Viruses and collapsing glomerulopathy: a brief critical review

Preeti Chandra1 and Jeffrey B. Kopp2

1Nephrology Division, University of Maryland School of Medicine, Baltimore, MD, USA and 2Kidney Disease Section, NIDDK, NIH, Bethesda, MD, USA

Correspondence and offprint requests to: Jeffrey B. Kopp; E-mail: jbkopp@nih.gov

Abstract

Background. Collapsing glomerulopathy may occur in an idiopathic (primary) form and in association with a wide spectrum of infectious and inflammatory conditions and medications. The association of collapsing glomerulopathy with human immunodeficiency virus (HIV)-1 infection is well established; less certain is the association with other viral infections.

Methods. We searched PubMed for articles in all languages that addressed glomerulopathies associated with parvovirus B19, cytomegalovirus (CMV), Epstein-Barr virus (EBV), hepatitis C virus (HCV) and simian virus 40 (SV40).

Results. Case reports and small-case series link infection with these common viruses and glomerular injury. The evidence for a pathogenic role is generally stronger for glomerulonephritis than for collapsing glomerulopathy.

Conclusions. The evidence linking collapsing glomerulopathy with CMV is relatively strong but not yet conclusive, while the evidence for a pathogenic role for EBV and parvovirus B19 is weaker.

Keywords: collapsing focal segmental glomerulosclerosis; cytomegalovirus; Epstein-Barr virus; parvovirus B19; podocyte

Collapsing glomerulopathy may be considered either a variant of focal segmental glomerulosclerosis (FSGS) [1] or a distinct pathologic entity [2], but there is general agreement on the key histologic features. The term ‘collapsing glomerulopathy’ was first described by Weiss et al. to describe a distinct entity with progressive renal failure and pathological features characterized by segmental or global glomerular capillary collapse and visceral epithelial cells swelling and hyperplasia with hyaline droplets and extensive tubulointerstitial inflammation [3]. Subsequent studies from Detwiler et al. and Valeri et al., including subjects with and without HIV infection, reported a similar pathologic phenotype. Compared with patients with classic FSGS, patients with collapsing glomerulopathy were more likely to be of African descent and had higher serum creatinine, more proteinuria at the time of kidney biopsy and worse renal survival [4, 5]. Compared with classic FSGS, collapsing glomerulopathy less likely causes sclerosis and hyalinosis of the capillary tuft and capsular adhesions. The collapsing variant of FSGS is defined by the Columbia classification as the presence of segmental capillary tuft collapse (wrinkling and folding) in at least one glomerulus. It is characterized by global or segmental collapse of the glomerular capillary walls associated with wrinkling of the glomerular basement membrane. There is marked hypertrophy and hyperplasia of the visceral epithelial cells sometimes forming pseudocrescents. These are differentiated from true crescents by the lack of intercellular matrix or attachment to the Bowman’s capsule [6].

The mechanism that leads to the altered phenotype of the visceral epithelial cells in collapsing glomerulopathy is not well understood. Barisoni et al. noted that these cells lacked certain podocyte differentiation markers, including Wilms tumor 1, synaptopodin, podocalyxin, PTPRO/GLEPP1 and C3B receptor [7]. Further, podocytes are normally terminally differentiated, non-dividing cells. In collapsing glomerulopathy, the visceral epithelial cells re-enter the cell cycle, as evidenced by Ki-67 expression [7] and in a genetic mouse model of collapsing glomerulopathy, expression of cyclin D1 and reduced expression of cyclin-dependent kinase inhibitors such as p27 and p57 [8]. Evidence has been put forward implicating increased levels of human telomerase reverse transcriptase and Wnt signaling in the altered podocyte phenotype [9]. These features suggested a process of podocyte dysregulation, distinct from either injury or dedifferentiation. This morphological and gene–protein expression profile is similar in HIV-associated and idiopathic collapsing glomerulopathy. An important advance has been the recognition that in collapsing glomerulopathy, visceral epithelial cells bear markers of the parietal epithelium [10] and may represent replenishment from a glomerular stem cell pool that is present within the parietal epithelium [11, 12]. The molecular mechanisms that lead to the postulated excessive and dysregulated stem cell replacement remain to be defined. Relevant to the current topic, a better understanding of the role that viral infection and viral gene products play in altered glomerular cell biology may help define relevant physiologic pathways and may suggest possible therapeutic targets for
collapsing glomerulopathy for diverse forms of collapsing glomerulopathy.

Collapsing glomerulopathy has been associated with several conditions including infections due to viruses, *Myco- coccus tuberculosis*, filariasis (Loa loa), leishmaniasis, *Campylobacter enteritis*; autoimmune diseases, including lupus and adult Still’s disease; malignancies, including natural killer cell leukemia and hemophagocytic syndrome; genetic mutations, including certain mitochondrial disorders; medications, including interferon alpha, beta and gamma, and pamidronate; and following kidney transplantation. A number of excellent recent reviews are available [13, 14]. For many associations, the number of cases is few, particularly considering the frequency of the co-occurring disease, which raises the possibility of a chance occurrence.

Our goal will be to review the strength of the evidence linking particular viral infections with collapsing glomerulopathy and to some extent, with glomerular disease in general (Table 1). Our focus will be on three DNA viruses: Epstein-Barr virus (human herpesvirus-4), cytomegalovirus (CMV, human herpesvirus-5) and parvovirus B19. Individuals are most commonly infected with these viruses in childhood, although in the industrialized world primary infection may be delayed until adolescence or adulthood. All three viruses may establish latent infections in particular tissues.

The association between HIV-1 infection and collapsing glomerulopathy has been well established and will not be reviewed further here. When we move beyond HIV infection, there are reports that suggest that other viral infections are associated with collapsing glomerulopathy. What are the criteria that might be applied to determine causation in these settings, given the practical difficulties in applying Koch’s four postulates to many of these clinical problems? The following might be considered as important elements in building a case that a particular virus is the cause of collapsing glomerulopathy.

- **Demonstration of collapsing glomerulopathy occurring in multiple cases involving viral infection.** It is quite possible that viral infection of the glomerulus may cause collapsing glomerulopathy in only a fraction of patients with a particular viral infection. Further, a virus may cause collapsing glomerulopathy in some patients and cause another histologic variant in other patients, or cause minimal pathologic changes. HIV infections provide an example, where individuals with the risk genotype (two *APOL1* risk alleles) most likely manifest either collapsing glomerulopathy [15] or classic FSGS [16] and individuals lacking risk alleles (e.g. persons of European descent) tend not to have these forms of glomerulopathy.

- **Demonstration of clear-cut collapsing glomerulopathy including elements of both glomerular tuft collapse and altered visceral epithelial cell phenotype.** There can be a range of manifestations, from a single glomerulus with a cluster of prominent podocytes coupled with a collapsed glomerular segment to global glomerular collapse with numerous cells in Bowman’s space. Thresholds for the diagnosis of collapsing glomerulopathy may differ among pathologists, which can be problematic.

- **Demonstration of viral protein or nucleic acid within glomerular cells, and particularly localization to podocytes.** Evidence for infection should be sought, but it is possible that glomerular injury may be found in the absence of evidence for infection, if collapsing glomerulopathy is due to a bystander effect of virus-driven inflammatory response or due to circulating viral gene products. Further, it may not be possible to use conventional markers of differentiated podocytes such as nephrin, podocin, synaptopodin and protein tyrosine receptor phosphate type O (alternatively known as GLEPP1), as expression of these markers may be lost. In this case, the infected cells are shown to be dysregulated podocytes or stem cells by expression of particular markers or inferred to be these cell types by their characteristic location, e.g. within Bowman’s space. Appropriate controls are important, ideally including non-viral forms of collapsing glomerulopathy.

- **Demonstration that viral infection in experimental animals induces some of (or all) the features of collapsing glomerulopathy, which may be a difficult

| Collapsing glomerulopathy | Other glomerulopathies associated with infection | Tubulointerstitial disease |
|---------------------------|---------------------------------------------|----------------------------|
| HIV-1 Established cause   | FSGS, Mesangial proliferative glomerulonephritis, including IgG predominance, IgA predominance and ‘full house’ immunoglobulins, Thrombotic microangiopathy | Microcystic tubular dilatation is characteristic |
| Parvovirus B19 Possible cause | Mesangial proliferative glomerulonephritis, leukocyte infiltration, mesangiolysis (some) cases, IgG predominance, Mesangial proliferative glomerulonephritis with predominance of IgA (Henoch-Schönlein-like), Thrombotic microangiopathy | Interstitial nephritis |
| CMV Probable cause        | Thrombotic microangiopathy, Diffuse mesangial sclerosis, Membranous nephropathy, Thrombotic microangiopathy | Interstitial nephritis |
| EBV Possible cause        | Mesangial proliferative glomerulonephritis, Crescentic glomerulonephritis, Membranous nephropathy | Interstitial nephritis |

Four viruses are plausible causes of collapsing glomerulopathy, and each of these viruses has other renal manifestations.
propagation when the susceptible host range is restricted. Alternatively, it may be possible to demonstrate that viral gene products expressed in transgenic animals induce collapsing glomerulopathy.

Parvovirus B19 was first associated with glomerulonephritis in seven patients with sickle cell disease, in whom acute infection and the consequent aplastic crises were associated with segmental proliferative glomerulonephritis and associated at a later stage with FSGS [17]. Tubulointerstitial changes varied from severe to absent. Subsequent case reports described similar findings in children and adults with primary parvovirus B19 infections, showing either segmental or global endocapillary proliferation, sometimes with mesangiolysis and with immunoglobulin and complement C3 deposition most intense along the glomerular capillary wall and electron dense deposits located in the subendothelial space and the mesangium [18], [19]. In one case, there was predominantly IgA deposition resembling Henoch-Schönlein syndrome [20]. Murer et al. described four cases of thrombotic microangiopathy occurring in kidney transplant during acute parvovirus B19 infection [21].

Parvovirus DNA was present in kidney tissue [22–24], although the presence of viral genome in circulating immune cells cannot be excluded as a source of this signal and DNA was localized to glomerular endothelial cells [20] but not to visceral epithelial cells. In some but not all cases studied, parvovirus antigens were localized to glomerular cells that were not further characterized [19, 23], but were entirely absent in six cases [25]. Thus, a major unresolved issue is the localization of parvoviral nucleic acid and protein within kidney tissue from individuals with parvovirus B19 infection; it is unclear whether this is due to methodologic or biologic differences or to inclusion of subjects lacking parvoviral infection or at different stages of glomerular disease evolution [26].

An association between parvovirus B19 and collapsing glomerulopathy was first noted by Moudgil et al. in a renal transplant patient [27]. Subsequently, these investigators reported the following rates of PCR detection of parvovirus B19 DNA in archival, paraffin-embedded kidney tissue: collapsing glomerulopathy, 18 of 23 cases (78%); HIV-associated nephropathy, 3 of 19 cases (16%); FSGS, 6 of 22 cases (22%); controls including hematuria, thin basement membrane disease, minimal-change nephropathy and tumor nephrectomy samples, 6 of 27 cases, (22%) [28]. Parvoviral DNA was localized to glomerular parietal cells and visceral epithelial cells using in situ hybridization. In a replication study, Tanawattanacharoen et al. amplified parvoviral B19 DNA from frozen tissue blocks and found a marginally higher prevalence of parvoviral DNA in collapsing glomerulopathy (9 of 10 cases) and FSGS (8 of 10 cases) but also found viral DNA in membranous nephropathy (6 of 10 cases), minimal-change nephropathy (5 of 10 cases) and tumor nephrectomy (2 of 4 cases) [29]. In situ hybridization studies were not able to identify the cellular location of the parvoviral DNA, despite suitable positive control tissue (parvovirus B19 infected placental tissue), suggesting that viral copy number may be low in kidney tissue. Thus, while the results of the two studies differ numerically, the combined dataset suggests that parvoviral DNA is frequently detected in kidney tissue, and the rates are highest in diseases associated with podocyte/visceral epithelial cell dysfunction. These data may indicate that kidney tissue is a location where latent DNA remains long after primary infection with this common childhood viral illness, and that re-emergence may be associated with collapsing glomerulopathy or FSGS in particular. The data, at present, appear to be insufficiently strong to establish a causative role for parvovirus B19 in collapsing glomerulopathy; the data could also be due to glomerular or other cells experiencing reactivation of a latent viral infection as a result of cell injury or dysregulation (podocyte) or differentiation (parietal epithelial stem cell).

The first glomerular diseases associated with CMV infection were in the setting of congenital infection, and included proliferative glomerulonephritis, sometimes with necrotizing features [30], and diffuse mesangial sclerosis [31]. Evidence for a direct role of viral infection included cytomegalic inclusion bodies and/or viral particles within glomerular cells. CMV-associated renal infections have perhaps been most frequently described in the setting of renal transplant. In a recent series from India, Rane et al. described 10 cases with involvement of glomeruli (three cases), tubulointerstitium (six cases) or both (one case) [32]. The characteristic viral cytopathic changes were seen in glomerular endothelial cells and podocytes; three patients had thrombotic microangiopathy. Other glomerular disease reports associated with CMV infection have described membranoproliferative glomerulonephritis [33], membranous nephropathy [34] and thrombotic microangiopathy [35]; the links are based on the temporal relationship with infection and in some cases, response to anti-viral therapy.

Collapsing glomerulopathy has been reported in three cases of acute CMV infection, occurring in immunocompetent individuals. Presne et al. described a 16-year-old male with abrupt onset of nephrotic syndrome, renal biopsy showing collapsing glomerulopathy and improvement following therapy with glucocorticoids and ganciclovir [36]. Tomlinson et al. described a 60-year-old woman with acute nephrotic syndrome illness and acute CMV infection, with viremia and IgM antibodies [37]. A renal biopsy showed collapsing glomerulopathy and CMV DNA was detected (but not localized) in the renal biopsy by PCR. The patient progressed rapidly to end-stage kidney disease. In this issue of the Clinical Kidney Journal, Grover et al. described a 34-year-old man who presented with acute CMV infection with viremia and IgM titers, nephrotic-range proteinuria and renal failure requiring hemodialysis [38]. A renal biopsy showed findings consistent with collapsing glomerulopathy. CMV DNA was not detected in renal biopsy tissue, although the methods were not described. Therapy with ganciclovir (4 weeks) and glucocorticoids (6 months) was associated with improved renal function, allowing her to stop chronic dialysis. All three individuals were of Caribbean descent, either African or Hispanic ancestry, and thus, possibly carry APOL1 renal risk alleles that predispose to collapsing glomerulopathy [39]. None of the cases showed characteristic cytomegalic changes in the kidney, specifically not in glomeruli. Nevertheless, taken together, these cases provide substantial evidence that acute CMV infection is a cause of collapsing glomerulopathy and further suggest that therapy with glucocorticoids and anti-viral therapy may be beneficial in stabilizing or reversing glomerular cell injury.

Epstein-Barr virus (EBV) infection has been associated with idiopathic chronic interstitial nephritis, with viral DNA localized to tubular epithelial cells [40]. Viral DNA was similarly localized in the case of an HIV-infected individual with interstitial nephritis [41], but viral DNA
was not found in cases of idiopathic acute interstitial nephritis [42, 43]. Kunimoto et al. hypothesized that EBV contributes to IgA nephropathy, but no viral DNA was found in kidney tissue in a case series [44]. A case report described a case of acute EBV infection associated with crescentic glomerulonephritis; while viral protein and nucleic acid localization studies were not performed, but the temporal association suggests that EBV played a role, possibly via immunologic response [45]. Four cases of EBV-associated membranous nephropathy have been reported [46–48]—one case with acute viral syndrome, one case associated with chronic viremia and two cases with malignancy. Recently, Joshi et al. reported in the Clinical Kidney Journal a case of a young woman who presented with acute EBV infection and collapsing glomerulopathy [49]. In situ hybridization studies failed to identify EBV DNA in kidney tissue. To summarize the glomerular diseases reported with EBV infection, it appears that EBV may cause membranous nephropathy (likely via a planted antigen and in situ formation of immune complexes), crescentic glomerulonephritis (possibly via immunologic mechanisms) and perhaps collapsing glomerulopathy (via unknown mechanisms), although more cases will be required to strengthen the association.

It is quite possible that other viruses cause idiopathic collapsing glomerulopathy. Hepatitis C virus (HCV) has been associated with collapsing glomerulopathy in four cases, excluding those cases involving co-infection with HIV [50, 51], but viral RNA or protein has not been localized to glomeruli and to date there has been no evidence that HCV plays a pathogenic role. Simian virus 40 (SV40) is a monkey virus present in primary rhesus monkey kidney cells that were used to grow poliovirus in the period 1955–1963; whether SV40 infections occur in human population at present is a subject of considerable controversy [52, 53]. Four groups have investigated a possible role of SV40 in human glomerular disease, including collapsing glomerulopathy and FSGS. Two reports presented evidence against such infections [54, 55] and two reports (including a report by one of the present authors) presented preliminary evidence for such infections [56, 57]. Viral DNA and protein have not been localized to the kidney. It is our present opinion that the evidence for SV40 infections associated with human glomerular disease is weak and that a causal role is unlikely.

In conclusion, the evidence suggesting that several viruses, certainly HIV-1, probably CMV and possibly parvovirus B19 and EBV, have the potential to cause collapsing glomerulopathy. The diagnosis of a viral infection has therapeutic relevance, particularly for HIV and possibly for CMV. For the non-HIV infections, the diagnostic criteria remain to be established, with the clinical role of immunostaining and in situ hybridization not part of routine pathologic practice. For these infections, the evidence base linking these viruses to collapsing glomerulopathy deserves further systematic investigation. Further work should also address the molecular mechanisms of glomerular cell injury and the role of host responses, including the role of host genetic susceptibility.

Acknowledgement. Funding. This work was supported in part by the NIDDK, NIH Intramural Research Program.

Conflicts of interest statement None declared.

References

1. D’Agati VD, Kaskel FJ, Falk RJ. Focal segmental glomerulosclerosis. N Engl J Med 2011; 365: 2398–2411
2. Barisoni L, Schnaper HW, Kopp JB. Advances in the biology and genetics of the podocytopathies: implications for diagnosis and therapy. Arch Pathol Lab Med 2009; 133: 201–216
3. Weiss MA, Daquioag E, Margolin EG et al. Nephrotic syndrome, progressive irreversible renal failure, and glomerular ‘collapse’: a new clinicopathologic entity? Am J Kidney Dis 1986; 7: 20–28
4. Detwiler RK, Falk RJ, Hogan SL et al. Collapsing glomerulopathy: a clinically and pathologically distinct variant of focal segmental glomerulosclerosis. Kidney Int 1994; 45: 1416–1424
5. Valeri A, Barisoni L, Appel GB et al. Idiopathic collapsing focal segmental glomerulosclerosis: a clinicopathologic study. Kidney Int 1996; 50: 1734–1746
6. Albquumi M, Soos TJ, Barisoni L et al. Collapsing glomerulopathy. J Am Soc Nephrol 2006; 17: 2854–2863
7. Barisoni L, Kriz W, Mundel P et al. The dysregulated podocyte phenotype: a novel concept in the pathogenesis of collapsing idiopathic focal segmental glomerulosclerosis and HIV-associated nephropathy. J Am Soc Nephrol 1999; 10: 51–61
8. Barisoni L, Madaio MP, Eraso M et al. The kd/kd mouse is a model of collapsing glomerulopathy. J Am Soc Nephrol 2005; 16: 2847–2851
9. Shkreli M, Sarin KY, Pech MF et al. Reversible cell-cycle entry in adult kidney podocytes through regulated control of telomerase and Wnt signaling. Nat Med 2012; 18: 111–119
10. Dijkman H, Smeets B, van der Laak J et al. Thrombotic microangiopathy: a new clinicopathologic entity? Am J Kidney Dis 2011; 57: 246–250
11. Murer L, Zacchello G, Bianchi D et al. Idiopathic extracapillary proliferative glomerulonephritis associated with human parvovirus B19 infection. Clin Nephrol 2002; 57: 246–250
Viruses and collapsing glomerulopathy

23. Ohtomo Y, Kawamura R, Kaneko K et al. Nephrotic syndrome associated with human parvovirus B19 infection. Pediatr Nephrol 2003; 18: 280–282

24. Tolaymat A, Al Mousily F, MacWilliam K et al. Parvovirus glomerulonephritis in a patient with sickle cell disease. Pediatr Nephrol 1999; 13: 340–342

25. Takeda S, Takaeda C, Takazakura E et al. Renal involvement induced by human parvovirus B19 infection. Nephron 2001; 89: 280–285

26. Waldman M, Kopp JB. Parvovirus B19 and the kidney. Clin J Am Soc Nephrol 2007; 2: 547–556

27. Moudgil A, Shidban H, Nast CC et al. Parvovirus B19 infection-related complications in renal transplant recipients: treatment with intravenous immunoglobulin. Transplantation 1997; 64: 1847–1850

28. Moudgil A, Nast CC, Bagga A et al. Association of parvovirus B19 infection with idiopathic collapsing glomerulopathy. Kidney Int 2001; 59: 2126–2133

29. Tanawattanacharoen S, Falk RJ, Jennette JC et al. Parvovirus B19 DNA in kidney tissue of patients with focal segmental glomerulosclerosis. Am J Kidney Dis 2000; 35: 1166–1174

30. Beneck D, Greco MA, Feiner HD. Glomerulonephritis in congenital cytomegalic inclusion disease. Hum Pathol 1986; 17: 1054–1059

31. Besbas N, Bayrocki US, Kale G et al. Cytomegalovirus-related congenital nephrotic syndrome with diffuse mesangial sclerosis. Pediatr Nephrol 2006; 21: 740–742

32. Rane S, Nada R, Minz M et al. Spectrum of cytomegalovirus-induced renal pathology in renal allograft recipients. Transplant Proc 2012; 44: 713–716

33. Andresdottir MB, Assmann KJ, Hilbrands LB et al. Type I membranoproliferative glomerulonephritis in a renal allograft: A recurrence induced by a cytomegalovirus infection? Am J Kidney Dis 2000; 35: E6

34. Georgaki-Angelaki H, Lycopoulou L, Stergiou N et al. Membranous nephritis associated with acquired cytomegalovirus infection in a 19-month-old baby. Pediatr Nephrol 2009; 24: 203–206

35. Shiraiishi N, Kitamura K, Hayata M et al. Case of anti-glomerular basement membrane antibody-induced glomerulonephritis with cytomegalovirus-induced thrombotic microangiopathy. Intern Med 2012; 42: e7–e11

36. Presne C, Cordonnier C, Makdassi R et al. Collapsing glomerulopathy and cytomegalovirus, what are the links? Presse Med 2000; 29: 1815–1817

37. Tomlinson L, Boriskin Y, McPhee I et al. Acute cytomegalovirus infection complicated by collapsing glomerulopathy. Nephrol Dial Transplant 2003; 18: 187–189

38. Grover V, Gaikhi M, Schimmer J et al. Cytomegalovirus-induced collapsing focal segmental glomerulosclerosis. Clin Kidney J 2013; 6: 71–73

39. Kopp JB, Nelson GW, Sampath K et al. APOL1 genetic variants in focal segmental glomerulosclerosis and HIV-associated nephropathy. J Am Soc Nephrol 2011; 22: 2129–2137

40. Becker JL, Miller F, Nuovo GJ et al. Epstein-Barr virus infection of renal proximal tubule cells: possible role in chronic interstitial nephritis. J Clin Invest 1999; 104: 1673–1681

41. Frazao JM, Elangovan L, Felsenfeld AJ et al. Epstein-Barr virus-induced interstitial nephritis in an HIV-positive patient with progressive renal failure. Nephrol Dial Transplant 1998; 13: 1849–1852

42. Mansur A, Little MA, Oh WC et al. Immune profile and Epstein-Barr virus infection in acute interstitial nephritis: an immunohistochemical study in 78 patients. Nephron Clin Pract 2011; 119: c293–c300

43. Suzuki J, Komada T, Hirai K et al. An adult case of fulminant Epstein-Barr virus infection with acute tubulointerstitial nephritis. Intern Med 2012; 51: 629–634

44. Kunimoto M, Hayashi Y, Kuki K et al. Analysis of viral infection in patients with IgA nephropathy. Acta Otolaryngol Suppl 1993; 508: 11–18

45. Ranganath R, Pandey AR, Pavan M. Crescentic glomerulonephritis and leucocytoclastic vasculitis associated with acute EBV infection. Nephrology 2011; 16: 617

46. Araya CE, Gonzalez-Peralta RP, Skada-Smith S et al. Systemic Epstein-Barr virus infection associated with membranous nephropathy in children. Clin Nephrol 2006; 65: 160–164

47. Kim CS, Choi YD, Choi JS et al. EBV-positive diffuse large B-cell lymphoma in a patient with primary Sjogren’s syndrome and membranous glomerulonephritis. BMC Nephrol 2012; 13: 149

48. Meyer P, Soete S, Raynaud P et al. Acute inflammatory polyradiculoneuropathy and membranous glomerulonephritis following Epstein-Barr virus primary infection in a 12-year-old girl. Arch Pediatr 2010; 17: 1535–1539

49. Joshi A, Arora A, Cimbalku D et al. Acute Epstein-Barr virus infection-associated collapsing glomerulopathy. Clin Kidney J 2012; 5: 320–322

50. Lourinavicius A, Hurwitz S, Rennke HG. Collapsing glomerulopathy in HIV and non-HIV patients: a clinicopathological and follow-up study. Kidney Int 1999; 56: 2203–2213

51. Sperati J. Stabilization of Hepatitis C associated collapsing glomerulopathy in HIV and non-HIV patients: a clinicopathological and follow-up study. Kidney Int 1999; 56: 2203–2213

52. Carter JJ, Madeleine MM, Wipf GC et al. Lack of serologic evidence for prevalent simian virus 40 infection in humans. J Natl Cancer Inst 2003; 95: 1522–1530

53. Martini F, Corallini A, Balatti V et al. Simian virus 40 in humans. Infect Agent Cancer 2007; 2: 13

54. Galdenzi G, Lupo A, Anglani F et al. Is the simian virus SV40 associated with idiopathic focal segmental glomerulosclerosis in humans? J Nephrol 2003; 16: 350–356

55. Swaminathan S, Lager DJ, Qian X et al. Molecular identification of SV40 infection in human subjects and possible association with kidney disease. J Am Soc Nephrol 2002; 13: 2320–2330

56. MiIstone A, Vilchez RA, Geiger X et al. Polyomavirus simian virus 40 infection associated with nephropathy in a lung-transplant recipient. Transplantation 2004; 77: 1019–1024

Received for publication: 26.12.12; Accepted in revised form: 2.1.13