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TRAUMA AND TUMORIGENESIS: A COMPREHENSIVE NARRATIVE REVIEW WITH EMPHASIS ON MUSCULOSKELETAL TUMORS

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ABSTRACT

Whether physical trauma can precipitate tumorigenesis has been debated for more than a century. Orthopaedic literature contains numerous case reports and small series describing musculoskeletal (MSK) tumors at sites of prior injury, including osteosarcoma at old fractures or around fixation, fibrosarcoma/malignant fibrous histiocytoma (MFH/UPS) in chronic traumatic scars, synovial sarcoma after joint/extremity trauma, malignant peripheral nerve sheath tumor (MPNST) in traumatic neuromas, angiosarcoma in chronic osteomyelitis/foreign-body environments, giant cell tumor after bone injury, and desmoid tumor after vertebral fracture. Parallel evidence documents burn-scar malignancies (Marjolin's ulcers) and other scar-related epithelial cancers. Biologic plausibility rests on sustained inflammation, oxidative DNA damage, pro-angiogenic signaling, extracellular matrix (ECM) remodeling, and foreign body/chronic infection milieus that collectively create a pro-neoplastic niche.[206–214,20,79,215] However, causality remains challenging to prove: evidence is dominated by case reports subject to publication and recall bias, with heterogeneous latency definitions and potential reverse causation (occult tumors presenting after minor trauma). Contemporary methodological reviews generally do not support a universal population-level causal link, while acknowledging plausible site-specific risks in chronic injury contexts.[50,218] This narrative review synthesizes pathophysiological mechanisms and the reported human evidence, prioritizing musculoskeletal tumors yet summarizing other trauma-associated entities. We conclude that trauma may act as a rare co-factor in tumorigenesis at specific MSK sites, whereas absolute risk after ordinary injury is likely very low; better registry-linked designs are required to quantify risk and define which lesions merit targeted surveillance.

KEYWORDS: trauma, musculoskeletal tumors, osteosarcoma, fibrosarcoma, synovial sarcoma, MPNST, angiosarcoma, Marjolin's ulcer, chronic osteomyelitis, narrative review.

INTRODUCTION

The trauma–cancer hypothesis extends back to early surgical oncology observations and remains of practical interest to orthopaedic surgeons who follow long-standing injuries, scars, and chronic osteomyelitis. Clinically, the most persistent pattern is the emergence of sarcomas—especially osteosarcoma—at old fracture sites or near orthopedic implants, fibrosarcoma/MFH in chronic traumatic scars, synovial sarcoma in previously injured extremities, angiosarcoma in chronically inflamed bone or foreign-body contexts, and classic burn-scar carcinomas (Marjolin's ulcers).[5,6,49,51,53,55,63,69,74,76,79,84,88,91,101,104,106,107,110,116,171,172,174] Biologically, the hypothesis is compatible with modern cancer hallmarks: chronic inflammation, angiogenesis, stromal/ECM remodeling, and immune dysregulation.[206–214] Yet the literature also contains negative or equivocal data at a population level, reflecting methodological constraints inherent to rare outcomes.[50,218] This review organizes the evidence around musculoskeletal tumors after trauma and situates other tumor types as secondary domains.

Pathophysiological Mechanisms (MSK-relevant)

1) Chronic inflammation & oxidative injury: Repetitive injury sustains cytokines (e.g., IL-1, IL-6, TNF-α) and ROS/RNS, increasing DNA damage and genomic instability while driving proliferation—conditions conducive to neoplastic evolution in injured bone/soft tissue.[206–210]

2) Wound angiogenesis: Hypoxia and VEGF-dependent angiogenesis, essential for repair, can also supply nascent malignant clones.[211,212]
3) ECM remodeling & stromal crosstalk: Myofibroblasts, MMPs, and remodeling alter the biophysical niche, lowering barriers to invasion and enabling tumor–stroma reciprocity.[214]
4) Foreign body and infection: Retained fragments and chronic osteomyelitis create decades-long inflammatory milieus; rare angiosarcoma and other sarcomas have arisen in such contexts.[20,79]
5) Historical orthopaedic observations: Early bone tumor series already noted malignancies at prior trauma sites, anticipating mechanisms now familiar in tumor biology.[215,50]

Evidence from Human Literature

A. Musculoskeletal Tumors (Core Focus)

Summarized subsections on osteosarcoma, chondrosarcoma, fibrosarcoma/MFH, synovial sarcoma, MPNST, angiosarcoma, giant cell tumor, desmoid tumor, and Marjolin's ulcers.

B. Other (Non-MSK) Tumors After Trauma (Brief Overview)

Summary of brain tumors after head trauma, cutaneous carcinomas in scars, and rare visceral tumors.

Table 1: Mechanistic themes relevant to trauma-associated tumorigenesis.

Mechanism	MSK-relevant microenvironmental effect	Key references
Chronic inflammation/ROS	DNA damage, proliferation, genomic instability	[206–210]
Angiogenesis (VEGF)	Nutrient supply to expanding clones	[211,212]
ECM remodeling	Invasion-permissive niche, altered mechanics	[214]
Foreign body/osteomyelitis	Persistent cytokine signaling, endothelial activation	[20,79]
Historical clinical signal	Early bone tumor reports at injury sites	[215,50]

Table 2: Musculoskeletal tumor entities reported after trauma.

Entity	Prototypical setting	Typical latency	Illustrative references
Osteosarcoma	Old fracture ± fixation (tibia/femur/humerus/etc.)	Years	[5,6,51,57,58,62,66,70,77,81,85,88,101,104, 106,107,116,121,171,172,174]
Chondrosarcoma	Pelvis/femur/scapula/tibia post-trauma	Years	[53,86,99,107,67,172]
Fibrosarcoma / MFH (UPS)	Chronic traumatic scars/soft-tissue	Years to decades	[55,69,76,108,113,118,122,96]
Synovial sarcoma	Previously injured joint/extremity	Variable (often years)	[63,74,98,105,110,119,44]
MPNST	Traumatic neuroma / nerve injury	Years	[19,75,170]
Angiosarcoma	Chronic osteomyelitis / foreign body	Long latency	[20,79,47]
Giant cell tumor	After bone injury	Variable	[16]
Desmoid tumor	After fracture/abdominal wall trauma	Variable	[35,78]
Burn-scar cancers (Marjolin etc.)	Long-standing burn/ulcer scars	Decades	[49,84,91,54,11,12,13,15,17,29,45,95,102]

Table 3: Selected non-MSK tumors after trauma.

Domain	Examples	Notes	Illustrative references
Neuro-oncology	Glioma/GBM; Meningioma at trauma sites	Many cases; reverse causation concern	[7,8,23,32,56,59,61,72,124–168,217]
Cutaneous (non-burn)	BCC/SCC/sebaceous carcinoma in scars	Chronic injury/chemical burns/laser	[11,12,13,17,29,45,54,95]
Other	Lymphoma, rare visceral tumors	Isolated case reports	[27,34,37,38,41,42,46]

DISCUSSION

The relationship between trauma and tumorigenesis has been a recurring theme in surgical and oncological literature for more than a century. From early clinical observations of osteosarcoma developing in previously fractured bones to modern descriptions of soft-tissue sarcomas arising in chronic scars, the idea that physical injury may set the stage for malignant transformation remains both compelling and controversial. The available body of evidence, though substantial in case-based reports, is methodologically heterogeneous, often anecdotal, and confounded by biases of ascertainment

and causation. In this expanded discussion, we synthesize mechanistic plausibility, examine patterns across musculoskeletal and non-musculoskeletal tumors, assess methodological strengths and weaknesses of the literature, and outline clinical as well as research implications.

Mechanistic Basis Linking Trauma and Tumorigenesis

Several biologically plausible mechanisms support the hypothesis that trauma may act as a co-factor in neoplastic evolution. Chronic inflammation at the site of injury sustains a milieu rich in cytokines such as IL-1, IL-6, and TNF-α, along with reactive oxygen and nitrogen species that increase DNA damage and genomic instability.[206–210] This microenvironment mirrors recognized hallmarks of cancer, in which unresolved inflammation facilitates clonal expansion and selection of mutated progenitors.[211–214] Similarly, angiogenesis, essential for wound healing, can inadvertently supply emerging neoplastic clones with nutrients and oxygen, thereby favoring survival and progression.[211,212] The remodeling of extracellular matrix (ECM) and activation of myofibroblasts, matrix metalloproteinases, and stromal cross-talk create a permissive niche for invasion and metastasis.[214] Finally, the presence of foreign bodies or chronic infections, as in long-standing osteomyelitis, sustains decades of persistent cytokine signaling, exemplified by cases of angiosarcoma and other sarcomas in such contexts.[20,79] Collectively, these mechanisms render the trauma-associated microenvironment a potential incubator for malignant transformation, though only a small fraction of injuries appear to follow this pathway.

Evidence in Musculoskeletal Tumors

The most reproducible trauma-tumor associations are observed in musculoskeletal oncology. Osteosarcoma has been repeatedly reported at sites of previous fractures or around orthopedic implants, with latency periods ranging from years to decades.[5,6,51,57,58,62,66,70,77,81,85,88,101,104,106,107,116,121,171,172,174] While causation remains debated, the recurrence of such cases across diverse populations and time periods suggests that repetitive injury or local instability may provide a setting for neoplastic initiation. Similarly, chondrosarcoma has been described following trauma to the pelvis, femur, or scapula, though fewer cases exist compared to osteosarcoma.[53,67,86,99,107,172] Fibrosarcoma and malignant fibrous histiocytoma (now classified as undifferentiated pleomorphic sarcoma) appear particularly linked to chronic traumatic scars, where persistent inflammation and remodeling likely play pivotal roles.[55,69,76,96,108,113,118,122] Synovial sarcoma is another entity that has surfaced in previously injured extremities or joints, with variable latency; whether trauma accelerates an underlying translocation-driven tumor is unclear, but clinical recognition is consistent.[44,63,74,98,105,110,119] Malignant peripheral nerve sheath tumors have arisen in traumatic neuromas, highlighting how aberrant nerve repair may predispose to sarcomatous change.[19,75,170] Angiosarcoma in chronic osteomyelitis or foreign-body environments represents one of the most compelling associations, with decades-long latency and highly aggressive behavior.[20,47,79] Finally, rarer entities such as desmoid tumors or giant cell tumors have occasionally been documented in traumatized bone or soft tissue, though these may reflect coincidental occurrence given their known links to local tissue irritation.[16,35,78]

Non-Musculoskeletal Tumors

Beyond the musculoskeletal system, trauma-associated neoplasia has been described in multiple domains. Marjolin's ulcers—squamous cell carcinomas arising in long-standing burn scars—constitute the most established and widely recognized non-MSK example.[49,84,91,95,102] Chronic traumatic scars have also yielded basal cell carcinomas, sebaceous carcinomas, and other cutaneous malignancies.[11–13,17,29,45,54] In neuro-oncology, an extensive literature describes gliomas, meningiomas, and glioblastomas developing at sites of prior head injury.[7,8,23,32,56,59,61,72,124–168,217] While biologic plausibility exists—particularly via mechanisms of gliosis, inflammation, and scarring—reverse causation is a major concern, as occult tumors may precipitate the initial trauma rather than arise from it.[50,218] Rare visceral tumors, including lymphomas or carcinomas in traumatized organs, have been described but remain isolated case reports with weak causal inference.[27,34,37,38,41,42,46]

Methodological Limitations of the Literature

A critical limitation of this body of evidence is its reliance on case reports and small series, which are inherently subject to publication bias, selective reporting, and recall bias. Patients and clinicians are more likely to note trauma if a malignancy subsequently arises, whereas instances of trauma without tumor formation go unreported. Furthermore, latency definitions vary widely: some reports document sarcomas within months of trauma, while others emphasize decades-long gaps, making mechanistic linkage less convincing. Reverse causation is another significant challenge, particularly in brain tumors where minor trauma may prompt imaging and reveal an already-present lesion.[50,218] Population-level epidemiological studies, including registry analyses, have largely failed to confirm a universal trauma-cancer link, though they acknowledge plausible site-specific risks in chronic injury milieus.[50,218] Thus, while the case-based literature supports a repeating pattern, the absence of robust longitudinal and registry-linked designs hampers definitive conclusions.

Clinical Implications

For the practicing orthopaedic surgeon and oncologist, the implications of this literature are nuanced. On one hand, the absolute risk of malignancy following routine trauma is extremely low, and widespread surveillance is not justified. On the other hand, awareness of specific

high-risk contexts is essential. These include sarcomas developing in old fractures or around fixation devices, fibrosarcoma/UPS in chronic scars, angiosarcoma in chronic osteomyelitis or foreign-body settings, and carcinomas in burn scars. Clinicians should maintain a low threshold for imaging or biopsy when faced with enlarging, painful, or non-healing lesions at previously injured sites. Persistent ulcers, draining sinuses, or enlarging masses in scars warrant timely oncologic referral. Equally, traumatic neuromas that grow or change in character may merit close follow-up or MRI neurography. The practical approach involves integrating vigilance into routine trauma and reconstructive follow-up without overstating risk to patients.

Research Directions

Future research must address the methodological shortcomings of the current literature. Registry-linked designs, leveraging national cancer and trauma databases, could help quantify absolute risks and define latency windows. Molecular studies comparing trauma-associated tumors with their sporadic counterparts may elucidate whether unique mutational or transcriptomic signatures exist, thereby strengthening or refuting causality. For instance, the role of APOBEC mutational signatures in trauma-linked head and neck cancers suggests that chronic injury may leave distinct molecular fingerprints.[50,218] Moreover, prospective observational cohorts of patients with chronic osteomyelitis, large traumatic scars, or retained foreign bodies could clarify incidence rates and justify targeted surveillance strategies. Finally, translational studies on inflammation, ECM remodeling, and angiogenesis in injured versus uninjured tissues may deepen mechanistic understanding.

Balancing Plausibility and Evidence

The debate over trauma-induced tumorigenesis epitomizes the tension between mechanistic plausibility and epidemiological proof. On one side, robust biologic rationale and recurrent case patterns point toward trauma as a rare but real co-factor. On the other, the lack of consistent epidemiological confirmation underscores that trauma is not a generalizable carcinogen. The truth likely lies in between: trauma may accelerate tumorigenesis only in specific contexts, such as chronic scars, foreign-body reactions, or unstable fracture sites, where local biology provides fertile ground for malignant transformation. This perspective aligns with contemporary cancer biology, which emphasizes the interaction between genetic lesions and microenvironmental conditions in dictating tumor evolution.[206–214]

Summary of Key Points

In summary, the extended discussion highlights several conclusions. First, the most reproducible trauma-tumor associations are observed in musculoskeletal oncology, with osteosarcoma, fibrosarcoma/UPS, synovial sarcoma, MPNST, and angiosarcoma providing the clearest signals. Second, non-MSK entities such as Marjolin's ulcers and post-traumatic brain tumors illustrate broader relevance but remain more vulnerable to bias. Third, while mechanistic plausibility is compelling, methodological weaknesses prevent population-level causality from being established. Fourth, clinicians should adopt a targeted vigilance approach, focusing on chronic, non-healing, or enlarging lesions. Finally, future research must prioritize registry-based epidemiology and molecular profiling to delineate whether trauma leaves a definable oncogenic imprint.

The most reproducible trauma–tumor associations lie in musculoskeletal oncology: osteosarcoma at previous fractures/implants; chondrosarcoma after trauma; fibrosarcoma/MFH in scars; synovial sarcoma after injury; MPNST in traumatic neuromas; angiosarcoma in chronic osteomyelitis/foreign bodies; and desmoid/GCT examples. Mechanistic frameworks (inflammation, angiogenesis, ECM remodeling) align with tumor biology.[206–214] However, causality remains unproven at a population level: the literature is case-based and subject to bias. Rigorous, registry-linked studies are needed to better define risk.

CONCLUSION

Across musculoskeletal tissues, recurrent clinical patterns are reported: osteosarcoma at prior fracture/fixation sites, chondrosarcoma after trauma, fibrosarcoma/MFH in chronic scars, synovial sarcoma after localized injury, MPNST in traumatic neuroma, angiosarcoma in chronic osteomyelitis/foreign-body environments, and burn-scar carcinomas (Marjolin's ulcers). While the absolute risk after ordinary trauma is very low and causality is unproven, the repeatability of site-specific cases suggests trauma may act as a rare co-factor in tumorigenesis. Further registry-linked studies are needed to quantify risk and identify which chronic lesions merit close follow-up.[50,218]

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