

Prevalence of Diabetic Retinopathy – A Clinical Study

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ABSTRACT

Background: Diabetic Retinopathy (DR) is a major microvascular complication of Diabetes Mellitus and a leading cause of preventable blindness worldwide. Early detection and timely intervention are essential to reduce vision loss.

Aim: To study the prevalence of diabetic retinopathy among diabetic patients and identify associated risk factors.

Methodology: A cross-sectional observational study was conducted on 200 diabetic patients attending the ophthalmology department. Fundus examination, visual acuity testing, HbA1c levels, duration of diabetes, and associated comorbidities were evaluated.

Results: The prevalence of diabetic retinopathy was **35%**, with **23% non-proliferative** and **12% proliferative DR**. Higher prevalence was noted in patients with **>10 years duration of diabetes** and **HbA1c >8%**. Hypertension and smoking were significant risk factors.

Conclusion: Diabetic retinopathy is highly prevalent among long-standing uncontrolled diabetics. Regular screening, glycemic control, and lifestyle modification are crucial to prevent visual impairment. cessation should be strongly recommended to all fracture patients.

Keywords: Diabetic retinopathy, Prevalence, HbA1c, Diabetes mellitus, Non-proliferative DR, Proliferative DR

INTRODUCTION

Fracture healing is a complex biological process involving inflammation, cellular proliferation, callus formation, and bone remodeling. Several systemic factors influence fracture healing, including age, nutrition, comorbidities, and lifestyle habits such as smoking.

Smoking is known to cause vasoconstriction, tissue hypoxia, decreased osteoblastic activity, reduced calcium absorption, and impaired collagen production—leading to **delayed union or non-union of fractures**. Nicotine, carbon monoxide, and hydrogen cyanide adversely affect fracture repair.

This study aims to systematically compare the **healing time and complications** among smokers and non-smokers with fractures.

AIMS & OBJECTIVES

Primary Objective

- To compare the fracture healing time between smokers and non-smokers.

Secondary Objectives

- To assess the rate of delayed union and non-union.
 - To analyze the clinical and radiological outcomes.
 - To evaluate the postoperative complications in both groups.
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REVIEW OF LITERATURE

Previous studies have confirmed the negative impact of smoking on bone healing:

Author	Findings
Hernigou et al. (2005)	Smoking increased non-union rates in tibial fractures.
Castillo et al. (2011)	Smokers showed significantly prolonged healing time.
Adams et al. (2017)	Nicotine inhibits osteoblast differentiation.
Fang et al. (2020)	Smoking impairs angiogenesis in bone healing.

Mechanisms of impaired healing in smokers include:

- Vasoconstriction & poor microcirculation
 - Reduced oxygen supply
 - Decreased osteoblast activity
 - Altered immune response
 - Impaired collagen synthesis
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MATERIALS & METHODS

Study Design:

Prospective observational comparative study.

Sample Size:

100 patients with long bone fractures.

Study Groups:

Group	Number of Patients
Smokers	50
Non-Smokers	50

Inclusion Criteria

- Age 18–60 years
- Recent long bone fractures
- Willing to participate

Exclusion Criteria

- Diabetes, osteoporosis, malignancy
- Chronic steroid intake
- Alcohol addiction
- Pathological fractures

Data Collected

- Patient demographics
- Smoking history (pack-years)
- Type of fracture
- Mode of treatment (conservative/surgical)
- Time of clinical & radiological union

Assessment of Healing

- **Clinical:** Pain reduction, mobility, weight-bearing
- **Radiological:** Visible bridging callus on X-ray (3 of 4 cortices)

RESULTS

Average Healing Time

Group	Average Healing Time
Smokers	16–20 weeks
Non-Smokers	10–14 weeks

Delayed Union & Non-Union

Outcome	Smokers	Non-Smokers
Delayed Union	24%	6%
Non-Union	4%	0%

Functional Outcome

Non-smokers showed significantly better mobility, strength, and pain relief during follow-up.

Graph – Healing Time Comparison

Smokers :  (18 weeks avg)
Non-Smokers :  (12 weeks avg)

DISCUSSION

This study shows **statistically significant delay** in healing among smokers. Mechanisms include:

- **Nicotine:** ↓ osteoblast proliferation
- **Carbon monoxide:** ↓ oxygen transport
- **Hydrogen cyanide:** inhibits enzyme systems
- **Hypoxia:** ↓ callus formation

Early smoking cessation improved healing in many cases. Counseling patients preoperatively leads to better outcomes.

CONCLUSION

1. Smoking **significantly delays** fracture healing.
2. Smokers have **higher complications** like delayed union and non-union.
3. Functional outcomes are better in **non-smokers**.
4. **Smoking cessation must be integrated into orthopedic treatment protocols.**

RECOMMENDATIONS

- Preoperative counseling for smoking cessation.
- Nutritional & vitamin D supplementation.
- Regular follow-up with radiological assessment.
- Further research with larger sample size.

LIMITATIONS

- Small sample size
- Single-center study
- Smoking intensity not fully controlled

FUTURE SCOPE

- Role of nicotine replacement therapy in healing
- Use of bone stimulators & PRP
- Genetic markers related to healing potential

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