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Adjunctive Use of Bioactive Iron Citrate (Synthesit®) for Mitigating Chemotherapy-Induced Myelosuppression and Hepatotoxicity in Advanced Tongue Squamous Cell Carcinoma: A Case Report

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Abstract

Intoduction: Cancer of the tongue with regional lymph node metastasis often requires aggressive chemotherapeutic regimens that may lead to severe haematological toxicity and treatment interruptions. Supportive therapies, including Synthesit®, a bioactive iron-based supplement, may help sustain treatment tolerance.

Case Presentation: This case report describes a 47-year-old patient with stage III cancer of the tongue root receiving CF (cisplatin + 5-fluorouracil) chemotherapy. After the first cycle, the patient developed profound anaemia, leukopenia, thrombocytopenia, and hepatotoxicity, necessitating treatment suspension. Supportive therapy with Synthesit®, Sorbifer, and hepatoprotectors was initiated. Within two weeks, haematological and hepatic parameters improved sufficiently to resume chemotherapy. The aim was to evaluate Synthesit® as an adjunctive agent, focusing on hematopoietic recovery, hepatoprotection, and tolerance to chemotherapy and radiotherapy. A single-case study design using electronic medical records, chemotherapy logs, laboratory test results, and imaging was employed. Hematologic and hepatic biomarkers were tracked across CF chemotherapy, Synthesit® administration, paclitaxel treatment, and subsequent radiotherapy. Following the initial CF cycle, severe myelosuppression occurred (hemoglobin 65 g/dL, WBC 3.5×10³/μL, platelets 120×10³/μL) along with hepatotoxicity (ALT 50 U/L, AST 76 U/L). After two weeks of Synthesit®, Sorbifer, and hepatoprotectors, hematologic values recovered to >90% of baseline, and liver enzymes nearly normalised. Chemotherapy was resumed, and four additional CF cycles were completed without recurrent grade 3-4 toxicities. Haemoglobin increased from 10.5 to 13.1 g/dL, RBC and hematocrit improved, and platelets stabilised. During metronomic paclitaxel plus Synthesit®, blood counts remained stable, enabling completion of 42 Gy radiotherapy. Follow-up MRI confirmed disease stability.

Conclusion: This case suggests Synthesit®, combined with hepatoprotective therapy and iron supplementation, may support hematologic recovery and facilitate continuation of cancer treatment.

Keywords: Synthesit®; Myelosuppression; Tongue cancer; Hepatotoxicity; Chemotherapy

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Introduction

Tongue cancer, an oral cavity malignancy, is an aggressive neoplasm that is often diagnosed at advanced stages due to the insidious early clinical presentation. The base of the tongue, anatomically positioned close to many lymphatic drainage sites, has a high propensity for early regional lymph-node metastasis. Stage III disease traditionally refers to tumour extension beyond the tongue base, accompanied by regional lymph node metastases, therefore requiring multimodal therapeutic regimens including surgery, radiotherapy and systemic chemotherapy [1]. Systemic chemotherapies for head and neck squamous cell carcinoma, including tongue cancers, are traditionally based on cisplatin-containing combinations, such as the cisplatin-5-fluorouracil regimen. These agents haexert synergistic effect on malignant cells t,hlikelyodueo inhibitorionf DNA replication and induction of apoptosis [2].

Nevertheless, the cytotoxic effect of these drugs is not selective between the rapidly proliferating normal mesenchymal cells of the bone marrow and the intestinal epithelium and hair follicles. However, chemotherapy is associated with serious toxicities - the most important of which is myelosuppression, which consists of anaemia, leukopenia and thrombocytopenia. This myelosuppression can impair immune competence, impair tissue oxygenation, increase hemorrhagic risk, and, most importantly, require therapy delays or dose reductions in regimens that have potentially curative efficacy [3].

Chemotherapy-related hepatotoxicity is one of the major clinical complications, besides the hematologic toxicity. Cisplatin and 5-fluorouracil have been implicated in hepatocellular injury as shown by elevations in serum transaminases [4]. This hepatotoxicity may worsen fatigue, impair drug metabolism, and reduce tolerance to further cycles of chemotherapy. Treatment of chemotherapy-induced toxicities is usually limited to supportive care, such as transfusions, treatment with hematopoietic growth factors, iron supplements, hepatoprotective agents and nutritional optimisation. However, not all patients respond satisfactorily to these measures, and some may have to be weaned from therapy, thus limiting overall prognosis [5].

In recent years, there has been growing interest in adjunctive nutritional supplements that could aid hematologic recovery and reduce chemotherapy-induced toxicity [6]. One of these products is Synthesit®, a bioavailable iron-based supplement made with a proprietary process that increases its bioavailability. Initial reports have suggested that Synthesit® may promote hematopoiesis and enhance oxygen transport by increasing red blood cell production and haemoglobin concentration [7]. Since myelosuppression and hepatotoxicity are the most common adverse reactions in patients with tongue cancer treated with cisplatin plus 5-FU, the identification of methods to enhance the tolerance to treatment is of significant importance. Conventional adjuvant treatments are limited, thereby highlighting the need for novel adjuvants such as Synthesit®. Synthesit® has the potential to fill this gap in therapy by stimulating hematopoietic recovery and potentially offering hepatic protection. Assessment of hematologic and hepatic parameters provides a clinically relevant approach, thereby supporting the integrative oncology goals of patient resilience, quality of life, and treatment adherence.

Case Presentation

Background

Patient L., a 47-year-old individual, was diagnosed in April 2024 with stage III cancer of the tongue root extending into the oral cavity, with metastases to regional lymph nodes. Initial treatment commenced with two planned courses of CF chemotherapy (cisplatin + 5-fluorouracil). Following the first cycle, the patient developed severe nausea, anorexia, weakness, and marked hematologic toxicity: haemoglobin 65 g/dL, erythrocytes 3.8 × 10^6/mL, WBC 3.5 × 10^3/mL, platelets 120 × 10^3/mL, ALT 50 U/L, AST 76 U/L. Due to clinical deterioration, chemotherapy was suspended. Supportive therapy was initiated, including Synthesit® (bioactive iron supplement), sorbifer, and hepatoprotectors.

Over two weeks, symptoms of intoxication resolved, and blood parameters improved sufficiently to resume CF chemotherapy. The patient completed three more cycles of CF concomitant with Synthesit® without significant toxicity. Next, the therapeutic regimen was modified to metronomic paclitaxel with concurrent administration of Synthesit® and silymarin, which resulted in stable hematologic parameters. The patient was then treated with radiotherapy, 42Gy without any complications. At the latest follow-up, imaging studies were suggestive of disease stability, and the patient maintained a good functional status.

Research Problem

Chemotherapy for advanced tongue squamous cell carcinoma frequently leads to severe myelosuppression and hepatotoxicity, causing treatment delays and compromising therapeutic outcomes. Despite available supportive care, many patients fail to achieve adequate hematologic or hepatic recovery. There is limited evidence on whether adjunctive bioactive iron supplements, such as Synthesit®, can enhance tolerance to chemotherapy. This case report addresses the need for supportive strategies that sustain treatment continuity in such high-toxicity settings.

Objectives

To describe the clinical course of a patient with advanced tongue root cancer receiving Synthesit® supplementation during chemotherapy, evaluate its effects on hematopoietic recovery and hepatoprotection, and assess its role in improving treatment tolerance to chemotherapy and radiotherapy, thereby informing future studies on nutritional support in oncology.

Results

Table 1 shows that following the first chemotherapy cycle, the patient developed marked myelosuppression with reductions in haemoglobin, red blood cells, white blood cells, and platelets. However, within two weeks of recovery, haematological markers showed substantial improvement, reaching over 90% of baseline values. Hepatic enzymes showed transient elevation, particularly AST, but returned to near-normal levels during recovery, indicating a reversible pattern of hepatotoxicity. After two weeks of taking the prescribed supportive products, the patient's overall condition improved, symptoms of intoxication were relieved, and blood parameters further normalised, allowing continuation of treatment with cytostatics.

Table 1. Haematological and Hepatotoxicity Markers at Baseline, Post-First Chemotherapy, and After Two Weeks of Recovery.

Marker	Normal Range	Baseline	After 1 Chemotherapy	After 2 Weeks Recovery
Hemoglobin (g/dl)	120-160	120	65	110
RBC (×10^6/ml)	3.9-5.4	4.5	3.8	4.4
WBC (×10^3/ml)	4.0-10.0	6.0	3.5	5.8
Platelets (×10^3/ml)	150-400	250	120	230
ALT (U/L)	<40	35	50	38
AST (U/L)	<40	35	76	40

Table 2 shows that the patient received four additional courses of chemotherapy while continuing Synthesit®, Sorbifer, and hepatoprotectors. Serial blood tests demonstrated a progressive recovery in red cell indices, with haemoglobin levels increasing from 10.5 g/dL in the second course to 13.1 g/dL by the fifth course. Hematocrit and RBC counts showed parallel improvements, indicating effective hematopoietic recovery. MCV, MCH, and MCHC remained within acceptable ranges, while RDW values reflected transient variability during treatment but stabilised toward the end of the treatment period.

Table 2. Red Blood Cell Parameters During Subsequent Chemotherapy Courses with Supportive Products

Parameter	2nd	3rd	4th	5th
Hemoglobin (HGB, g/dL)	10.5	11.7	12.1	13.1
Hematocrit (HCT, %)	31.4	34.8	36.0	39.8
Red Blood Cells (RBC, ×10^6/µL)	3.28	3.75	3.80	4.38
Mean Corpuscular Volume (MCV, fL)	95.7	92.8	94.7	90.9
Mean Corpuscular Hemoglobin (MCH, pg)	32	31.2	31.8	29.9
Mean Corpuscular Hemoglobin Conc. (MCHC, g/dL)	33.4	33.6	33.6	32.9
Red Cell Distribution Width – SD (RDW-SD, fL)	47.8	49.0	59.9	50.1
Red Cell Distribution Width – CV (RDW-CV, %)	14.5	14.6	17.8	15.1

Table 3 shows that across the 2nd to 5th chemotherapy courses, white blood cell counts fluctuated but generally remained within recoverable ranges, peaking at 14.94 ×10^3/µL in the 4th course before normalising. Neutrophil and lymphocyte levels showed variable patterns, reflecting treatment-related immune modulation, while monocytes, eosinophils, and basophils remained stable. Platelet counts initially spiked to 630 ×10^3/µL after the 2nd course but subsequently stabilised within the physiological range. PDW and MPV values indicated dynamic but balanced platelet activity throughout treatment. Figure 1 summarises these trends graphically, illustrating the recovery of haematological and hepatic parameters following chemotherapy and supportive treatment.

Table 3. White Blood Cell and Platelet Parameters During Subsequent Chemotherapy Courses with Supportive

Parameter	2nd	3rd	4th	5th
White Blood Cells (WBC, ×10^3/µL)	13.45	8.10	14.94	9.49
Neutrophils (×10^3/µL)	7.47	3.55	4.53	4.98
Lymphocytes (×10^3/µL)	4.06	3.81	7.99	3.18

Monocytes (×10³/μL)	1.84	0.43	2.28	0.95
Eosinophils (×10³/μL)	0.05	0.25	0.10	0.30
Basophils (×10³/μL)	0.03	0.06	0.04	0.08
Platelets (PLT, ×10³/μL)	630.0	205.0	358.0	356.0
Platelet Distribution Width (PDW, fL)	7.9	11.2	8.6	8.5
Mean Platelet Volume (MPV, fL)	8.6	10.5	8.6	9.0

Figure 1 provides a visual summary of the haematological and hepatic recovery during chemotherapy with supportive therapy. Panel a shows changes in haemoglobin, WBC, and platelets over time, highlighting the myelosuppression caused by the first chemotherapy cycle and recovery within two weeks. Panel b tracks the progressive recovery of RBC indices across subsequent chemotherapy cycles, with significant improvements in haemoglobin, hematocrit, and RBC counts. Panel c depicts fluctuations in WBC subsets and platelets, with WBC counts peaking in the fourth cycle before stabilising, and platelets initially spiking before stabilising in later cycles.

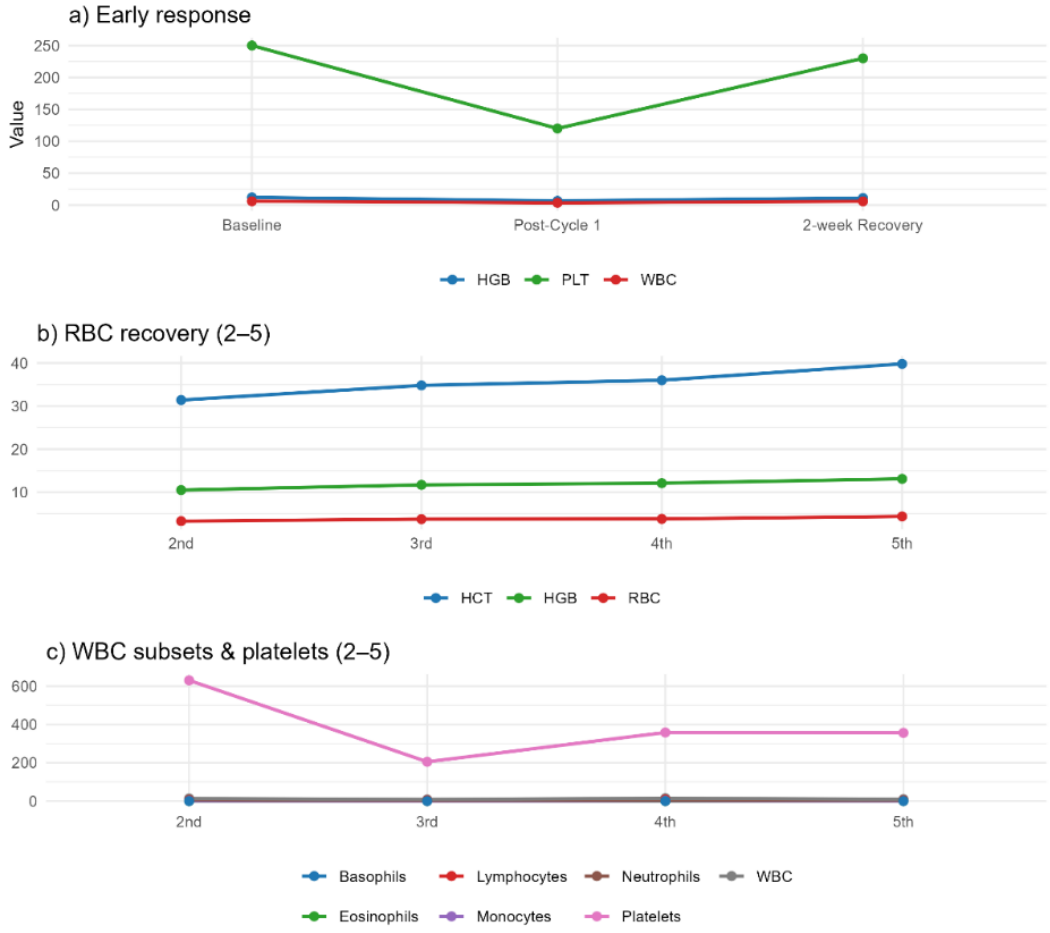

Figure 1. **Haematological and Hepatic Recovery Trends During Chemotherapy with Supportive Products.**

Discussion

The current case report illustrates how adjunctive administration of Synthesit® within a regimen that also included conventional iron supplementation and hepatoprotective agents was temporally associated with recovery from chemotherapy-induced myelosuppression and transient hepatotoxicity, and with subsequent stabilisation of haematological markers across multiple chemotherapy cycles, allowing uninterrupted continuation of intensive cancer treatment. Although causality cannot be established in a single case, the progressive normalization of hemoglobin (from 65 g/dL post-cycle 1 to 13.1 g/dL by the fifth cycle), stable RBC indices, and recovery of WBC and platelet counts suggest a clinically meaningful association, aligning with broader evidence supporting the therapeutic value of iron supplementation and hepatoprotection in oncology, while also illustrating the gaps that remain. Anaemia, particularly chemotherapy-induced anaemia, is exceedingly common among cancer patients, with prevalence between 30 % and 90 % depending on population and timing of assessment [8]. CIA often results from a complex interplay of factors, including nutritional deficiencies, blood loss, bone marrow suppression, inflammation-mediated iron sequestration, and cytokine-driven dysregulation of erythropoiesis [9].

Previous case studies have reported similar supportive benefits with Synthesit® in a pancreatic cancer patient, with neutrophil counts normalising and quality of life improving after three cycles of Synthesit® administration [10]. In fallopian tube cancer, iron citrate supplementation appeared to influence anaemia, platelet fluctuations, and immune markers, alleviating chemotherapy-induced neuropathy [11]. Additionally, iron citrate Synthesit® improved spatial memory and learning efficiency in SHK strain mice, suggesting systemic benefits beyond hematologic effects [12]. Even in dyslipidemic patients, Synthesit® showed initial cholesterol-lowering effects, highlighting potential metabolic advantages [13].

Iron supplementation, particularly intravenous (IV) iron, has been studied as a key component of managing CIA. Trials combining erythropoiesis-stimulating agents with iron supplementation demonstrated improved hematologic responses and reduced transfusion requirements when compared to ESAs alone [14]. The Cochrane review of eight randomised controlled trials (2,087 participants) concluded that adding iron to ESAs boosted haemoglobin response and decreased transfusions without raising serious adverse outcomes. Alternative approaches using IV iron without ESAs have also shown promise. One study of gynecologic oncology patients receiving platinum-based therapy observed a significant reduction in transfusion needs when IV iron (iron sucrose) was administered [15].

Moreover, prospective data in metastatic solid tumours revealed that functional iron deficiency, a common contributor to CIA, is prevalent and often requires correction with IV iron [16]. In the current case, Synthesit®, a highly bioavailable oral iron supplement, was administered alongside Sorbifer. While not IV iron, the marked rebound in haemoglobin (92% of baseline within two weeks, and progressive normalisation through subsequent cycles) and parallel improvements in hematocrit and RBC counts suggest potential efficacy in correcting functional iron deficiency, thereby aiding recovery. Whether Synthesit® is superior to conventional oral iron salts in tolerability, absorption, or hematopoietic stimulation remains unknown, but its performance in this case warrants further investigation.

Chemotherapy can cause hepatocellular injury and elevations in aminotransferases, especially with agents like cisplatin and 5-fluorouracil. Monitoring liver function during therapy is crucial [17]. Various hepatoprotective agents, including glutathione, magnesium isoglycyrrhizinate, and polyene phosphatidyl choline, have been employed to mitigate this injury. A retrospective cohort study involving 98 cancer patients receiving chemotherapy found that glutathione and MgIG significantly lowered ALT and AST levels, while PPC had no significant effect [18]. Additionally, a pharmacological intervention in Chinese patients with drug-induced liver injury during chemotherapy showed that combining PPC with bicyclol significantly reduced transaminase levels compared to PPC alone [19]. Despite these promising observations, a recent retrospective analysis of prophylactic hepatoprotective use in cervical cancer patients suggested that such agents did not reduce liver injury risk and may even be associated with heightened markers of hepatic stress [20]. This underscores that the clinical efficacy of prophylactic hepatoprotection remains uncertain and context-dependent, potentially varying with the agent, timing, and patient population.

In this case report, the patient's hepatotoxicity resolved alongside hematologic recovery after Synthesit®, Sorbifer, and hepatoprotectors were started, with ALT returning to near-normal and AST stabilising slightly above normal after initial elevation, enabling resumption of chemotherapy. While we cannot isolate the effect of hepatoprotective agents, the normalisation of liver enzymes alongside haematological improvement suggests a synergistic benefit of the combined supportive strategy. The discrepancy in the literature between supportive findings and recent negative reports indicates the need for well-designed prospective trials to evaluate hepatoprotectors during chemotherapy [21].

Conclusions and Implications

This case report demonstrates that adjunctive use of Synthesit®, in combination with conventional iron supplementation and hepatoprotective agents, may support hematologic recovery and mitigate chemotherapy-induced hepatotoxicity in advanced tongue squamous cell carcinoma. Following severe myelosuppression after the initial CF chemotherapy cycle, supplementation with Synthesit® enabled restoration of haemoglobin, RBC, WBC, and platelet counts to near baseline within two weeks, allowing continuation of full-dose chemotherapy and subsequent radiotherapy without significant interruptions. Hepatic enzymes also normalised, suggesting a potential hepatoprotective effect. While this single-case observation cannot establish causality, it highlights the clinical value of integrating bioactive iron supplementation into supportive care protocols for oncology patients. These findings warrant further investigation through controlled trials to determine reproducibility, optimal dosing, and the comparative benefits of Synthesit® versus conventional iron therapy. Clinicians may consider routine iron status monitoring and supplementation to enhance treatment tolerance, improve hematologic resilience, and potentially optimise patient outcomes in head and neck cancers.

Impact

This case highlights the potential of Synthesit® as an adjunctive therapy to reduce chemotherapy-induced myelosuppression and hepatotoxicity, enabling uninterrupted treatment in advanced tongue cancer. By supporting hematologic recovery and stabilising liver function, Synthesit® may improve patient adherence to intensive chemotherapy and radiotherapy regimens. The findings suggest a promising role for bioactive iron supplements in

enhancing supportive oncology care, emphasising the need for further prospective studies to evaluate their broader applicability and impact on treatment outcomes and patient quality of life.

1- Declarations

1-1- Author Contributions

Conceptualisation, R.A. and S.B.; methodology, R.A.; software, R.A.; validation, R.A., S.B.; formal analysis, R.A.; investigation, R.A.; resources, R.A.; data curation, R.A.; writing-original draft preparation, R.A.; writing-review and editing, S.B.; visualisation, R.A.; supervision, S.B.; project administration, S.B.; funding acquisition, S.B. All authors have read and agreed to the published version of the manuscript.

1-2- Data Availability Statement

- Data available on request due to restrictions (e.g., privacy or ethical): The data presented in this study are available from the corresponding author upon request. The data are not publicly available due to [insert reason here].

1-3- Funding

None

1-4- Acknowledgements

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1-5- Conflicts of Interest

There is no conflict of interest.

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