

A kidney biopsy report is one of the most medically technical documents most patients will ever receive. Written for pathologists and nephrologists, it contains terminology that can be deeply frightening when encountered without context - words like 'fibrosis', 'sclerosis', 'necrosis', and 'obsolescence' carry menacing connotations even when they describe findings whose clinical significance varies greatly. This post decodes the most common terms found in kidney biopsy reports and explains what they mean for you.

HOW A KIDNEY BIOPSY IS ANALYSED - THE THREE TECHNIQUES

A kidney biopsy core is examined using three complementary techniques that together build a complete picture of what is happening in the kidney: Light microscopy examines the kidney tissue with standard microscopy using various stains - PAS (Periodic Acid-Schiff), which stains carbohydrate-containing structures pink-magenta; silver stains, which outline the glomerular basement membrane in black; and Masson's trichrome, which stains collagen (fibrosis) blue. Immunofluorescence microscopy uses fluorescent antibodies to identify deposits of immunoglobulins (IgA, IgG, IgM) and complement proteins (C3, C1q) within the kidney tissue - crucial for identifying immune-mediated kidney diseases. Electron microscopy examines the kidney tissue at the ultrastructural level - revealing changes that are invisible under light microscopy, such as the foot process effacement of nephrotic syndrome and the subepithelial deposits of membranous nephropathy.

THE MOST COMMON TERMS IN A BIOPSY REPORT - WHAT THEY MEAN

GLOMERULAR TERMS: Mesangial hypercellularity or mesangial expansion - the mesangium is the supporting tissue between the capillary loops within the glomerulus. Increased mesangial cells or matrix is seen in IgA nephropathy, early diabetic nephropathy, and lupus nephritis. Focal and segmental - 'focal' means affecting some glomeruli (not all), 'segmental' means affecting part of an individual glomerulus (not all of it). This is the defining description of FSGS. 'Diffuse' and 'global' are the opposites - all glomeruli, entire glomerulus. Crescents - a crescent of cells forming in Bowman's space (the space around the glomerular tuft). Crescents indicate active, aggressive glomerular injury and are seen in rapidly progressive glomerulonephritis (RPGN) - the finding that most urgently requires treatment. Even a small proportion of glomeruli showing crescents indicates a medical emergency. Glomerulosclerosis or obsolescence - scarred glomeruli that are no longer functional. A report may state 'X% globally sclerosed glomeruli' - this percentage indicates the proportion of nephrons that have been permanently lost and cannot be recovered. Higher percentages of sclerosis indicate more advanced chronic irreversible damage. Glomerular basement membrane (GBM) - thickening suggests membranous nephropathy;

thinning is a feature of thin basement membrane disease and early Alport syndrome.

TUBULOINTERSTITIAL TERMS: Interstitial fibrosis and tubular atrophy (IFTA) - the degree of fibrosis and tubular damage in the tissue between the glomeruli. IFTA is typically graded as mild (less than 25%), moderate (25-50%), or severe (greater than 50%). IFTA represents chronic, largely irreversible damage and is one of the strongest predictors of long-term kidney function decline and progression to kidney failure. A biopsy showing severe IFTA, even if the underlying active disease is treated, suggests that a significant proportion of kidney function has already been permanently lost. Interstitial inflammation (interstitial nephritis) - inflammatory cells in the interstitium can represent acute tubulointerstitial nephritis (AIN) - often medication-induced and potentially reversible - or chronic inflammation from ongoing immune activation.

VASCULAR TERMS: Arteriosclerosis - hardening and thickening of small renal artery walls, typically from long-standing hypertension or ageing. Arteriolar hyalinosis - accumulation of hyaline material in arteriole walls, characteristic of hypertensive and diabetic vascular damage. These findings indicate vascular injury that may be contributing to kidney function decline independently of glomerular disease.

THE OVERALL INTERPRETATION - ACTIVE vs CHRONIC LESIONS

One of the most important distinctions in a kidney biopsy report is between active lesions (inflammation, cellular crescents, immune deposits - potentially reversible with treatment) and chronic lesions (fibrosis, tubular atrophy, sclerosed glomeruli - permanent and irreversible). A biopsy showing predominantly active lesions with limited chronic change suggests that aggressive treatment may significantly improve or stabilise kidney function. A biopsy showing predominantly chronic changes with limited active inflammation suggests that the opportunity for treatment-mediated recovery has largely passed - though slowing further decline remains the goal. The 'activity index' and 'chronicity index' in lupus nephritis biopsies explicitly quantify these two components.

QUESTIONS TO ASK WHEN RECEIVING YOUR BIOPSY RESULTS

Ask your nephrologist when discussing your biopsy results: What proportion of my glomeruli are scarred and non-functional? What is the degree of interstitial fibrosis? Are there active inflammatory lesions that are potentially reversible with treatment? Does the biopsy confirm the clinical diagnosis, or does it suggest a different or additional diagnosis? What does this biopsy mean for my prognosis and my response to treatment?

💬 Have you received a kidney biopsy report - and were you given a clear explanation of what it meant for your specific situation? Share below. The gap between what is written in a pathology report and what patients understand about it is one of the most significant communication failures in nephrology. ❤️

📖 RESOURCES

- > Kidney biopsy results explained: [kidneyresearchuk.org](https://www.kidneyresearchuk.org) (search 'kidney biopsy results')
- > Understanding IgA nephropathy biopsy: [kidney.org](https://www.kidney.org) (search 'IgA nephropathy Oxford classification')
- > Kidney pathology guide for patients: niddk.nih.gov