Precision imaging in chronic liver disease management

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Abstract

Metabolic dysfunction-associated fatty liver disease (MAFLD) affects over a quarter of the global population, with up to 30% developing Metabolic Dysfunction-Associated Steatohepatitis (MASH), a progressive form that can silently lead to fibrosis, cirrhosis, and liver cancer. Current diagnostic methods, including blood-based scores and imaging, are insufficient for early detection, leading to late-stage diagnoses in most patients. Liver biopsy remains the diagnostic gold standard but is invasive, costly, and prone to high inter- and intra-reader variability, limiting its utility in routine care and clinical trials. Our research highlights myosteatosis—fat infiltration in skeletal muscle—as a potential early, non-invasive marker of MASH. In preclinical models and clinical studies, myosteatosis correlated with the presence of MASH and distinguished it from isolated steatosis. Notably, reductions in myosteatosis following interventions such as bariatric surgery or dietary regimens were associated with histological improvements in MASH, suggesting a potential role in predicting treatment response. In larger cohorts, myosteatosis was identified as a strong predictor of all-cause mortality. In parallel, we utilized a VCAM-1-targeted molecular imaging technique and demonstrated a high accuracy in detecting inflammation in preclinical MASH models. This technology is now advancing to clinical trials for validation in humans. Taken together, our data support that targeted medical imaging may enable early, non-invasive diagnosis and monitoring of MASH, reducing reliance on liver biopsy and improving patient outcomes.(Acta gastroenterol belg., **2025**, 88, **61-66**).

Keywords: Infliximab, CT-P13, Subcutaneous therapy, Crohn's disease, Ulcerative colitis.

Main

Metabolic dysfunction-associated fatty liver disease (MAFLD), a condition typified by excess fat accumulation in the liver, often due to unhealthy lifestyle such as high-fat, high-sugar diets and inactivity, affects more than a quarter of the global population, hence over 2 billion people worldwide (1–4). Most individuals with MAFLD have liver steatosis with no or mild inflammation. However, up to 30% of them develop a progressive, inflammatory form of the disease, known as Metabolic Dysfunction-Associated Steatohepatitis (MASH) (1–4). MASH is a typically silent condition that eventually leads to liver fibrosis, cirrhosis, increased susceptibility to liver and other organ cancers, and liver transplantation (1–3).

At present, there is no reliable biomarker to diagnose and monitor MASH (5). Liver enzyme levels are poorly sensitive (6). As a result, most patients remain undiagnosed until the disease progresses to advanced, often terminal, stages. Current imaging technologies hinge on the evaluation of structural liver changes that occur with MAFLD progression. At one end of the spectrum, this includes the quantification of liver fat (using MRI or other tools) (7–9). However, this information is of limited value as the correlation between liver fat content and the risk of developing MASH is weak (10). At the other end of the spectrum, fibrosis-associated liver stiffness can be evaluated with elastography techniques, such as Fibroscan© and Magnetic Resonance Elastography (MRE). These techniques, though, are unreliable in the absence of severe fibrosis – which occurs after years of silent progression (5). Furthermore, liver stiffness is not solely determined by collagen and extracellular matrix (ECM) deposition, but is also influenced by steatosis, inflammation, and hemodynamic changes (11,12).

Liver Biopsy for MASH: Gold Standard or Necessary Compromise?

Liver biopsy remains the gold standard for diagnosing MASH, but is an invasive and painful technique, burdened by cost constraints, high sampling bias (13) (only a few mm^3 of tissue being evaluated), and possible bleeding risks. The disease activity is evaluated with different histological scores, for instance the Steatosis, Activity and Fibrosis (SAF) score (14) or NAFLD Activity Score (NAS) (15), which sum up numerical grades for steatosis, inflammation and ballooning. These semi-quantitative methods have major limitations: they force continuous measures into discrete grading bins, which can obscure improvements or worsening. For example, an inflammation score of 2 per the NAS scoring could represent either 2 or 4 inflammatory foci per field – essentially a doubled inflammatory load difference. This lack of granularity is even more pronounced for ballooning, where the scoring is effectively limited to "none," "few," or "prominent". This grading system leads to high inter- and intra-reader variability (16,17). The concordance between observers can be as low as 0.3 for inflammation (16). Yet, at present, none of the existing biomarkers offer a viable alternative to liver biopsy for the evaluation of MASH (5).

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Submission date: 07/01/2025
Acceptance date: 02/02/2025

Challenges in Clinical Trials

One of the most compelling indicators of the poor performance of current biomarkers is the overwhelming screen failure rate in MASH clinical trials—up to 80% (18,19) — despite their use to enrich and optimize patient selection. Furthermore, biopsy-derived histological scores are the only surrogate endpoints currently recognized by regulatory bodies such as the EMA and FDA (20). Therefore, paired liver biopsies at inclusion as well as post-treatment are necessary to enroll patients and determine treatment efficacy in clinical trials. In the interim, we have no reliable means to distinguish responders from non-responders upstream of the second biopsy (typically after 52 weeks). Notwithstanding, clinically-meaningful changes in disease activity may go undetected by semi-quantitative scoring (see above). Such a high level of ambiguity contributes to significant logistical and financial challenges in conducting MASH clinical trials. This squandering of resources could be mitigated with early, non-invasive means of diagnosing MASH and monitoring treatment response.

Myosteatosis: An Overlooked Phenomenon in Metabolic Diseases?

The concept that disrupted "muscle health" may be a sign and/or a pathophysiological contributor to various diseases has gathered support in recent years (21–24). In the context of liver diseases, we have challenged the long-held belief that low muscle mass is the primary musculoskeletal abnormality linked to MAFLD progression (25–27). Our research proposes a shift in focus to myosteatosis, or fat infiltration within skeletal muscles (Figure 1), as an early and independent marker of liver disease severity.

Preclinical Insights: Myosteatosis as an Early Marker of MASH

We longitudinally followed preclinical models of MAFLD and found that muscle mass did not decrease (but rather increased) over the course of MAFLD progression (25). Muscle strength, a critical indicator of sarcopenia (28), only declined in models with severe fibrosis. By contrast, myosteatosis was consistently found in three preclinical MASH models, both in early as well as in fibrosing MASH. In fact, by solely measuring myosteatosis non-invasively using a micro-CT scan, we could differentiate animals with steatohepatitis from those with normal liver or isolated steatosis with a high diagnostic accuracy (AUROC = 0.96, $p < 0.001$) (25). Further preclinical studies are required to establish whether myosteatosis is merely a marker of metabolic dysfunction or whether it plays a causal role in liver disease progression. This finding, however, raised an important question: Could myosteatosis serve as an early indicator of progressive liver disease in humans?

Translational Validation

In a prospective clinical study (27), we performed low-dose CT-scan dedicated to skeletal muscle analysis with an induced effective radiation dose ~1 mSv (millisievert), 10 times lower than that for a standard abdominal CT scan and equivalent to that for a standard abdominal plain film (29,30). CT scan was feasible in all patients, and CT images were used to evaluate muscle mass and myosteatosis in obese patients with MAFLD, before and after a dietary intervention (27). We found that the degree of myosteatosis was strongly associated with liver stiffness at baseline (27). Interestingly, sarcopenia was rare (8.3%) and did not correlate with liver

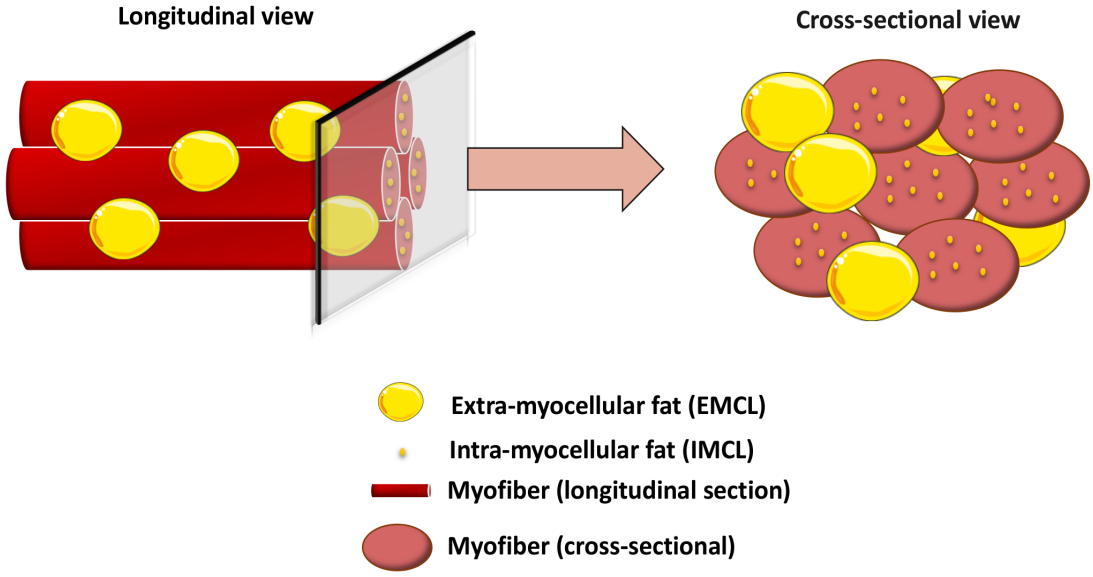

Figure 1. — Myosteatosis encompasses lipids that accumulate extra-myocellularly as adipocytes lying in between myofibers and intra-myocellularly as protein-coated lipid droplet within myofibers cytoplasm.

stiffness, supporting the notion that myosteatosis, not low muscle mass, might be the primary musculoskeletal abnormality associated with liver disease progression. Following a 3-month dietary intervention, patients who exhibited reduced myosteatosis had a significant decrease in liver stiffness, regardless of weight loss or metabolic improvements (27). One key limitation of this study, however, was the absence of liver biopsies - thus we could not discriminate isolated steatosis from (fibrosing) MASH.

To address this shortcoming, we extracted imaging data from patients that underwent a liver biopsy and an abdominal CT-scan at the UZA (Antwerp, Belgium) (26). Here also, muscle mass was higher in patients with MAFLD than in those without (56.8 ± 9.9 vs. 47.4 ± 6.5 cm2/m2, p<0.0001). There was no association between sarcopenia and MASH. However, we found a robust association between myosteatosis and histologically-defined MASH. In fact, among a comprehensive range of anthropometric, metabolic, and serum markers, myosteatosis was the only significantly different parameter between patients with isolated liver steatosis and those with early-MASH (i.e., no or mild fibrosis; F0-1). The association between myosteatosis and MASH remain significant after adjustment for multiple confounders.

Following treatment, including bariatric surgery or dietary interventions, myosteatosis decreased to a greater extent in patients in which MASH improved when compared to those with limited or no therapeutic response. We stratified patients based on whether they experienced myosteatosis reduction greater or less than 11% (Youden's index) following intervention, and assessed the corresponding histological changes. Specifically, we evaluated the proportion of patients who achieved MASH improvement or complete resolution of any histological feature of MASH after intervention. Remarkably, 100% of patients with myosteatosis reduction greater than 11% achieved MASH improvement (14 out of 14), and all patients in this group resolved either steatosis or inflammation (16 out of 16) (Figure 2). Of note, the association between myosteatosis and MASH has since been replicated by independent research groups (31–35,35–37), consolidating the idea that the evaluation of myosteatosis may represent a useful tool to guide the management of patients with MAFLD.

Beyond the liver: Myosteatosis as a Broader Signal of Health Risks

Differentiating MASH from isolated liver steatosis is critical for assessing the risk of liver disease progression; however, extrahepatic complications such as cardiovascular events and increased risk of cancers are of great concern in MASH patients. This invites further exploration into whether myosteatosis might shed light on all-cause mortality - beyond traditional metabolic factors such as diabetes, liver steatosis or (visceral) obesity. To test this hypothesis, we studied a cohort of 8,982 adults who underwent low-dose CT for colorectal cancer screening, followed over a median of 8.8 years, and with 507 deaths that occurred during follow-up. Myosteatosis was present in 55% of those who died (278 of 507), making it the most common abnormal metabolic feature linked to mortality (illustrated in Figure 3). The absolute 10-year mortality risk for patients with myosteatosis was 15.5%, significantly higher than the risk associated with obesity (7.6%), liver steatosis (8.5%), or myopenia (9.7%). Myosteatosis was the strongest predictor of mortality (HR 4.33, 95% CI: 3.63–5.16; p < 0.001), surpassing other body composition markers. Even after adjusting for age, sex, smoking, type 2 diabetes, obesity, liver steatosis, visceral fat, and cardiovascular history, myosteatosis remained an independent risk

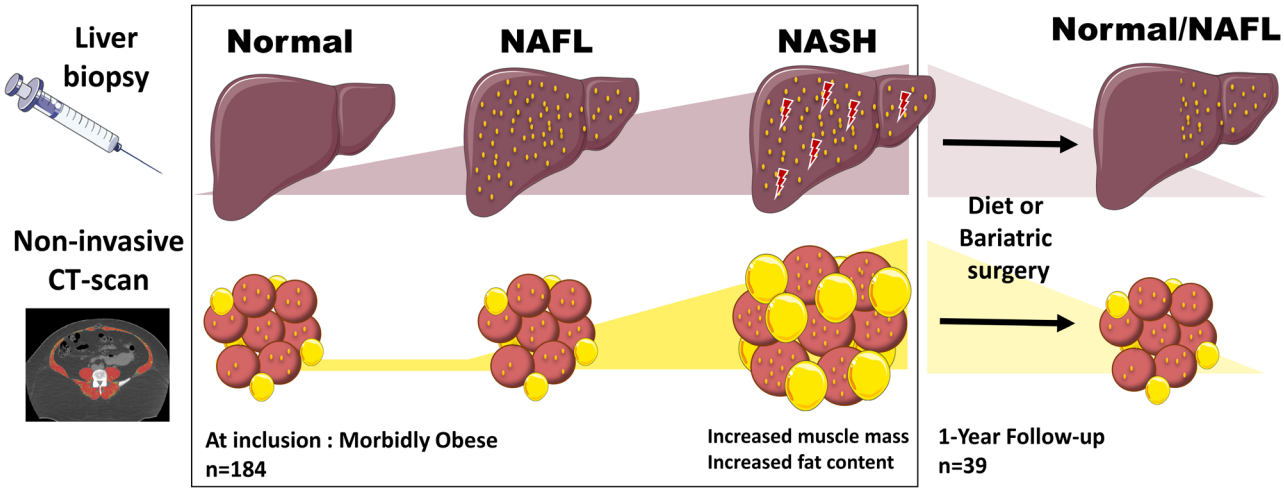

Figure 2. — Higher muscular fat content (myosteatosis) is observed in morbidly obese patients with MASH (NASH) compared to those with a normal liver or simple steatosis (NAFL). Improvement in liver disease—marked by resolution to a normal liver state or simple steatosis—is associated with a concomitant reduction of myosteatosis.

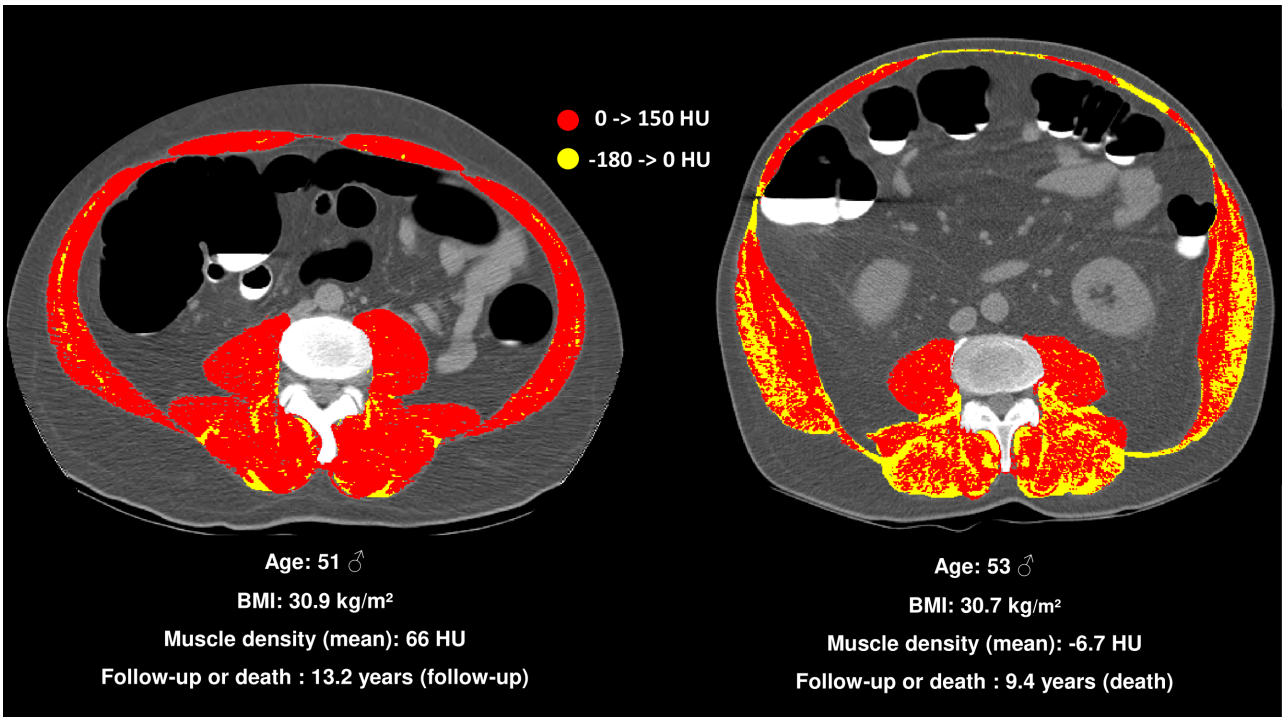

Figure 3. — (A) Abdominal CT image with a Hounsfield unit–based color scale of skeletal muscles in a 51-year-old man with obesity, smoking history, no type 2 diabetes, and no history of cardiovascular events at inclusion shows mild fatty infiltration in the muscles (myosteatosis, yellow), with most voxels in the positive range of Hounsfield units (red). The patient was lost to follow-up after 13.2 years. (B) Abdominal CT image with a Hounsfield unit–based color scale of skeletal muscles in a 53-year-old man with obesity, smoking history, no type 2 diabetes, and no history of cardiovascular events at inclusion shows severe fatty infiltration in the muscles (myosteatosis, yellow), mostly distributed in the paravertebral (ie, erector spinae and multifidus) and oblique muscle groups. The patient died after 9.4 years of follow-up. BMI = body mass index.

factor (HR 1.89, 95% CI: 1.52–2.35; p < 0.001). Further analysis using a multistate Markov model revealed that myosteatosis significantly increased the risk of death following cardiovascular events. In fact, patients with myosteatosis who experienced at least one major adverse cardiovascular event (MACE; i.e. cerebrovascular disease, myocardial infarction, heart failure, or aortic aneurysm) had a 61% higher risk of death (HR 1.61, 95% CI: 1.09–2.37; p = 0.02). This risk escalated further after two cardiovascular events, with a 91% increased mortality risk (HR 1.91, 95% CI: 1.07–3.42; p = 0.03). Notably, myosteatosis was the only body composition parameter that remained significantly associated with mortality at each transition point—from initial MACE to subsequent events and eventual death. These findings suggest that, in addition to being a marker of metabolic dysfunction, myosteatosis is an indicator of vulnerability. Based on available data, we thus speculate that myosteatosis assessment could be a valuable adjunct in the management of patient with suspected or established MASH. However, the causal link has yet to be proven and, if so, the mechanisms behind a muscle-liver axis have yet to be identified.

Advancing Non-Invasive Inflammation Imaging

Our data support that myosteatosis may indicate the presence of MASH, but it does not reflect on the severity of liver inflammation—a key driver of MASH progression (see above). To address this gap, we evaluated the potential of radiolabeled anti-VCAM-1 nanobody (99mTc-cAbVCAM1-5) coupled with SPECT imaging (38), a molecular imaging technique developed in collaboration with VUB (Belgium) and UGA (France). This technology was originally conceived to evaluate inflammatory recruitment in atherosclerotic plaques (39,40), but should be suitable to evaluate hepatic inflammation in MASH. Indeed, VCAM-1 is expressed by liver sinusoidal endothelial cells (LSECs) under the control of pro-inflammatory signals and was deemed to be a key driver of inflammatory cell recruitment in MASH (41). Notwithstanding, given the size of the liver, an excellent signal-to-noise ratio should be achievable. This hypothesis was rigorously tested, leading to a proof-of-concept in five relevant preclinical models (38). Indeed, VCAM-1 expression was strongly correlated with inflammatory markers of MASH, including the amount of F4/80 macrophages (r = 0.97, p<0.0001), histological inflammatory scores (r = 0.81, p<0.0001). At the protein level, VCAM-1 content was significantly higher in MASH livers compared to those with isolated liver steatosis. The 99mTc-cAbVCAM1-5 liver uptake correlated closely with VCAM-1 expression. In both in vivo as well as ex vivo analyses, the SUV (standardized uptake value) was significantly higher in livers of animal with MASH.

The 99mTc-cAbVCAM1-5 signal had a high diagnostic accuracy for detecting liver inflammation, with area under the curves (AUCs) of 0.93 and 0.91 ($p<0.0001$) for identifying any degree of inflammation (score 1–3 vs 0) and moderate-to-severe inflammation (score 2–3 vs 0-1), respectively. Of note, in the context of steatotic hepatomegaly, we hypothesized that SUVLean Liver (SUV corrected for steatosis) and %ID Liver (total liver uptake) could more accurately reflect inflammatory cell recruitment than unadjusted SUV values. Both SUVLean Liver and %IDLiver showed strong correlations with histological inflammatory scores ($r = 0.89$ and $r = 0.82$, respectively, $p < 0.0001$). When inflammation was assessed based on SUVLean Liver or %IDLiver, diagnostic performance was excellent (AUCs 0.86–0.99, $p < 0.05$). AUCs of ≥ 0.95 were consistently achieved for detecting moderate-to-severe inflammation (score 2–3 vs. 0–1), the most clinically-relevant endpoint as defined by current FDA and EMA guidelines. These data provide a strong foundation for clinical translation, with a Phase I/IIa clinical trial set to begin within the next two years.

Conclusion

Targeted medical imaging might lead to a paradigm shift in chronic liver disease management, enabling early detection and monitoring without the need for invasive biopsies. Continued research and clinical validation are essential to solidify their role in improving patient outcomes and advancing therapeutic development.

Abbreviations

BMI – Body Mass Index; CT – Computed Tomography; ECM – Extracellular Matrix; EMA – European Medicines Agency; FDA – Food and Drug Administration; HR – Hazard Ratio; kPa – Kilopascal; LSEC – Liver Sinusoidal Endothelial Cell; MACE – Major Adverse Cardiovascular Event; MAFLD – Metabolic Dysfunction-Associated Fatty Liver Disease; MASH – Metabolic Dysfunction-Associated Steatohepatitis; MRI – Magnetic Resonance Imaging; MRE – Magnetic Resonance Elastography; NAS – NAFLD Activity Score; NAFLD – Non-Alcoholic Fatty Liver Disease; SAF – Steatosis, Activity, and Fibrosis; SPECT – Single Photon Emission Computed Tomography; SUV – Standardized Uptake Value; VCAM-1 – Vascular Cell Adhesion Molecule-1.

Acknowledgements

I would like to express my deepest gratitude to Pr. Isabelle Leclercq (UCLouvain, Belgium) for her supervision and critical intellectual input throughout all aspects of this work. Her guidance, together with the support of Pr. Yves Horsmans (UCLouvain, Belgium), has been pivotal in shaping the research presented. I also extend my sincere thanks to Professor Greetje Van de Velde (KULeuven, Belgium) for her collaboration in conducting the preclinical experiments, which formed the foundation of this series of studies. I am very grateful to Pr. Sven Francque (UZA, Belgium), Dr. Wilhelmus Kwanten (UZA, Belgium), and Pr. Nicolas Lanthier (UCLouvain, Belgium) for their collaboration on the clinical cohorts. Their willingness to provide access to key clinical data and their significant intellectual contributions greatly facilitated the translation of preclinical findings into clinical insights. I would like to thank Pr. Perry Pickhardt (UW-Madison, US) and Pr. Ronald Summers (NIH, US) for their collaboration in evaluating the impact of myosteatosis on mortality in a large U.S cohort. Their support has been instrumental in broadening the scope and impact of this research. Finally, I thank Dr. Alexis Broisat (UGA, France), Dr. Pascale Perret (UGA, France) and Pr. Nick Devoogdt (VUB, Belgium) for their essential contribution with the VCAM-1 studies in mice.

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