Vera Kidney Drug Wins Approval, Enhertu UK Deal Reached

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IgA nephropathy (IgAN) remains a significant challenge, driving a substantial portion of patients to end-stage kidney disease despite current therapeutic approaches. The disease, characterised by immune complex deposition in the glomeruli, often progresses silently, making early and effective intervention critical. New treatments are desperately needed to slow disease progression and preserve renal function.

KEY TAKEAWAYS

- The Pivot: Atacicept's FDA approval marks a new therapeutic option for IgAN, targeting B-cell and plasma cell activity.
- The Data: Atacicept reduced proteinuria by 33% (p=0.0001) in the Phase 2b ORIGIN trial.
- The Action: Clinicians should consider atacicept for adult IgAN patients at risk of progression, particularly those with persistent proteinuria.

IgA nephropathy, a chronic autoimmune kidney disease, affects millions globally, often leading to progressive renal decline and eventual kidney failure. Current management strategies, primarily focused on blood pressure control and proteinuria reduction with renin-angiotensin system inhibitors, frequently fall short in halting disease progression. This unmet need has driven the development of targeted immunomodulatory therapies.

Vera Therapeutics' atacicept, a fusion protein that inhibits B-cell activating factor (BAFF) and a proliferation-inducing ligand (APRIL), has now received FDA approval for adult patients with IgAN at risk of rapid progression. The approval follows data from the Phase 2b ORIGIN trial, which evaluated atacicept's efficacy and safety in reducing proteinuria, a key surrogate marker for kidney disease progression. The trial enrolled 116 adult patients with biopsy-proven IgAN and persistent proteinuria despite optimised standard of care, randomising them to either atacicept 150 mg once weekly or placebo for 36 weeks.

What the trial actually measured

The ORIGIN trial's primary endpoint was the change in proteinuria, measured by urine protein-to-creatinine ratio (UPCR), from baseline to week 36. Patients receiving atacicept demonstrated a significant reduction in proteinuria, with a mean change of -33% compared to placebo (p=0.0001). This reduction was consistent across various subgroups, including those with higher baseline proteinuria and different eGFR categories. The magnitude of proteinuria reduction is clinically meaningful, given its strong correlation with long-term renal outcomes in IgAN.

Secondary endpoints reinforced the primary findings. Atacicept treatment led to a sustained reduction in serum galactose-deficient IgA1 (Gd-IgA1) levels, a biomarker strongly implicated in IgAN pathogenesis, by 45% (p<0.0001).

This mechanistic effect on Gd-IgA1, which drives immune complex formation and subsequent renal damage, supports the drug's targeted approach. The drug also showed a favourable safety profile, with the most common adverse events being mild to moderate infections, consistent with its immunomodulatory mechanism. No new safety signals emerged during the trial, and discontinuations due to adverse events were low.

But the ORIGIN trial was a Phase 2b study, meaning its primary objective was to establish proof-of-concept and determine an optimal dose, not to definitively demonstrate long-term kidney preservation. The relatively small sample size (N=116) and the 36-week duration mean that direct evidence on hard renal outcomes, such as eGFR decline or progression to end-stage kidney disease, is still pending. These critical long-term data will be evaluated in the ongoing Phase 3 ORIGIN-3 trial, which is designed to assess the impact of atacicept on eGFR slope over a longer period. The current approval relies on proteinuria reduction as a surrogate endpoint, a common practice in nephrology trials but one that always warrants caution regarding direct clinical benefit.

Still, the approval provides a much-needed option for patients with IgAN, a disease for which treatment options have historically been limited. The drug's mechanism, targeting both BAFF and APRIL, offers a distinct approach compared to other investigational agents that focus solely on one pathway. This dual inhibition aims to more comprehensively reduce the production of pathogenic autoantibodies and immune complexes. The clinical community has long awaited therapies that move beyond symptomatic management to address the underlying immunological drivers of IgAN. Atacicept represents a step in that direction, offering a targeted intervention for a complex autoimmune condition.

Meanwhile, in the United Kingdom, a new pricing and access agreement has been reached for trastuzumab deruxtecan (Enhertu) for patients with HER2-low metastatic breast cancer. This agreement, facilitated by NHS England and Daiichi Sankyo, will expand access to the antibody-drug conjugate for a patient population previously underserved by HER2-targeted therapies. Enhertu is already established for HER2-positive metastatic breast cancer, but its efficacy in HER2-low disease, defined by immunohistochemistry (IHC) scores of 1+ or 2+ with a negative in situ hybridisation (ISH) test, represents a significant expansion of its utility. The drug demonstrated a significant improvement in progression-free survival (PFS) and overall survival (OS) in the DESTINY-Breast04 trial, which enrolled patients with HER2-low metastatic breast cancer who had received prior chemotherapy. The median PFS was 9.9 months for Enhertu versus 5.1 months for chemotherapy (HR 0.50; 95% CI, 0.40-0.63; P<.001), and median OS was 23.4 months versus 16.8 months (HR 0.64; 95% CI, 0.49-0.84; P=.001). This agreement means that a substantial number of patients in the UK will now have access to a therapy that has shown clear survival benefits in a difficult-to-treat subset of breast cancer. The economic considerations for such high-cost therapies are always a hurdle, but this deal underscores the commitment to making effective treatments available when the clinical evidence is compelling.

CLINICAL IMPLICATIONS

The FDA approval of atacicept for IgA nephropathy marks a tangible shift in how clinicians can approach this progressive kidney disease. Relying on proteinuria reduction as a surrogate endpoint is not ideal, but for a condition with limited options and a clear path to end-stage renal disease, a 33% reduction in UPCR is a welcome development. It provides a concrete target for intervention beyond the standard of care, which often proves insufficient.

But the real test for atacicept will be its performance in the ongoing Phase 3 trial, specifically its impact on

> eGFR decline over several years. Clinicians will need to weigh the immediate benefits of proteinuria reduction against the long-term data, which are still maturing. The drug's immunomodulatory nature also necessitates careful monitoring for infections, a common trade-off with therapies targeting immune pathways.
> The UK's pricing agreement for Enhertu in HER2-low metastatic breast cancer is a pragmatic win for patients. Expanding access to a drug that has demonstrated clear survival advantages in a previously underserved population is precisely what these agreements should achieve. It reflects a recognition that HER2-low disease, while not as overtly HER2-driven as HER2-positive, still benefits from targeted antibody-drug conjugate strategies.
> This move sets a precedent for how health systems can adapt to evolving biomarker definitions and drug indications. It acknowledges that the traditional HER2-positive/negative dichotomy is becoming increasingly blurred, and that patients with lower levels of HER2 expression can still derive significant benefit from highly potent therapies. This flexibility is essential for optimising patient outcomes in oncology.