, prevalence, and natural history of glomerular involvement in various forms of filariasis are poorly documented. This condition is usually found in areas with poor vector control and inadequate health-care facilities. Similarly, the treatment strategies have not been evaluated.

RESEARCH RECOMMENDATION

- Epidemiological studies of kidney involvement in regions endemic for these conditions are required. The effect of population-based treatment with filaricidal agents on the course of kidney disease should be studied.

MALARIAL NEPHROPATHY

BACKGROUND AND RATIONALE

Infection with *Plasmodium falciparum* usually results in AKI or proliferative GN. Chronic infection with the protozoal malarial parasites *Plasmodium malariae* (and, to a lesser extent, *Plasmodium vivax* or *ovale*) has been associated with a variety of kidney lesions, including MN and membranoproliferative GN.[465] In the past, this has been known as "quartan malarial nephropathy".[465,466] Nephrotic syndrome, sometimes with impaired kidney function, is a common clinical manifestation; it is principally encountered in young children. The glomerular lesions are believed to be caused by deposition of immune complexes containing antigens of the parasite, but autoimmunity may participate as well. The clinical and morphological manifestations vary from country to country.[467] Nowadays, the lesion is much less common, and most children in the tropics with nephrotic syndrome have either MCD or FSGS, rather than malarial nephropathy.[467,468] HBV and HIV infection and streptococcal-related diseases are also now more common causes of nephrotic syndrome than malarial nephropathy in Africa.[467–469]

There are limited observational studies and no RCTs for an evidence-based treatment strategy for malarial nephropathy. Patients with GN and concomitant infection with *Plasmodium* species (typically *Plasmodium malariae*) should be treated with an appropriate antimalarial agent (such as chloroquine or hydroxychloroquine) for suffcient duration to eradicate the organism from blood and hepatosplenic sites. Observational studies have suggested improvement in clinical manifestations in some—but not all—patients, following successful eradication of the parasitic infection. There does not appear to be any role for steroids or immunosuppressant therapy in malarial nephropathy,[465,466] although controlled trials are lacking. Dosage reductions of chloroquine or hydroxychloroquine may be needed in patients with impaired kidney function.

RESEARCH RECOMMENDATIONS

- Studies of the incidence and prevalence of malarial nephropathy, and its response to antimalarial therapy are needed, especially in endemic areas of West Africa.
- RCTs are needed to investigate the role of corticosteroids and immunosuppressive agents when malarial nephropathy progresses, despite eradication of the malarial parasite.

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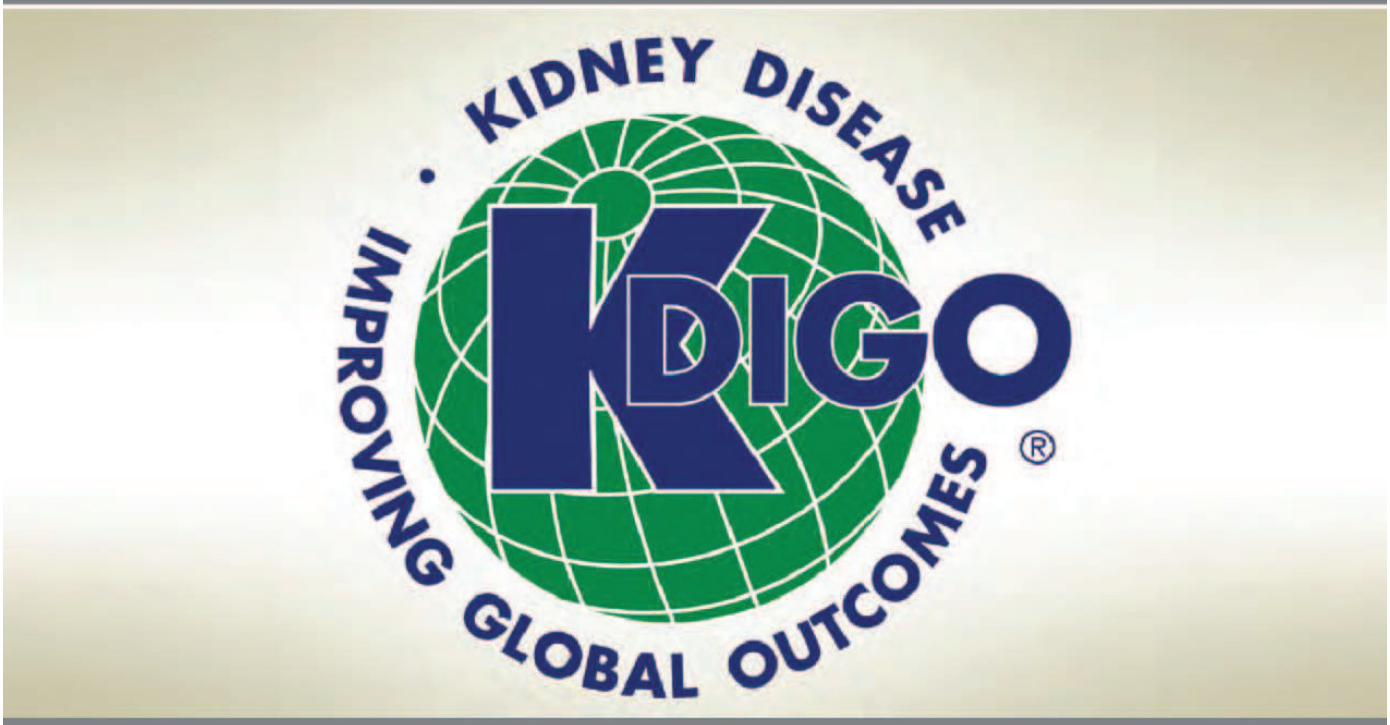

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RESPOSTA A RECURSO

MÉDICO JUDICIÁRIO CLASSE "R" – ESPECIALIDADE: CLÍNICA MÉDICA OU MEDICINA INTERNA

Nº DA QUESTÃO: 52

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RESULTADO: DEFERIDO

JUSTIFICATIVA: Favorável a anulação. Houve troca das assertivas quando adaptada a questão.