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Blood level of neurofilament light chain as a biomarker for neurological disorders

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Cite this as: *BMJMED* 2025;4:e000958. doi:10.1136/bmjmed-2024-000958

Received: 30 April 2024
Accepted: 11 December 2024

Blood based assay for neurofilament light chain could be a valuable clinical tool for a range of neurological disorders, argue Martin Turner and colleagues, but a clear understanding of the context for optimal use and current limitations is essential

Introduction

Neurofilaments are proteins that form the key scaffolding components of nerves. Raised levels in the cerebrospinal fluid have been noted in response to neuronal injury from the full range of upstream mechanisms. Neurofilaments are heterogeneous but neurofilament light chain and phosphorylated heavy chain have emerged as the leading analytes. Advances in assay technology over the past decade have allowed neurofilaments, particularly neurofilament light chain, to be reliably detected in blood, with increasing sensitivity.[1] Consequently, most applications have focused on neurofilament light chain. Levels of neurofilament light chain in blood and cerebrospinal fluid have a close relation with many disease states,[2] but less so in health.[3] In pure peripheral nerve disorders, raised blood levels of neurofilament light chain can occur in the absence of increased levels in cerebrospinal fluid. While neurofilaments have mostly been used in the context of research to date, new technological developments mean that, with some caveats, there is greater potential for use in the clinical setting.

Although neurofilament release and detection consistently reflect loss of neuronal integrity, the level in progressive disorders seems to indicate the rate of pathological progression (disease aggressivity) rather than the absolute level of neuronal damage or loss. This effect means that rapidly progressive neurological disorders (eg, many patients with amyotrophic lateral sclerosis) will have a high level whereas conditions with a natural history evolving over several years (eg, Alzheimer's disease and Parkinson's disease) will typically have a correspondingly more modest increase in levels of neurofilaments[4] (figure 1).

A comparison has been drawn between increased levels of neurofilaments as a non-specific marker of pathology of the nervous system and the use of C reactive protein as an indicator of systemic inflammation.[5] Major confounds exist in the measurement of neurofilaments at one time point, however, particularly in relation to increasing age, with a substantial increase in levels in those aged >70 years.[6 7] This finding likely reflects the increasing occurrence of subclinical cerebral pathology,[8] but might also relate to structural pathology, such as cervical spinal spondylotic disease or reduced turnover of cerebrospinal fluid.[9] Thus the population in which neurofilaments could offer an attractive screening tool for occult neurological disease might also have a higher occurrence of false positives results. Conversely, low levels of neurofilaments might have substantial rule-out value, but low levels are also associated with slowly progressive disorders and hence false negative conclusions, which might necessitate carefully designed cut-off points. In common with all medical investigations, the clinical context is critical to understanding its value. We now consider specific conditions in relation to neurofilaments.

KEY MESSAGES

- ⇒ Raised blood levels of neurofilament light chain are found in a range of conditions associated with neurological injury
- ⇒ Use of neurofilament light chain is highly dependent on the clinical context, with low independent diagnostic specificity and limited sensitivity to slowly progressive neuropathology
- ⇒ In multiple sclerosis and amyotrophic lateral sclerosis, neurofilament light chain measurement could have an independent role in categorising and evaluating therapeutic response for drug development
- ⇒ For the primary dementias, measuring levels of neurofilament light chain might have diagnostic and prognostic value as part of a wider panel of evaluations
- ⇒ In primary care, the positive predictive value of high neurofilament levels for the earlier detection of subclinical, progressive central nervous system pathology warrants study

Neurofilaments in acute diseases

Stroke

After acute neuronal injury, levels of neurofilaments are raised in a range of acute cerebrovascular events, including aneurysmal subarachnoid haemorrhage[10] and arterial dissection.[11] The initial detection and decision to treat thrombotic stroke depends on the medical history of the patient and physical signs, supported by neuroimaging to exclude major haemorrhage or an alternative pathology. Evidence might exist for neurofilament measurement in predicting stroke, but currently the level of neurofilaments in an individual does not have a role in diagnostic or acute management decisions.

Neurofilaments in serum also increase after hypoxic-ischaemic brain injury in the days after cardiac arrest. Increased levels of neurofilaments are closely related to long term outcome at time points from 24 hours from the event, and outperform clinical measures and CT scanning in predicting poor outcome.[12] Neurofilament assays that are widely

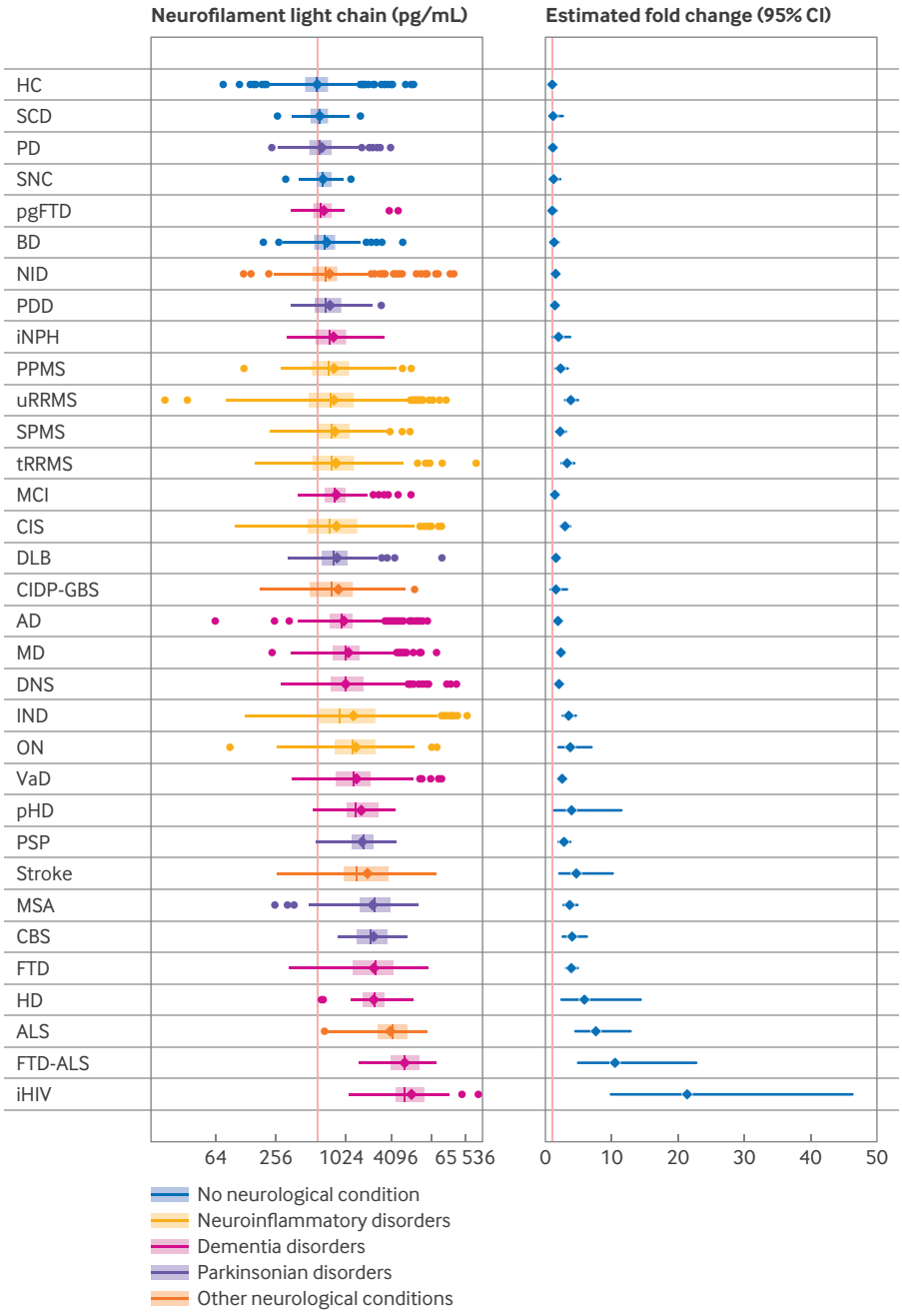

Figure 1 | Levels of neurofilament light chain in cerebrospinal fluid, corrected for age and sex (left), and estimated fold changes compared with healthy controls (right). Values are median (interquartile range). AD= Alzheimer's disease; ALS=amyotrophic lateral sclerosis; BD=bipolar disorder; CBS=corticobasal syndrome; CIDP-GBS=chronic inflammatory demyelinating polyradiculopathy and Guillain-Barré syndrome; CIS=clinically isolated syndrome; DLB=dementia with Lewy bodies; DNS=dementia not specified; FTD=frontotemporal dementia; FTD-ALS=combined frontotemporal dementia and amyotrophic lateral sclerosis; HC=healthy controls; HD=Huntington's disease; iHIV=HIV positive with cognitive impairment; IND=inflammatory neurological disorders other than multiple sclerosis; iNPH=idiopathic normal pressure hydrocephalus; MCI=mild cognitive impairment; MD=mixed dementia; MSA=multiple system atrophy; NID=non-inflammatory neurological disorders; ON=optic neuritis; PD=Parkinson's disease; PDD=Parkinson's disease dementia; pgFTD=presymptomatic genetic frontotemporal dementia; pHD=premanifest Huntington's disease; PPMS=primary progressive multiple sclerosis; PSP=progressive supranuclear palsy; SCD=subjective cognitive decline; SNC=subjective neurological complaint; SPMS=secondary progressive multiple sclerosis; tRRMS=treated relapsing-remitting multiple sclerosis; uRRMS=untreated relapsing-remitting multiple sclerosis; VaD=vascular dementia. Reproduced with permission from Bridel et al[7]

available with a rapid turnaround on random access instruments (ie, not requiring batching), and development of other point-of-care testing modalities, are essential for cost effective use of

neurofilaments in this context, given the necessary short time scale for decision making.

Traumatic brain injury

Neurofilament levels consistently increase, sometimes to very high levels, after traumatic brain injury.[13] Levels have been linked to outcomes, and establishing that neurofilament levels have returned to normal in the months after trauma might also have intuitive value.[14] A more complex dilemma in clinical practice is understanding why some individuals with apparently milder traumatic brain injury develop disproportionately prolonged symptoms, often cognitive and behavioural, included in the broader term, post-concussive syndrome. Establishing normal neurofilament levels in this setting, recognising the importance of timing of the measurements after injury, might give patients and clinicians more confidence in focusing on more holistic, practical support measures, such as aerobic training.

Multiple sclerosis

The diagnosis of multiple sclerosis is largely based on clinical history in combination with findings on magnetic resonance imaging, measurement of antibodies in cerebrospinal fluid by oligoclonal bands or kappa free light chains, and consideration of specific antibody mediated syndromes (eg, aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG)). Although neurofilaments currently lack any useful specificity in early differential diagnosis, levels of neurofilament light chain in cerebrospinal fluid have been repeatedly shown to reflect disease activity as well as prognosis in those diagnosed with multiple sclerosis.[15 16] Levels were reduced in response to disease modifying treatments.[17] Thus sharp increases during acute inflammatory episodes gives information on newly developed lesions (developing within the past six weeks)[18] and has prognostic value for long term (10 years) disability and atrophy, especially when measured during relapses.[19] Reduced levels within 6-12 months after the start of treatment indicate treatment efficacy.[1] The development of blood based assays has greatly improved the usefulness of neurofilament measurement, and it has become a routine biomarker in the management of patients with multiple sclerosis receiving treatment in many specialised clinics.

Encephalitis

Neuronal damage associated with infectious and autoimmune forms of encephalitis has been associated with an increase in levels of neurofilament light chain.[20 21] A role in clinical prognostication, however, is not established, and cytological and microbiological analysis of cerebrospinal fluid, with antibody assays, remain the basis of investigations for diagnosis and management.

Neurofilaments in chronic progressive diseases

Dementia

From postmortem pathological and fluid biomarker studies, the characteristics of dementia and other neurodegenerative disorders are present many years before the onset of symptoms. In Alzheimer's disease, a consequence of the emergence of treatments based on amyloid clearance is the need to give these treatments at the earliest stage of pathogenesis. Thus identifying patients with mild cognitive impairment who will progress to Alzheimer's disease dementia will become even more crucial. The diagnostic sensitivity and specificity of neurofilament light chain has so far proved less than that of amyloid β and τ measurements. In terms of its independent potential for the triage of those presenting to primary care with memory impairment, neurofilament light chain does not currently outperform detailed clinical evaluation,[22] but it has prognostic value in individuals with symptoms.[23]

More broadly, neurofilaments might have an important application in primary psychiatric disorders versus neurological disorders with psychiatric features, such as frontotemporal dementia[24] or Creutzfeldt-Jakob disease, with substantially increased levels prompting consideration of both primary brain parenchymal disease and adverse effects of psychiatric treatment. Dedicated studies in these populations are ongoing.

Emerging uses for neurofilaments in other neurological diseases

Other neurodegenerative disorders

In secondary care settings, levels of neurofilament light chain in blood differentiated between idiopathic Parkinson's disease and atypical syndromes (multiple system atrophy, progressive supranuclear palsy, and corticobasal degeneration), with corticobasal degeneration showing markedly higher levels (area under the curve >0.9).[25] The increasing sensitivity of assay platforms has resulted in blood levels of neurofilament light chain being used to predict the onset of Huntington's disease.[26] In Huntington's disease and other movement disorders associated with relatively lower levels of neurofilament light chain, another use could be as secondary outcome measures in therapeutic trials, including early warning of worsening pathology from sustained increased levels of neurofilament light chain. In fact, this finding was part of the decision to stop the trial of treatment with the experimental antisense oligonucleotide, tominersen, in Huntington's disease.[27]

Neurofilaments have particular value in amyotrophic lateral sclerosis, the most common form of motor neuron disease. In several large cohorts, blood levels of neurofilament light chain were strongly correlated with the rate of progression of disability, although within the limitations of the amyotrophic lateral sclerosis functional rating score. Consistent

Table 1 | Summary of current potential of measurement of blood levels of neurofilament light chain in specific neurological disorders

Neurological disorder	Diagnostic	Prognosis	Treatment outcome measure
Stroke	–	+	–
Traumatic brain injury	+	+	–
Alzheimer's disease	–	–	–
Atypical parkinsonian disorders	+	–	–
Motor neuron disease	–	+	+
Multiple sclerosis	–	+	+
Encephalitis	–	–	–
Guillain-Barré syndrome	–	+	–
Epilepsy	–	+	–
Spinal muscular atrophy	–	+	+

with the clinical observation that the rate of progression of disability in an individual with amyotrophic lateral sclerosis tends to be fixed throughout the course of the disease, levels of neurofilament light chain in individuals remain markedly stable in longitudinal studies.[28 29] Neurofilament light chain has a very limited role in the diagnosis of amyotrophic lateral sclerosis or its exclusion.[30]

Some of the earliest evidence for neurofilament light chain as a pharmacodynamic biomarker first emerged in relation to viral infection. Infection with HIV was associated with a rapid increase in levels of neurofilament light chain in cerebrospinal fluid, particularly in those with associated cognitive dysfunction, which decreased to normal levels within a few months of treatment with antiretroviral agents.[31] The predictable natural history of amyotrophic lateral sclerosis has resulted in neurofilament light chain becoming a potential pharmacodynamic biomarker.[32] In the pivotal trial of the antisense oligonucleotide, tofersen, for superoxide dismutase 1 (*SOD1*) related amyotrophic lateral sclerosis, blood levels of neurofilament light chain decreased in a similar way and time scale, crucially many months before disease slowing was evident, defined by clinical measures.[33] Neurofilament light chain was considered a "reasonably likely surrogate marker of clinical benefit" by the US Food and Drug Administration in relation to provisional licencing. Furthermore, reports that levels of neurofilament light chain begin to show a sustained increase in the months before the onset of symptoms in carriers of high risk genetic causes of amyotrophic lateral sclerosis led to the first preventive trial (ATLAS, A Study of BIIB067 (Tofersen) Initiated in Clinically Presymptomatic Adults With a Confirmed Superoxide Dismutase 1 Mutation). In asymptomatic *SOD1* pathological variant carriers, levels of neurofilament light chain were measured at regular intervals, and patients were then allocated randomly to receive tofersen or placebo when levels increased to a predetermined value.[34] In the much larger population of unpredictable, apparently sporadic forms of amyotrophic lateral sclerosis, the lack of positive outcome in drug trials over the past 30 years has led to interest in the use of blood levels of neurofilament light chain as a primary outcome measure in small group studies to screen drug candidates and potentially de-risk the cost of definitive phase 3 trials (www.experts-als.uk/).

Peripheral nerve disorders

Raised levels of neurofilaments in blood have been reported consistently for a range of hereditary and acquired peripheral nerve disorders. The use of neurofilament light chain in the triage of those presenting with non-specific, length dependent symptoms has yet to be shown but would be potentially appealing in primary care settings. Neurofilament light chain has value in the prognostication of an established diagnosis of Guillain-Barré syndrome,[35] and so might guide intervention and the anticipation of intensive care needs.

Epilepsy

Levels of neurofilaments in blood and cerebrospinal fluid are only slightly, if at all, increased in people with epilepsy. An exception is status epilepticus, which could be associated with particularly high levels in convulsive and non-convulsive settings[36] and might inform outcomes.[37] A potential role in differentiating dissociative from epileptic seizures has not yet been established.

Paediatric neurological disorders

Neurofilament light chain could be used as a prognostic and monitoring tool in neurological disorders occurring from birth. Levels decrease in response to genetic treatment in spinal muscular atrophy,[38] metachromatic leukodystrophy,[39] and paediatric multiple sclerosis. Levels in healthy young children are high, at a similar level as healthy individuals aged >75 years,[40] declining until adolescence before rising again. Therefore, interpretation for diagnosis of disease induced axonal degeneration requires comparison with age specific reference values.[38] Table 1 summarises the current potential use of

blood levels of neurofilament light chain in a range of neurological disorders.

Future clinical implications

Ease of sampling, increasing availability, reproducibility, and turnaround time of a blood neurofilament assay is generating interest as a clinical tool for a range of neurological disorders. Several large commercial manufacturers, who have been working in this area for several years, are expected to make point-of-care testing available in the near future. A neurofilament light chain assay lacks sensitivity as well as specificity for use on its own. In secondary care settings for those with an established neurological diagnosis, however, measuring neurofilament levels could guide the use of existing treatments and help develop new treatments more rapidly. In primary care, for those patients presenting with neurological symptoms, measuring levels of neurofilament light chain, particularly in the form of a rapid turnaround test, warrants focused study for its potential to improve triage of patients. Measuring levels of neurofilament light chain might provide information by increasing the index of suspicion for active pathology, leading to rule-in if not rule-out use.

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Contributors MRT conceived the article and is professor of clinical neurology and neuroscience, with expertise in biomarker development for amyotrophic lateral sclerosis, including some of the pivotal longitudinal cohort analyses with neurofilament light chain. AGT is a neurologist and associate professor with expertise in biomarker development for amyotrophic lateral sclerosis, including the modelling of neurofilament light chain reduction as a potential trial outcome measure. CET is professor of neurochemistry and international opinion leader on biomarkers for dementias and other neurological disorders, with particular interest in neurofilament light chain assay development. All authors contributed to the manuscript writing. MRT is the guarantor.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests We have read and understood the BMJ policy on declaration of interests and declare the following interests: none.

Patient involvement Patients and the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Provenance and peer review Commissioned; externally peer reviewed.

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