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Review Article **Nephrology Hemodialysis**

Renal Injury During Viral Infections

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Abstract

Renal pathologies following viral infections have become an emerging public health problem in both developed and developing countries. Diagnostic criteria are complex. In most cases, they involve correlation between clinical, biological and histological data, with occasional recourse to molecular biology techniques. Several mechanisms are involved in the pathogenesis of virus-related nephropathy, including virus tropism in the kidney, formation of immune complexes in situ or in the bloodstream, direct cytopathogenic effects, and multiple organ failure. The hepatitis C virus is responsible for three main types of kidney disease: membranoproliferative glomerulnephritis, cryoglobulinemia and membranous nephropathy. Hepatitis B virus is associated with membranous nephropathy, membranoproliferative glomerulnethritis, and IgA nephropathy. HIV (human immunodeficiency virus) infection is associated with several glomerular and tubular kidney damage. HIVAN (HIV-associated nephropathy), a specific entity mainly affecting Africans and African-Americans, presents as a rapidly progressive glomerulonephritis rapidly progressing to the terminal stage. Infections secondary to adenovirus, cytomegalovirus, epstein-barr virus, poliomavirus and coronavirus are often responsible for acute or chronic tubulointerstitial nephritis. Treatment is mainly symptomatic, based on nephroprotection measures, rarely combined with antiviral therapy. Prophylaxis with vacci-nation, when available, remains the best means of preventing viral nephropathy.

Keywords: Emerging public health, Renal pathologies, molecular biology techniques, organ failure, Treatment.

INTRODUCTION

Viral infections can cause a variety of kidney diseases. However, it is often not easy to establish a clear pathogenic link. Renal pathology in this sense is well defined when the viruses are renotropic and replicate within the renal parenchyma, whereas for other non-renotropic viruses the causal role is not clearly established. Diagnostic criteria are complex. In the majority of cases, they involve correlation between clinical, biological and histological data, with occasional recourse to molecular biology techniques such as polymerase chain reaction (PCR) or in situ hybridisation (FISH). This review will focus on the pathogenesis of renal damage during viral infections, its epidemiological, clinico-biological and histological aspects, therapeutic mechanisms and prevention tools.

Pathogeny [1]

Renal pathologies following viral infections have become an emerging public health problem in both developed and developing countries. Almost all viral infections can be complicated by kidney damage, which is why it is important to understand the pathophysiological mechanisms involved. Table I details the multiple processes involved in viral nephropathy, depending on the tropism of the virus for the kidney. In glomerular disease, particularly acute glomerulonephritis (AGN), the cytopathic effect of the virus on the glomerulus induces cell proliferation secondary to the release of cytokines. Nephropathy is reversible in most cases if the virus is rapidly eliminated. In chronic forms of glomerulonephritis, persistent viral infection provides continuous antigenic stimulation, leading to the production of potentially pathogenic antibodies and the formation of immune complexes derived from the bloodstream or formed in situ. In this lineage, hepatitis C virus (HCV)-induced membranoproliferative glomerulonephritis (MPGN) and circulating cryoglobulin production are considered to be an abnormal and excessive host response to infection. In acute renal failure associated with hantavirus or severe

acute respiratory syndrome (SARS) coronavirus infection, the pathogenic mechanisms of tubulointerstitial nephritis and multivisceral failure rather than immune complex formation predominate. Table II lists the main viruses known to induce kidney disease.

Table I: Mechanisms of viral-induced kidney damage

Directs mechanisms	Indirects mechanisms
- Direct cytopathogenic effect on glomerular and tubulointerstitial cells - In situ immune mediation involving viral antigens bound to glomerular structures - Expression of viral proteins or pathogenic pro-inflammatory factors in renal tissue, inducing multiple reactions: • Cell death by necrosis or apoptosis • Increased or decreased cell matrix synthesis, or both • Release of cytokines, adhesion molecules and growth factors	- Circulating immune complexes involving viral antigens and host antiviral antibodies, as well as endogenous antigens modified by viral lesion and host autoantibodies - Hemodynamic disorders, multivisceral failure - Rhabdomyolysis accompanying certain viral infections - Nephrotoxicity of antiviral treatment

Table II: Main viral infections causing nephropathy

Acute glomerulonephritis	Chronic glomerulonephritis	Tubulointerstitial nephritis (Sometimes accompanied by aggravating factors such as hypotension, multivisceral failure, rhabdomyolysis and hepatorenal syndrome)
- Parvovirus B19 - Hepatitis A virus - Measles - Yellow fever - Epstein-Barr virus	- Hepatitis B virus - Hepatitis C virus - HIV (Human Immunodeficiency Virus) - Parvovirus B19	- Hantavirus - SARS Coronavirus - BK virus - Dengue fever - Epstein-Barr virus - Influenza A- Virus Coxsackie B - Cytomegalovirus

Table III: Classification of HIV-associated kidney disease

Glomerular damage	Les atteintes tubulo-interstitielles
- Segmental and focal glomerulosclerosis • Collapsing glomerulopathy - Membranous glomerulopathy • Co-infection with HBV • Co-infection with syphilis - Membranoproliferative glomerulonephritis • Co-infection with HCV • Mixed cryoglobulinemia - Lupus-like glomerulopathy - IgA nephropathy - Post-infectious AGN - TMA - Immunotactoid glomerulopathy	- Acute tubulointerstitial nephritis • CMV co-infection • Drug nephrotoxicity - Crystalline nephropathy • Indinavir - Acute tubular necrosis

CMV: Cytomegalovirus; AGN: acute glomerulonephritis; TMA: thrombotic microangiopathy; HBV: hepatitis B virus; HCV: hepatitis C virus.

Epidemiological, clinico-biological and histological aspects

Viral nephropathies can be divided into two groups, those occurring in an immunocompromised host and those preferentially affecting the immunocompetent subject. Although there may be some overlap, we will discuss the epidemiological, clinico-biological and histological aspects of kidney damage during viral infections, which have a heterogeneous geographical distribution, depending on immune status.

Viral nephropathy in immunocompetent subjects

Viral hepatitis C (HCV)

HCV is an RNA virus belonging to the flaviridae family, responsible for three main types of kidney disease: MPGN, cryoglobulinaemia and membranous glomerulopathy (MG).

MPGN [2, 3]

Chronic HCV infection remains the leading cause of infectious MPGN, whether or not accompanied by the development of cryoglobulinemia. It corresponds to so-called "immune complex" MPGN. Clinical symptoms include hematuria, nephrotic proteinuria and mild-to-moderate renal failure. Serum complement is often low, particularly the C3 fraction. Approximately 50% of patients present with circulating cryoglobulins. Histologically, this is an immune-complex MPGN with endocapillary proliferation, accentuated matrix synthesis giving a double-contoured appearance, and C3 deposits on the outer side of the glomerular basement membrane. Subendothelial and mesangial deposits are rarer. On immunofluorescence, C3 deposits are abundant, associated with predominantly polyclonal IgG kappa and lambda deposits.

Cryoglobulinaemia type II or III (mixed) [4-6]

Cryoglobulinaemia corresponds to small-vessel vasculitis with joint, skin, neurological and renal involvement. HCV accounts for 40% to 90% of the causes of type II cryoglobulinaemia. Renal involvement is manifested by MPGN with immune complexes, present in over 30% of mixed cryoglobulinaemias and concomitant with diagnosis in 20% of cases. The clinical picture is one of chronic glomerulonephritis with haematuria and nephritic syndrome in 50% of cases, rapidly progressive glomerulonephritis (RPGN) in 14% and impure nephrotic syndrome in 21% of cases. Histologically, it is a form of MPGN characterised by predominant mesangial proliferation and lobulation of the flocculus, which is frequently infiltrated by activated monocytes and T lymphocytes. There are also classically voluminous PAS+ subendothelial and mesangial deposits which correspond to immune complexes deposited along the MBG and to cryoprecipitate in immunofluorescence. These cryoglobulin deposits are organised into microtubules which can be seen by electron microscopy. In 30% of cases, there are lesions of renal vasculitis associated with luminal thrombi, fibrinoid necrosis of small vessels and a perivascular infiltrate of monocytes and CD8+ lymphocytes. Classically, there are also intravascular hyaline thrombi which correspond to cryoprecipitate in immunofluorescence.

MG [7-9]

Chronic viral hepatitis due to HCV can be complicated by GEM. It is manifested by a clear nephrotic syndrome and, unlike MPGN, serum complement levels are normal. Serum cryoglobulin levels are also normal. The histological appearance is identical to that seen in idiopathic GEM. The glomeruli are normocellular, with diffuse thickening of the glomerular basement membrane (GBM). Immunofluorescence always shows diffuse granular staining of the glomerular capillary wall, with IgG and C3 predominating.

Diagnosis in the 3 entities (MPGN, cryoglobulinaemia and GEM) is based on serological screening detecting anti-HCV antibodies and molecular confirmation tests detecting or quantifying HCV RNA by PCR. It is important to remember that immunocompromised patients (haemodialysis patients and kidney transplant recipients) may lack serological evidence of HCV (false negative). In this sub-group, HCV RNA testing becomes crucial for diagnostic confirmation.

Viral hepatitis B (HBV)

HBV is a DNA virus belonging to the hepadnaviridae family, responsible for three types of kidney disease: GEM, MPGN and IgA nephropathy (IgAN).

MG **[8, 10, 11]**

MG is common in chronic HBV infection. It is commonly observed in chronic HBV carriers and is typical of endemic areas. Its clinical manifestation is a nephrotic syndrome in most cases, often accompanied by microscopic haematuria. The histological signs are similar to those of MG secondary to HCV infection described above. The presence of HBV antigen-antibody immune complexes on immunofluorescence in the glomeruli further confirms the direct causal relationship between viral infection and glomerular disease.

MPGN [11]

MPGN secondary to HBV infection is less common than GEM. Its clinical presentation is hypertension, renal failure and nephrotic syndrome. Cryoglobulins are not normally found, so PAS-positive protein thrombi are not a feature of MPGN in HBV infection. The histological appearance of MPGN in HBV infection is similar to other non-cryoglobulinemic MPGN.

IgA nephropathy (NIgA) [9]

IgA nephropathy in HBV infection is most often manifested by microscopic haematuria and nephrotic proteinuria. Histologically, there is mesangial proliferation with immunofluorescence of mesangial IgA deposits (sometimes associated with IgG and C3 deposits), a feature also found in other idiopathic forms of NIgA.

Diagnosis in the 3 entities (GEM, MPGN and NIgA) is based on serological screening for HBV (HBs antigen, HBe antigen, anti-Hbs and anti-Hbc antibodies) combined with molecular confirmation tests detecting or quantifying HBV DNA by PCR.

Hantavirus [18, 19, 20]

Hantaviruses are RNA viruses of the Bunyaviridae family, with endothelial tropism, whose main carriers are rodents (rats, bats). Hantavirus infection leads to two major forms of the disease: haemorrhagic fever with renal syndrome (HFRS), which

predominates in Europe and Asia, and hantavirus cardiopulmonary syndrome (HCPS), which is rife in the United States and China. Contamination occurs through inhalation of dust containing rodent excreta. Contamination by bite is exceptional. The clinical manifestations of HFRS include cutaneous and mucosal haemorrhaging, fleeting visual disturbances, with varying degrees of shock, and renal failure with cardiogenic pulmonary oedema. Biologically, there is almost constant thrombocytopenia in the first few days. Liver damage mimicking viral hepatitis is found in 50% of cases. The histological picture is that of an acute interstitial nephritis composed of a mononuclear inflammatory infiltrate with haemorrhagic interstitial suffusions predominating in the medulla. Immunofluorescence and electron microscopy are non-specific. Hanta virus may be detected in endothelial cells by immunohistochemistry. Diagnosis of the virus is serological, with detection of immunoglobulins M and G (IgM/IgG) against the N-nucleoprotein antigen (N-antigen).

SARS-CoV-2 [15]

SARS-CoV-2 is an RNA virus belonging to the coronaviridae family. It shares approximately 79% genetic similarity with SARS-CoV, the agent responsible for the SARS pandemic of 2002-2003, and 51.8% similarity with the Middle East Respiratory Syndrome coronavirus (MERS-CoV). Human angiotensin-converting enzyme 2 (ACE2) has been identified as the functional receptor for SARS-COV-2 in humans. Acute renal failure occurs in approximately 25-30% of patients admitted to intensive care following Covid 19. The most common form of renal involvement is acute tubular necrosis associated with low proteinuria, aminoaciduria, hypouricaemia and hypophosphoraemia, reflecting proximal tubular dysfunction.

Viral nephropathy in immunocompromised patients

This is a group of viral nephropathies secondary to viral infections by viruses with the particularity of persisting in a latent state after primary infection in the target organs, particularly the kidney, and reactivating in situations of immunosuppression such as renal transplantation, chronic haemodialysis, infection by the human immunodeficiency virus (HIV), etc.

BK virus nephropathy [22, 23]

The BK virus or Poliomavirus is a DNA virus belonging to the JC virus family, with a prevalence of infection of up to 80% in the general population. Infection occurs via the respiratory route. BK virus nephropathy (BKN) is currently the most common viral disease affecting kidney transplant recipients, with a prevalence 10 to 20 times higher than that of Cytomegalovirus kidney infections (1 to 5.5% in various transplant centres). It is manifested by varying degrees of graft dysfunction. Histologically, it is an interstitial nephritis characterised by the presence of intranuclear viral inclusions in tubular cells, with foci of focal necrosis. The diagnosis is confirmed immunohistochemically by detection of the SV40 T antigen, common to all Poliomavirus infections, or by in situ PCR in search of the viral genome.

Adenoviruses (ADV) [24, 25]

Adenoviruses are DNA viruses with epithelial tropism. Renal disease is caused by the B subgroup, which has a tropism for tubular cells. Clinical symptoms in renal transplant patients include flu-like symptoms, fever, pain in the kidney graft, macroscopic haematuria, most often due to localised bladder damage (haemorrhagic cystitis), and extrarenal damage such as pneumopathy and/or severe enterocolitis, sometimes life-threatening. Biologically, there is acute oliguric renal failure, with low tubular proteinuria and aseptic leukocyturia. The histological picture is that of acute interstitial nephritis, with an inflammatory infiltrate composed of lymphocytic mononuclear cells, histiocytes, neutrophils and necrotising granulomas, as well as haemorrhagic suffusions. Viral inclusions, similar to those seen in BKN, are found in tubular cells, where the genome can be detected by immunohistochemistry. Serological diagnosis is of little value, and only detection of the viral genome by PCR or immunohistochemistry provides a definitive diagnosis.

Cytomegalovirus CMV [20, 30]

CMV or human herpes virus 5 (HHV-5) is a DNA virus belonging to the Herpesviridae family. Contamination occurs horizontally through direct contact with contaminated biological fluids. In transplant patients, CMV infection is the most common opportunistic virus infection. A distinction is made between CMV infection, which corresponds to a positive diagnostic test (culture, antigen detection or PCR) in the absence of symptoms, and CMV disease, which corresponds to detection of the virus in the blood compartment in the presence of clinico-biological symptoms such as fever, pancytopenia and organ damage. Renal damage is manifested as tubulointerstitial nephritis with graft dysfunction. This may induce or exacerbate pre-existing anaemia, as CMV has been shown to inhibit αHIF2 (Hypoxia Inducible Factor) in CMV-infected patients. Kidney biopsy reveals a nodular infiltrate of mononuclear inflammatory cells in the interstitium, with bulky intranuclear inclusions separated from the nuclear membrane by a clear halo, giving a characteristic "owl's eye" appearance. Diagnosis is based on the clinical context, histological examination, PCR on blood samples or any other biological fluid, and cell culture. The latter provides viral strains and enables antivirograms to be performed in the event of resistance to treatment.

Epstein-Barr virus (EBV) [9]

EBV or human herpes virus 4 (HHV-4) is a DNA virus belonging to the Herpesviridae family, with a strictly human reservoir and essentially salivary transmission, since the virus has a tropism for the

oropharynx. In kidney transplants, the EBV virus induces a series of disorders known as PTLD (Post Transplant Lymphoproliferative Disorders). The clinical picture is non-specific, and may manifest as a prolonged, unexplained fever. Histological examination of the graft reveals interstitial inflammation with an infiltrate of nodular mononuclear cells. The presence of lymphoblasts associated with other lymphoid cells is highly characteristic of PTLD. The diagnosis is confirmed by immunohistochemistry (predominance of CD 20+ B lymphocytes) or in situ hybridisation.

Parvovirus B19 (PVB) [21]

PVB is a DNA virus of the Parvoviridae family whose only host is man. Contamination, often during childhood, occurs through inhalation of the virus in aerosol droplets, but also from mother to foetus, during blood transfusions and organ transplants. Symptomatic infection with PVB occurs in 2% to 12% of patients during the first year following renal transplantation. Clinically, non-renal manifestations include hydramnios in pregnant women, erythema infectiosum or "fifth disease" in children, and acute anaemia secondary to a transient aplastic crisis and chronic red cell aplasia in haemodialysis patients and kidney transplant recipients. The renal manifestations are diverse and often represent post-infectious glomerulonephritis with endocapillary proliferation, but also non-proliferative damage such as LGM, HSF and GEM. Rarely, parvovirus infection can lead to acute or chronic renal graft rejection. Serology for specific antibodies to viral capsid antigens is the most common diagnostic method in immunocompetent patients. Identification of viral DNA by PCR is the preferred method for diagnosis in immunocompromised patients.

Human immunodeficiency virus (HIV) [23-27]

HIV is an RNA virus belonging to the retroviridae family. It can affect all organs, with renal involvement common, with an estimated prevalence of 30% of patients. Whether it occurs acutely or chronically is closely linked to the degree of immunodepression, viral load, blood volume status and infections. Table III lists the different forms of renal impairment associated with HIV infection.

HIVAN (HIV associated nephropathy), a specific entity, occurs in 2 to 10% of the HIV-infected population at all stages of the disease, almost exclusively in black adults and children, and is associated with severe renal impairment. It is typically a rapidly progressive glomerulonephritis, often associated with an impure nephrotic syndrome progressing rapidly to the terminal stage. Biological investigations sometimes reveal autoantibodies, even in the absence of any signs of autoimmune disease, with cryoglobulinaemia being more common in patients co-infected with HCV. Histologically, it is a collapsing HSF with proliferative and enlarged podocytes giving a pseudo-crescent appearance pathognomonic of HIVAN. Tubulointerstitial involvement is frequent, with an interstitial infiltrate of CD8 T lymphocytes and very characteristic microcystic tubule dilatations. Immunofluorescence shows IgM and C3 deposits in the mesangium and capillary walls.

Therapeutic principles and preventive measures [21, 28, 29]

The management of viral nephropathy is generally based on symptomatic nephroprotective measures, particularly in at-risk populations, by maintaining good hydration, avoiding examinations requiring the use of iodinated contrast media, stopping the use of non-steroidal anti-inflammatory drugs, diuretics and all nephrotoxic drugs, and regular monitoring of glomerular filtration rate. Renal replacement therapy is exceptional. Recommendations suggest a reduction in immunosuppression for viral reactivation in renal transplant patients. Specific antiviral therapy is only available for the eradication of a few viruses (HCV, HBV, HIV and CMV). In the case of GNMP associated with cryoglobulinaemia, antiviral treatment is not always sufficient to overcome the renal manifestations, especially in the case of GNRP. Eradication of immune complexes by plasma exchange and specific immunosuppressive treatment targeting B lymphocytes, such as rituximab, are essential therapeutic approaches in the treatment of Cryo-HCV. In the case of HIV infection, cardiovascular risk management through physical exercise, smoking cessation and the prescription of antiplatelet agents and statins must be an integral part of treatment. Finally, vaccination against certain viruses such as HBV remains an effective way of protecting against the damaging effects of viral infections on the kidney.

CONCLUSION

Renal damage during viral infections is common. The SARS CoV2 pandemic is the main example. Viruses induce nephropathy in a variety of ways, depending essentially on their tropism for the renal cell. Diagnosis is based essentially on serology and detection of viral nucleic acids by PCR or IHC. In the absence of specific antiviral therapies, symptomatic management is the basis of treatment. Prevention, especially in at-risk populations, through systematic screening, nephroprotection measures and vaccination remains the cornerstone of viral nephropathy management.

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