

Predicting GIST mitotic index before surgery: Where conventional CT fits in oncology precision?

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Abstract

Risk stratification for gastrointestinal stromal tumor (GIST) depends on postoperative mitotic index (MI), a histologic marker unavailable before surgery to guide neoadjuvant therapy and operative strategy. In 2020, Ren et al. constructed a routine CT nomogram to preoperatively identify localized primary GIST featured with high mitotic activity >5 mitoses per 50 high-power fields (>5/50 HPF), showing strong discriminative power in single-center training and internal validation cohorts. This commentary frames this imaging model within precision oncology paradigms, elaborating its clinical value while unpacking the key flaws of retrospective single-center prediction research. We further analyze the mismatches between macroscopic CT phenotypes and KIT/PDGFRA molecular profiles, specify strict clinical boundaries for the nomogram, and propose necessary translational validation workups. Although CT-based risk scores effectively streamline sarcoma MDT referrals, biopsy scheduling and intraoperative rupture prevention, they cannot substitute for the formal histopathology or mutational testing. Ultimately, visual CT nomograms only serve as auxiliary triage tools instead of main decision evidence in comprehensive GIST clinical management.

Keywords: Gastrointestinal stromal tumor, Mitotic index, Computed tomography, Nomogram, Preoperative risk stratification, Precision oncology, Sarcoma multidisciplinary team

Highlights

1. Conventional CT morphological nomograms are auxiliary triage tools and cannot displace histopathology or KIT/PDGFRA sequencing for definitive GIST treatment decisions.
2. Widespread molecular-morphology mismatches are present in GIST, and exclusive reliance on CT risk scores may lead to inappropriate imatinib and delayed avapritinib administration for PDGFRA exon 18 D842V-mutated tumors.
3. Single-center retrospective design without external validation constrains the generalizability of the CT mitotic index prediction model proposed by Ren and colleagues.
4. Strict clinical eligibility must be followed. The nomogram is only applicable to suspected primary localized GIST to facilitate MDT discussion, biopsy prioritization and surgical planning.

Introduction

Risk-adapted individualized treatment represents the core paradigm for localized gastrointestinal stromal tumor (GIST) clinical management, with all guideline-recommended therapeutic strategies determined by predicted malignant biological behavior of primary lesions [1]. Globally accepted risk stratification frameworks, including modified NIH criteria and AFIP pathological grading systems, depend on two core pathological indicators: maximum tumor diameter and mitotic index (mitotic figures per 50 high-power fields, HPF) [2]. Preoperative cross-sectional CT reliably quantifies tumor

size, anatomical location, and macroscopic morphological features with excellent inter-examination reproducibility. However, definitive mitotic index quantification can only be completed via postoperative histopathological slide counting, creating a critical clinical bottleneck for upfront treatment planning [3].

This unmet clinical challenge creates a practical limitation in routine GIST care. The unconfirmed high mitotic risk prior to surgical resection constrains MDT risk assessment, targeted biopsy scheduling, and surgical technique design for preventing intraoperative tumor rupture. While endoscopic ultrasound (EUS)-guided or percutaneous core needle biopsy is generally safe for selected patients and preoperative tissue confirmation may alter treatment pathways, biopsy carries minor risks of tumor seeding and hemorrhage and small gastric GISTs may be directly managed via endoscopic resection without pre-operative biopsy [4,5]. The non-invasive preoperative imaging biomarkers for assessing mitotic risk would be of tangible clinical value for sarcoma patient triage.

Against this background, Ren and colleagues constructed a conventional CT feature-based nomogram to preoperatively identify patients with high mitotic index, providing a plausible low-cost, widely accessible preoperative risk prediction tool [6,7]. This commentary expands upon this single-center retrospective cohort research, assesses the translational advantage and inherent limitations of visual CT prediction models, compares conventional CT nomograms with emerging radiomics and liquid biopsy platforms, and establishes standardized clinical implementation boundary to guide safe, evidence-based real-world application of this predictive tool in precision GIST oncology.

The Contribution of the Focal Article

The landmark retrospective cohort study by Ren et al. enrolled 250 patients with pathologically confirmed primary localized GIST across a 5-year single-center dataset, randomly split at a 7:3 ratio into training cohort (n=176) and internal validation cohort (n=74) [8]. The univariate and multivariate binary logistic regression screening identified three independent CT imaging predictors of high mitotic index (>5/50 HPF): larger tumor size, intratumoral liquefaction and necrosis, and irregular tumor contour. The visual CT nomogram yielded an AUC of 0.851 in the training set and 0.836 in internal validation, demonstrating strong discriminative ability to distinguish high versus low mitotic risk GISTs using routine unenhanced and contrast-enhanced abdominal CT sequences available in all general radiology departments worldwide.

From surgical and medical oncology perspectives, the primary translational advantage of this model lies in its utilization of standard, qualitative CT morphological features without advanced post-processing, artificial intelligence reconstruction, or high-throughput radiomic feature extraction [9,10]. Each imaging biomarker carries clear biological underpinnings for aggressive mesenchymal tumor proliferation. Rapid tumor cell proliferation outpaces intralesional angiogenesis, leading to tissue hypoxia, liquefactive necrosis, and low-density non-enhancing areas on CT imaging. Irregular, lobulated tumor margins reflect asymmetric, invasive growth patterns, distinct from smooth, well-circumscribed borders observed in indolent, low-mitotic GIST subtypes [11]. In short, the three readily identifiable CT phenotypes enable radiologists to generate a preoperative numerical risk score without specialized software, streamlining rapid clinical risk triage before histology results.

Plausibly, the standard preoperative CT provides indirect, actionable surrogate markers of tumor proliferative kinetics, addressing the critical limitation of absent mitotic index data in pre-surgical patient evaluation [12,13]. However, robust discrimination statistics alone does not sufficiently justify standalone clinical decision-making, which requires layered clinical interpretation to avoid over-reliance on imaging surrogates.

Clinical Interpretation: Useful Signal but Not a Diagnostic Substitute

Despite favorable predictive performance metrics, clinicians have to interpret nomogram risk scores with strict bedside caution, with two clear clinical guardrails to prevent mismanagement.

First, preoperative CT morphological features could serve as quick triage aids rather than substitutes for histopathological mitotic quantification and molecular mutation testing. CT findings alone could hardly warrant neoadjuvant imatinib or other targeted therapies, as guideline-compliant neoadjuvant management requires histological validation plus KIT/PDGFR genotyping to confirm GIST and rule out imatinib-resistant subtypes [14]. Similar to endoscopic risk stratification for early gastric cancer, CT-based scoring is not to replace postoperative pathology to assess lymphovascular invasion and metastatic potential [15].

Second, high nomogram scores prompt urgent sarcoma MDT consultation, refined biopsy schedules and adjusted surgical strategies in prevention of intraoperative tumor rupture, an event that by itself worsens prognosis and elevates disease stage [16,17]. This nomogram mainly functions to triage high-risk patients for specialized sarcoma management, instead of replacing tissue diagnosis.

Overreliance on CT nomograms is prone to notable clinical risks. For example, abdominal mesenchymal mimics (leiomyosarcoma, inflammatory myofibroblastic tumor) with necrotic CT features may falsely yield high mitotic risk scores insensitive to imatinib. For metastatic GIST, these CT models could hardly guide cytoreductive surgery or systemic therapy sequencing, as metastatic management strictly depends on tumor burden and molecular genotype instead of primary lesion imaging surrogates [18].

Methodological Considerations for Prediction-Model Studies

To facilitate clinical adoption of this CT nomogram, we assessed its design per TRIPOD+AI guidelines to highlight critical methodological limitations [19].

Single-center retrospective cohort and internal validation bias

The retrospective single-center design inherently limits generalizability. Internal split-sample validation using uniform local CT protocols tends to overestimate real-world AUC, and multi-center external validation across varied equipment and populations is required to verify stable predictive performance.

Statistical modeling risks of overfitting

Stepwise logistic regression relying on p-values and AIC renders high overfitting risks for moderate cohorts. Standardized reporting of missing data and incomplete follow-up is also absent, a critical transparency shortfall [20].

Incomplete calibration and decision curve evaluation

High AUC does not guarantee clinical value, and practical models rely on validated calibration, inter-rater consistency and net clinical benefit [21]. The Hosmer-Lemeshow test is sample-size dependent and fails to assess full-range risk calibration. Subsequent validations should report complete calibration metrics and DCA to quantify clinical utility [22].

DCA thresholds demand clinical trade-offs: high-risk cutoffs balance detection of aggressive GISTs against unnecessary biopsy and overtreatment [23]. Qualitative CT features like irregular tumor margins are subject to radiologist judgment. Though consistent within this single center, inter-rater Kappa across general radiologists was unreported, restricting external applicability [24,25].

Molecular and Translational Context

When implementing CT nomograms for precision GIST management clinicians should account for tumor genomic drivers. Approximately 85% of sporadic GISTs harbor active KIT/PDGFRα mutations that determine proliferative potential and therapeutic response [26,27]. Mutation subtypes bear a non-linear relationship with mitotic index, and conventional CT morphological features fail to visualize subpixel molecular alterations underlying tumor progression.

Risk stratification relying on CT findings without genotyping frequently leads to misjudgment. Large necrotic gastric GISTs typically generate elevated nomogram risk scores, yet PDGFRα exon 18 D842V-mutated tumors exhibit primary imatinib resistance [28]. Administering neoadjuvant imatinib based solely on CT nomogram results delays avapritinib therapy and compromises patient survival outcomes [29,30]. Imaging biomarkers and molecular pathology serve as complementary, non-interchangeable diagnostic modalities.

While advanced radiomics and radiogenomics can extract subtle imaging phenotypes correlated with KIT/PDGFRα variants, these quantitative imaging techniques remain investigational and lack formal guideline endorsement [31]. Conventional CT nomograms cannot substitute mandatory molecular testing prior to systemic therapy in standard clinical workflows.

Clinical Implementation and Boundaries

This nomogram has strict clinical application limits. It benefits only suspected localized primary GISTs to guide MDT consultation,

biopsy and surgery planning, with its utility determined by safe clinical decision improvement [32].

Ren's cohort mainly consisted of gastric GISTs (76.7% of training set) [8]. Biopsy and neoadjuvant therapy for gastric lesions rely largely on tumor size and location, and low-risk cases merely need endoscopic resection and surveillance [33–35]. By contrast, small rectal or duodenal GISTs often demand high-morbidity radical operations like abdominoperineal or pancreaticoduodenal resection [36,37]. For extragastric GISTs, surgical planning prioritizes anatomy and operative risks instead of CT-predicted mitotic index, so the nomogram only serves as supplementary evidence in individualized MDT decisions [38].

Future directions

Four validation and comparative studies are necessary before routine clinical use of this CT nomogram.

1. It calls for multi-center external validation covering various CT devices, contrast protocols and populations, with standardized reporting of calibration metrics and DCA curves [39]. Similar disparities in imaging access and diagnostic consistency across demographic subgroups have been widely documented in breast cancer care, underscoring the need for population-diversified model validation [40].
2. It needs to integrate CT descriptive standards for necrosis and irregular margins to cut inter-reader variation and boost Kappa reliability in community radiology [41]. As unstandardized intraoperative endocrine assessment brings large observer bias, uniform abdominal tumor CT terminology is essential [42].
3. It requires head-to-head comparisons with radiomic machine learning and cfDNA liquid biopsy assays [43]. Currently, cfDNA molecular testing for localized GIST remains investigational and cannot replace tissue-based genotyping standards [44].
4. It demands for prospective trials to evaluate real-world outcomes, including intraoperative rupture, MDT efficiency, overall survival and cost-effectiveness of nomogram-based risk stratification [45].

For illustrating the auxiliary role of CT nomograms in standardized GIST preoperative management and for educational purpose, we summarize the complete clinical decision-making workflow as shown in **Figure 1**.

GIST Preoperative Risk-Stratified Care Pathway: CT Nomogram as an Aid

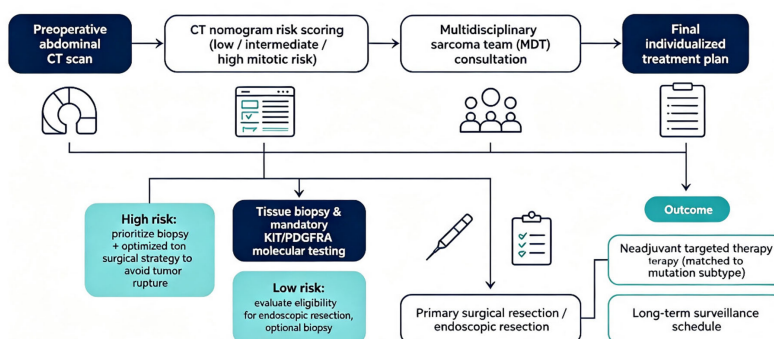


Figure 1.

Conclusions

Ren's CT nomogram is an affordable preoperative triage tool for localized primary GIST, using three routine CT features (large size, intratumoral necrosis, irregular margins) to predict high mitotic risk. It shows plausible discrimination in single-center internal validation and offers biological surrogates to optimize surgery and sarcoma MDT scheduling.

However, this morphology-based nomogram cannot serve as an independent clinical decision tool due to methodological, molecular and anatomical constraints. It provides the auxiliary evidence and would further be combined with postoperative histopathology, mandatory KIT/PDGFR genotyping and full sarcoma MDT assessment. Further multi-center external validation and prospective trials are necessary to define its clinical value in integrated GIST precision oncology.

Disclosures

Conflict of interest statement

All authors declare no conflicts of interest for this invited commentary manuscript.

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Author contributions

Yonghong Dong: Conceptualization, manuscript critical revision, corresponding author supervision; Junjie An: Literature retrieval, full manuscript drafting; Yingzheng Ren: Original focal study data interpretation, statistical model analysis; Yang Yang: Clinical oncology workflow validation, molecular pathology discussion revision. All authors approved the final submitted manuscript.

Data availability statement

No novel patient clinical or imaging datasets were generated or analyzed during the preparation of this commentary. All clinical model parameters, cohort characteristics, and statistical performance metrics discussed are sourced from the cited original published focal study.

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