How precision medicine treats an overlooked clinical variable: Time



Oncology

In oncology, time is not simply a measure of care; it is a clinical variable – and increasingly a therapeutic challenge. Every delay between diagnosis and effective treatment can carry consequences.

The stark results from a recent *JAMA Surgery* analysis underline this reality: time from diagnosis to first-course treatment increased across six major cancers between 2012 and 2023, rising from 34 to 45 days in breast cancer alone.

As treatment pathways grow more complex, the imperative to shorten that interval is obvious. But the objective is not simply to move a patient to *a* treatment faster. It is to shorten the distance to the treatment most likely to work.

That is where precision oncology can change the equation. By characterising the biology driving an individual tumour more deeply, it can narrow therapeutic choices earlier, reduce time spent on ineffective treatments and, ultimately, make the first treatment more likely to be the effective one.

Early treatment decisions demand greater precision

A failed first treatment costs more than time. It can mean continued disease progression, unnecessary toxicity, and another round of treatment decisions. In metastatic colorectal cancer, treatment can become a succession of these decisions: in one study, 62.4% of patients received a second line, 41.9% a third, and nearly one in four (23.7%) a fourth.

The goal of precision oncology is to move the most informed treatment decision as early in that sequence as possible. If we can identify what is driving a tumour before treatment begins, we can prioritise therapies more closely aligned with its biology, rather than spending valuable time on treatments that are less likely to work.

That requires moving beyond simply cataloguing mutations or establishing whether a target is present. By measuring proteins and their activation states, we can develop a more functional view of the biology driving a tumour and use that information to refine treatment selection.

At the system level, moving toward more biologically informed treatment selection earlier in the care pathway could reduce this cumulative burden and help make increasingly complex oncology care more efficient, not simply faster.

Matching therapeutic intensity to tumour biology

Choosing a cancer treatment is no longer simply a matter of identifying a mutation or finding a target. It is increasingly a question of how the tumour is functioning – and which treatment is most likely to disrupt the biology driving it.

Diseases once treated as broad diagnostic categories are now understood as molecularly distinct tumours, shaped by different genetic alterations, proteins, and signalling pathways. Breast cancer illustrates that progression: from identifying genetic drivers such as BRCA, to recognising proteins such as HER2 as therapeutic targets, to examining the broader networks of proteins and signalling pathways that govern tumour behaviour.

At a higher level, knowing that a particular marker or target is present does not necessarily tell us whether the biology it represents is active or whether a therapy directed at it is likely to benefit the patient.

HER2 provides a useful example. The amount of HER2 present does not necessarily correspond to how strongly it is activated. A relatively small amount can be highly active and drive downstream signalling, while a greater amount may be comparatively inactive. In this way, the biology behaves less like an on/off switch and more like a dimmer.

Proteomics, and particularly phosphoproteomics, can make that biology more visible and clinically actionable. By examining proteins and phosphorylation, these approaches can provide information about pathway activation and signalling activity, complementing genomic testing and immunohistochemistry.

The implication for treatment selection is significant: rather than asking only whether a potential target is present, clinicians can begin to ask what is functionally driving the tumour and at what level. That matters not only for avoiding the wrong treatment, but potentially the wrong *intensity* of treatment: too little may fail to address the disease, while too much can expose patients to unnecessary toxicity and avoidable harm.

Precision medicine's next frontier is prediction

The longer-term opportunity is to move from describing tumour biology to predicting treatment response. As clinical data increasingly connects protein signatures with therapies and outcomes, these datasets could eventually support machine-learning models that identify which treatments are most likely to work for a given tumour.

But that future requires evidence first: rigorous clinical studies showing that specific protein biomarkers can predict response and improve outcomes. Large trials and collaboration with clinical experts will be critical to establishing where these biomarkers can meaningfully inform care.

Access matters, too. Proteomic and phosphoproteomic testing is not yet standard of care and remains more common in academic and research settings than in community oncology. The challenge is making this information accessible and actionable – not replacing existing genomic and pathology testing, but adding another layer to help improve a treatment decision.

Ultimately, oncology care should not only be measured by how quickly we diagnose cancer or initiate treatment, but by how quickly we can characterise the biology driving an individual tumour and translate that biological insight into clinical action.

About the author



Dr Brian Corgiat is senior clinical research scientist at Ignite Proteomics, with extensive experience in clinical research environments. He has contributed to the development of a CAP/CLIA-accredited breast cancer clinical assay and has been involved in regulatory submissions supporting clinical trial assays. He has managed more than 20 clinical research contracts and provided detailed analysis of proteomic results across those studies.