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Acute Intraoperative Disseminated Intravascular Coagulation During Suppurative Keloid Excision: A Case Report

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Patient: Female, 62-year-old
Final Diagnosis: Intraoperative disseminated intravascular coagulation in chest suppurative keloid excision
Symptoms: Intraoperative diffuse • uncontrollable bleeding
Clinical Procedure: —
Specialty: Anesthesiology • Hematology • Plastic Surgery

Objective: Unusual clinical course

Background: Disseminated intravascular coagulation (DIC) is a severe coagulopathy characterized by widespread microvascular thrombosis and consumptive coagulopathy, leading to both thrombosis and hemorrhage. Whereas DIC is well documented in sepsis, trauma, and malignancy, its occurrence during surgery – particularly in the context of chronic inflammation and localized infected wounds – is exceedingly rare.

Case Report: Acute intraoperative DIC occurred in a 62-year-old woman during suppurative keloid excision. The patient had a history of recurrent keloids with chronic inflammation but no prior coagulopathy, and preoperative coagulation test results were normal. During the procedure, she developed excessive bleeding; postoperative laboratory results showed prolonged prothrombin time, low fibrinogen and platelet levels, and substantially elevated D-dimer, confirming DIC based on the International Society on Thrombosis and Haemostasis criteria. The patient received multimodal treatment, including aggressive blood product transfusion (fresh frozen plasma, cryoprecipitate, and platelet concentrate), broad-spectrum antibiotics (meropenem and daptomycin) targeting a presumed deep-seated infection, and continuous renal replacement therapy for acute kidney injury. Coagulation parameters improved within 14 hours; the patient achieved full recovery from the DIC episode and its acute complications.

Conclusions: This case highlights the potential for acute intraoperative DIC in patients with chronic inflammatory conditions and underscores the importance of early recognition, intraoperative coagulation monitoring, and a multidisciplinary management approach. Timely adherence to international DIC management guidelines may substantially improve patient outcomes in such rare but life-threatening scenarios.

Keywords: Plastic Surgery Procedures • Disseminated Intravascular Coagulation • Case Reports • Keloid • Hemorrhage

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/951241

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Introduction

Disseminated intravascular coagulation (DIC) is a life-threatening hematologic disorder characterized by widespread activation of the coagulation cascade, leading to simultaneous thrombosis and hemorrhage [1-3]. Whereas DIC is well documented in conditions such as sepsis, trauma, and malignancy, its occurrence in elective surgery remains rare [2]. Acute intraoperative DIC is particularly uncommon, especially in patients without preexisting coagulopathy.

DIC results from systemic activation of coagulation pathways, which causes microvascular thrombosis, depletion of clotting factors, and subsequent hemorrhagic complications [4]. In surgical settings, multiple factors – such as extensive tissue trauma, systemic inflammation, and underlying infection – can contribute to a procoagulant state, increasing DIC risk [2]. However, acute intraoperative DIC often presents abruptly with uncontrolled bleeding, making early recognition and timely intervention essential to prevent life-threatening complications [4].

Keloids are pathological scars characterized by excessive fibroblast proliferation and collagen deposition [5,6]. Suppurative keloids represent a severe complication, characterized by cystic cavities that predispose patients to infection and purulent drainage [7]. Although primarily considered a dermatologic condition, chronically inflamed and suppurative keloids can serve as persistent sources of systemic inflammation, potentially triggering coagulation abnormalities [6,8,9]. Despite extensive literature regarding keloid pathophysiology, no prior reports have documented acute intraoperative DIC associated with suppurative keloids.

Here, we present a rare case of acute intraoperative DIC that occurred during keloid excision. The patient had no prior history of coagulation disorders, and preoperative coagulation test results were within normal limits. We aim to explore potential links among chronic skin inflammation, bacterial infection, and acute intraoperative DIC through a detailed discussion of the case, underlying pathophysiology, diagnostic challenges, and management strategies. This case underscores the need for vigilant perioperative monitoring and a multidisciplinary approach to manage coagulopathy in surgical patients.

Case Report

A 62-year-old woman with no known comorbidities, history of coagulopathy, or use of anticoagulants presented with a suppurative keloid on the anterior chest. She had initially developed acne on her chest, which evolved into an itchy scar and subsequently a keloid. Two prior keloid excisions had been conducted 6 and 2 years earlier, both without complications. The current lesion was associated with chronic pruritus and purulent discharge but no pain or fever. The patient underwent local wound care, but no bacterial culture was obtained. Two weeks before surgery, she received metronidazole 500 mg intravenously twice daily and methylprednisolone 40 mg daily for 4 days at a local clinic. She had no history of smoking or tuberculosis and reported no systemic symptoms at admission. A preoperative consultation with the infectious disease department did not include tuberculosis as a differential diagnosis.

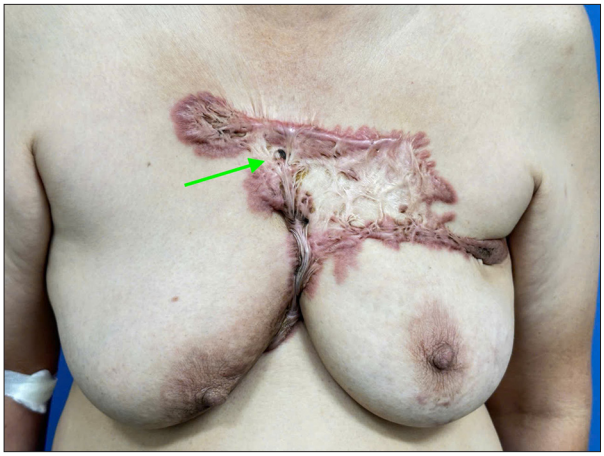

Figure 1. Extensive keloid on the chest with a cavity opening (green arrow).

On examination, the keloid was erythematous, raised, extended bilaterally across the chest, and exhibited purulent drainage (**Figure 1**). The white blood cell count was within normal limits, and no C-reactive protein (CRP) or procalcitonin (PCT) tests were performed. Preoperative coagulation parameters were within normal limits, including platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen.

Surgical Procedure and Intraoperative Complications

Surgical excision was considered first-line treatment because infection within the scar may promote keloid progression [10]. Prophylactic ceftriaxone was administered 30 minutes before surgery. The patient underwent keloid excision with local flap closure. The initial excision phase was uneventful and lasted approximately 45 minutes. However, during the flap design phase (approximately 100 minutes after the skin incision), diffuse and uncontrolled bleeding was observed at the dissection sites. Hemostasis could not be achieved with electrocautery or sutures, resulting in a total intraoperative blood loss of 700 mL. Immediate intraoperative resuscitation was initiated with administration of 1000 mL of crystalloids and 500 mL of colloids. Given the rapid and severe bleeding, an empirical loading dose of 1 g tranexamic acid (TXA) was administered via slow intravenous injection, despite the absence of

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Table 1. Laboratory results during the first 14 postoperative hours.

Parameter	Preoperative	Immediate postoperative	2 hours postoperative	9 hours postoperative (after FFP, cryoprecipitate, RBC transfusion)	14 hours postoperative (after platelet transfusion)
Platelet count (G/L)	289	185	180	61	110
PT (seconds)	14	35	77	21	13
PT (%)	90	28	12	55	102
PT-INR	1.01	2.67	6.25	1.47	0.93
aPTT (seconds)	31	66	96	41	21.6
rAPTT	1,06	2.23	3.26	1.39	0.73
Fibrinogen (g/L)	3.08	0.5	0.6	2.72	2.52
D-dimer (ng/mL)		>20 000			
Hemoglobin (g/dL)	14.6	12.2	7.2	6.0	6.4
Creatinine (µmoL/L)	68			158	198

aPTT – activated partial thromboplastin time; FFP – fresh frozen plasma; PT – prothrombin time; PT-INR – prothrombin time-international normalized ratio; rAPTT – ratio of activated partial thromboplastin time; RBC – red blood cells.

real-time coagulation testing. The unexpected and uncontrollable bleeding was unprecedented for our team during keloid excision; thus, priority was given to hemostasis and hemodynamic stabilization, which precluded acquisition of an intraoperative bacterial culture. The total operative time reached 210 minutes, and surgical drains were placed before transferring the patient to the intensive care unit.

Postoperative Course and Diagnosis

The patient developed persistent bleeding from the surgical site (200 mL/h during the first 6 hours postoperatively). Immediate postoperative laboratory tests revealed profound coagulopathy, including progressive thrombocytopenia (platelet count decreased from 289 G/L preoperatively to 61 G/L), prolonged PT-international normalized ratio [INR] (2.67), elevated aPTT (66 seconds), critical hypofibrinogenemia (fibrinogen decreased from 3.08 g/L to 0.5 g/L), and substantially elevated D-dimer levels (>20 000 ng/mL). The diagnosis of overt DIC was confirmed using the International Society on Thrombosis and Haemostasis (ISTH) scoring system, with a total score of 7 (calculated immediately after surgery; all score components corresponding to lab tests were obtained simultaneously):

- Platelet count below 100 G/L (1 point)
- Strong elevation of D-dimer (3 points)
- Prolonged PT (2 points)
- Fibrinogen below 1.0 g/L (1 point)

A score of 5 or higher is diagnostic for overt DIC in the hemorrhagic phase. Acute kidney injury developed, and oliguria progressed to anuria. Nevertheless, the patient remained hemodynamically stable without vasopressor support.

Management and Clinical Outcome

A multimodal therapeutic approach was initiated. The patient received transfusions of 9 units of packed red blood cells, 9 units of fresh frozen plasma (FFP), 10 units of cryoprecipitate, and 1 unit of platelet concentrate. These products were continuously administered during the immediate postoperative period; infusion rates and product selection were dynamically adjusted based on serial laboratory results. After the empirical 1 g intraoperative loading dose, TXA maintenance therapy was continued at 0.5 g intravenously every 8 hours to control excessive fibrinolysis. Given the high suspicion of an underlying infection, broad-spectrum antibiotics (meropenem and daptomycin) were initiated based on substantially elevated PCT levels (>100 ng/mL). Continuous renal replacement therapy was implemented to manage acute kidney injury. Anticoagulation therapy was not initiated due to the high risk of bleeding.

Within 14 hours, coagulation parameters greatly improved; the platelet count increased to 110 G/L and fibrinogen levels normalized (**Table 1**). A hematoma developed at the surgical site 2 days postoperatively (**Figure 2**). The patient continued to recover and was discharged after 40 days. The wound had healed well by 4 months postoperatively (**Figure 3**). Signs of keloid

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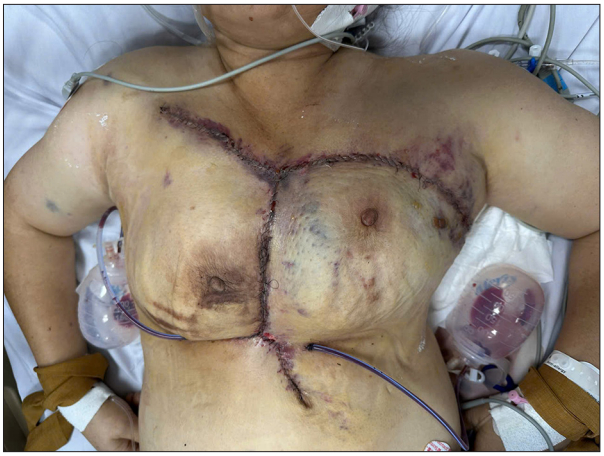

Figure 2. Hematoma and ecchymosis of both breasts at 2 days postoperatively.

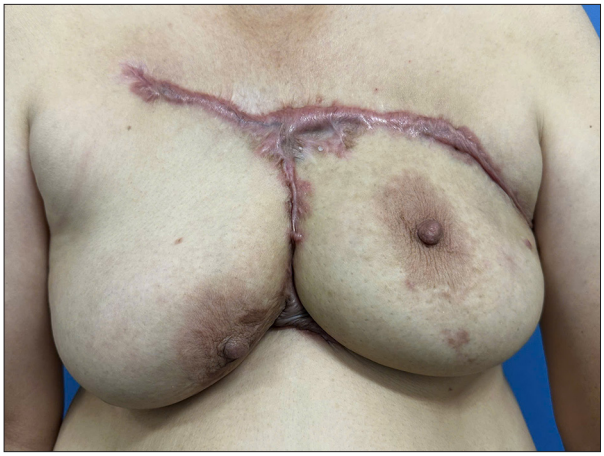

Figure 4. Signs of keloid recurrence and nipple-areola complex asymmetry at 14 months postoperatively.

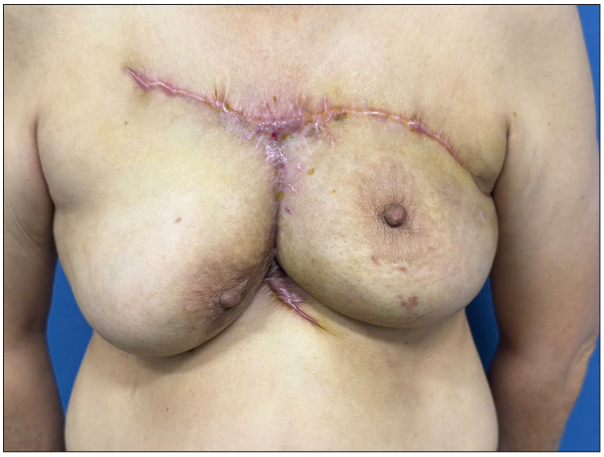

Figure 3. Well-healed wound with no signs of infection at 4 months postoperatively.

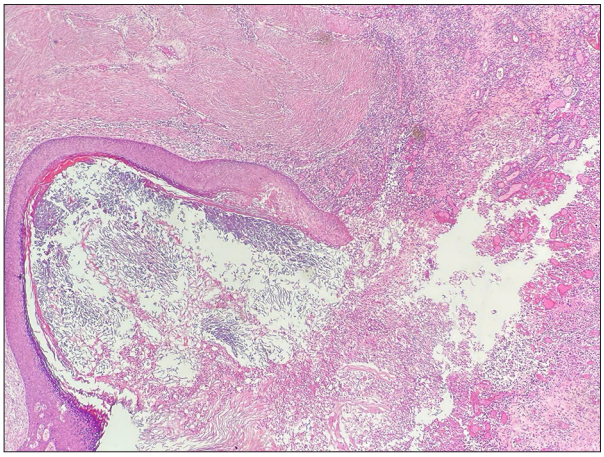

Figure 5. Histopathological assessment revealed epidermoid cysts and inflammatory infiltrates, including neutrophils, histiocytes, foreign body giant cells, and lymphocytes (hematoxylin and eosin stain, 40× magnification).

recurrence were observed 14 months after surgery, despite intralesional triamcinolone acetonide injection. Asymmetric nipples required surgical revision. No symptoms of chest infection or residual chronic kidney injury were observed (**Figure 4**).

Discussion

Pathophysiology of Acute Intraoperative DIC in a Suppurative Keloid

Histopathological examination of the excised keloid tissue revealed epidermoid cysts and inflammatory infiltrates (including neutrophils, histiocytes, foreign body giant cells, and lymphocytes), without evidence of malignancy (**Figure 5**).

DIC is a systemic coagulation disorder characterized by excessive activation of the coagulation cascade, resulting in both microvascular thrombosis and consumptive coagulopathy [2,3].

DIC is frequently associated with sepsis, trauma, and malignancy [1-3], but its intraoperative onset is rare, particularly in elective surgery. In the present case, a unique interplay among chronic skin inflammation, bacterial infection, and surgical trauma likely contributed to the onset of DIC. We speculate that the convergence of chronic inflammation, superimposed bacterial infection, and extensive surgical trauma acted as a "3-hit" mechanism precipitating acute intraoperative DIC. This framework suggests that the patient's chronic inflammatory baseline might have primed the coagulation system, which was subsequently overwhelmed by acute insults of surgical tissue injury and probable bacterial translocation. Although this model aligns with the observed clinical course and response to therapy, it remains a hypothesis requiring validation in larger cohorts.

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Chronic Inflammation as a Procoagulant State: Keloids are pathological scars characterized by persistent fibroblast hyperproliferation and excessive extracellular matrix deposition [5,6]. These lesions exhibit chronic inflammation [6,11,12], which contributes to hypercoagulability because inflammatory mediators can upregulate tissue factor expression and disrupt endothelial function, leading to systemic thrombin generation [3,4].

Superimposed Infection and Systemic Inflammatory Response: Suppurative keloids, particularly those colonized by *Staphylococcus aureus* and anaerobic bacteria, can trigger a systemic inflammatory response [7-9]. In the present case, substantially elevated PCT levels (>100 ng/mL) strongly suggested an infection-mediated inflammatory state that could have contributed to DIC onset despite negative blood culture findings.

Surgical Trauma and Endothelial Dysfunction: Extensive tissue dissection, particularly in chronically inflamed and suppurative tissues, exacerbates endothelial injury and increases the release of procoagulant factors [2,4]. The prolonged operative time (210 minutes) and considerable intraoperative blood loss (700 mL) likely further contributed to endothelial dysfunction in the present case, amplifying the patient's hypercoagulable and hyperfibrinolytic state [2,13]. These factors may explain the development of diffuse intraoperative bleeding despite normal preoperative coagulation parameters.

To situate our observation within a broader clinical context, chronic, deep-seated infections – beyond the canonical scenarios of overt sepsis and intra-abdominal or obstetric infections – can plausibly serve as persistent sources of systemic coagulation activation. In such foci, sustained inflammatory signaling and bacterial burden may prime immunothrombotic pathways; platelet-leukocyte-endothelium crosstalk, tissue factor expression, neutrophil extracellular traps, and endothelial injury could amplify thrombin generation and fibrinolysis [14]. Clinically analogous entities (eg, long-standing osteomyelitis or deep abscesses) reportedly occur along with coagulopathy or DIC, although infrequently [15,16]. Our patient's chronically suppurative cutaneous lesion and extensive surgical trauma may thus represent a biologically consistent "3-hit" scenario that could precipitate hemorrhagic-phase DIC; this perspective also explains the rapid correction once the trigger was controlled and hemostatic support was optimized.

Diagnostic Challenges

Acute intraoperative DIC presents substantial diagnostic challenges due to its dynamic nature and overlap with other coagulopathies, including dilutional coagulopathy, sepsis-associated coagulopathy, and liver-dysfunction-related coagulopathy [2,4]. Unlike classical DIC in sepsis or malignancy, acute intraoperative DIC often manifests abruptly with uncontrolled bleeding, requiring real-time recognition and management [1,2,13].

Limitations of Conventional Coagulation Tests

The ISTH DIC scoring system is widely used for diagnosis; it incorporates platelet count, fibrinogen, PT, and fibrin-related markers such as D-dimer [4,14,15]. However, conventional coagulation tests (PT, aPTT, and fibrinogen) are often performed postoperatively, delaying diagnosis. Early-stage DIC may not lead to prolonged clotting times, making reliance on these tests alone insufficient [2,15].

Distinguishing Acute Intraoperative DIC from Other Coagulopathies

Acute intraoperative DIC must be distinguished from other perioperative coagulopathies to ensure appropriate management. The following factors were considered in the present case (**Table 2**). First, surgical complications such as an unligated vessel were excluded because the bleeding presented as diffuse microvascular oozing from the entire dissection bed rather than a localized, pulsatile arterial source.

Second, allergic or anaphylactoid reactions, which can occasionally trigger sudden cardiovascular collapse and coagulopathy, were considered unlikely due to the absence of bronchospasm or cutaneous manifestations and the patient's maintenance of hemodynamic stability without vasopressor support.

Third, dilutional coagulopathy secondary to blood loss and fluid resuscitation is typically characterized by a proportional reduction in clotting factors and does not exhibit the profound hyperfibrinolysis detected in DIC [2,13]. In the present case, intraoperative blood loss (700 mL) and fluid resuscitation were relatively modest. The disproportionately severe hypofibrinogenemia (0.5 g/L) and substantially elevated D-dimer (>20 000 ng/mL) were inconsistent with dilution or blood loss alone, strongly supporting a consumptive coagulopathy [4].

Finally, sepsis-associated coagulopathy was considered given the potential infectious trigger; however, sepsis-associated coagulopathy typically presents with a thrombotic phenotype and less severe fibrinogen depletion than the overt hemorrhagic phenotype observed in the present case [2,15]. Liver-dysfunction-related coagulopathy was also excluded because it primarily involves impaired coagulation factor synthesis (rather than rapid consumption) and does not demonstrate the substantial D-dimer elevation characteristic of DIC [16]. The rapid clinical and laboratory improvement after DIC-targeted therapy (fibrinogen replacement and antifibrinolytics) further supports the diagnosis of acute consumptive coagulopathy, rather than these alternative etiologies [1,13].

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Table 2. Key distinguishing features between DIC and other coagulopathies.

Feature	Overt DIC (ISTH)	Dilutional coagulopathy	SAC	Liver-related coagulopathy	Our patient
Typical triggers	Infection, trauma, malignancy; systemic inflammation with coagulation activation	Massive crystalloid/colloid resuscitation or PRBC transfusion without balanced plasma/platelet/ fibrinogen replacement	Sepsis; may precede or progress to overt DIC	Acute or chronic liver failure; portal hypertension, hypersplenism	Chronically inflamed suppurative keloid + extensive surgical trauma; high PCT (>100 ng/mL)
Platelet count	Often below 100 G/L due to consumption	Mild to moderate decrease from dilution; improves with platelet transfusion and hemostasis	Often normal or slightly reduced early; may decrease with progression	Reduced in hypersplenism or advanced disease	61 G/L
Fibrinogen	Low (< 1.0 g/L) in bleeding-phase DIC (consumption ± hyperfibrinolysis)	Low from dilution; promptly increases with cryoprecipitate or fibrinogen concentrate	Normal or elevated initially (acute-phase reactant), may decrease later	Low in severe liver failure; dysfibrinogenemia possible	0.5 g/L
PT-INR	Prolonged (often >1.5)	Mild to moderate prolongation; improves with FFP	Mild prolongation common	Prolonged at baseline; may be greatly prolonged in liver failure	INR 6.25 (peak)
aPTT	Prolonged	Mild to moderate prolongation	Variable	Prolonged	96 seconds
D-dimer/ fibrinolysis	Substantially elevated D-dimer; hyperfibrinolysis common in bleeding phenotype	Normal or mildly elevated	Elevated but often less extreme than in overt DIC	Mild to moderate elevation possible	>20 000 ng/mL
Intraoperative bleeding pattern	Diffuse oozing from raw surfaces or needle holes; difficult hemostasis	Bleeding localized to surgical field; improves with balanced transfusion	Oozing may occur; risk of microvascular bleeding with progression	Variable; rebalanced hemostasis may mask bleeding tendency	Diffuse intraoperative oozing and early postoperative bleeding
ROTEM/TEG (typical)	Prolonged CT/CFT, low MCF; evidence of hyperfibrinolysis (eg, rapid clot lysis)	Low FIBTEM amplitude; improves after fibrinogen replacement	Variable; may become hypocoagulable with progression	"Rebalanced" profile; thrombin generation may be near normal	Not available (acknowledged limitation)
Preoperative coagulation	May be normal prior to trigger, then acutely deranged	Normal preoperatively	May show mild PT prolongation or platelet changes	Chronically abnormal PT-INR ± thrombocytopenia	Normal preoperatively

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Table 2 continued. Key distinguishing features between DIC and other coagulopathies.

Feature	Overt DIC (ISTH)	Dilutional coagulopathy	SAC	Liver-related coagulopathy	Our patient
Response to targeted therapy	Treat underlying trigger + component therapy (FFP, cryoprecipitate, platelets) ± TXA in hyperfibrinolysis; gradual correction	Rapid correction with balanced transfusion (PRBC: FFP: platelets) and fibrinogen	Treat sepsis; avoid unnecessary procoagulants; may progress to DIC	Variable response to plasma; bleeding often multifactorial	PRBC 9 U, FFP 9 U, cryoprecipitate 10 U, platelets 1 U + TXA; normalization by ~14 h
Overall impression	Consumptive coagulopathy with pronounced fibrinolysis in bleeding phenotype	Hemodilution of factors and platelets without systemic consumption	Sepsis-driven dysregulation; may progress to overt DIC	Chronic synthetic dysfunction with "rebalanced" hemostasis	Findings align with overt DIC rather than dilutional, SAC, or liver-related etiologies

aPTT – activated partial thromboplastin time; CFT – clot formation time; CT – clotting time; DIC – disseminated intravascular coagulation; FFP – fresh frozen plasma; FIBTEM – fibrin-based thromboelastometry; ISTH – International Society on Thrombosis and Haemostasis; MCF – maximum clot firmness; PRBCs – packed red blood cells; PT-INR – prothrombin time-international normalized ratio; ROTEM/TEG – rotational thromboelastometry/thromboelastography; SAC – sepsis-associated coagulopathy; TXA – tranexamic acid.

Role of Viscoelastic Coagulation Testing

Standard coagulation tests have limited sensitivity for early DIC detection. Viscoelastic assays, such as rotational thromboelastometry (ROTEM) and thromboelastography (TEG), provide real-time assessment of clot formation, stability, and fibrinolysis [4,13,15]. These modalities can help distinguish DIC-associated hyperfibrinolysis from other bleeding disorders, enabling targeted interventions such as antifibrinolytic therapy (eg, TXA) when appropriate.

Inflammatory Biomarkers for Early Detection

In the present case, greatly elevated PCT levels (>100 ng/mL) suggested an infection-driven inflammatory state despite negative blood culture findings. Given that occult infections can precipitate DIC, preoperative assessment of inflammatory markers (eg, CRP and PCT) may help identify at-risk patients [4,13,15].

Need for Intraoperative Monitoring

A major limitation in this case was the lack of intraoperative coagulation testing, which delayed early recognition of DIC. To clarify the clinical context, our institution does not have in-house access to viscoelastic testing (ROTEM/TEG), and sample transport to a neighboring facility is not feasible during an acute bleeding crisis. Additionally, the rare occurrence of profound coagulopathy during elective soft-tissue excision contributed to a low initial level of clinical suspicion, preventing timely requests for specialized coagulation assays. Standard laboratory tests are typically performed postoperatively; therefore, DIC could have already progressed to a critical stage when results became available. Real-time intraoperative monitoring with ROTEM/TEG should be considered during surgery for chronic inflammatory conditions, particularly in cases with prolonged operative time, excessive blood loss, or suspected infection [13].

Management Strategies: A Multidisciplinary Approach to Acute Intraoperative DIC

Management of acute intraoperative DIC is particularly challenging due to the need to balance hemorrhagic and thrombotic risks while simultaneously addressing the underlying cause. Successful management requires a multidisciplinary approach that integrates coagulation support, infection control, and organ protection to stabilize the patient and prevent further complications [2,13].

Hemodynamic stabilization is the primary goal in hemorrhagic-phase DIC. Our patient developed severe intraoperative bleeding requiring aggressive transfusion of blood products, including packed red blood cells, FFP, cryoprecipitate, and platelet concentrate. These transfusions were intended to restore coagulation factors, correct thrombocytopenia, and maintain adequate tissue oxygenation. Additionally, TXA was administered

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to mitigate excessive fibrinolysis, a key feature of hemorrhagic-phase DIC. The decision to administer TXA was based on the patient's hyperfibrinolytic phenotype. Although TXA is generally contraindicated in the thrombotic phase of DIC, our patient presented with uncontrolled bleeding, critical hypofibrinogenemia (0.5 g/L), and substantially elevated D-dimer levels (>20 000 ng/mL). In this context of hemorrhagic-phase DIC with confirmed excessive fibrinolysis, antifibrinolytic therapy was considered necessary to stabilize clot formation and reduce transfusion requirements. Current European Society of Anaesthesiology and Intensive Care guidelines emphasize a targeted transfusion strategy guided by laboratory parameters, rather than empirical blood product administration [13]. In the present case, serial monitoring of PT, aPTT, fibrinogen, and D-dimer played a critical role in guiding transfusion therapy.

Given the high likelihood of infection as a trigger for DIC, broad-spectrum antibiotics were promptly initiated. Despite negative blood culture findings, substantially elevated PCT levels (> 100 ng/mL) strongly suggested an underlying bacterial infection. Empiric meropenem and daptomycin were selected to provide broad coverage against both gram-positive and gram-negative pathogens, particularly targeting a potential deep-seated infection within the keloid tissue [7-9]. Our case underscores the importance of early screening for inflammatory markers (eg, CRP and PCT) in surgical patients with chronic inflammatory conditions, given that timely infection control can reduce the risk of perioperative coagulopathy.

As our patient's condition progressed, acute kidney injury occurred; oliguria progressed to anuria, which required continuous renal replacement therapy. This intervention provided renal support, facilitated clearance of circulating inflammatory mediators, and contributed to modulation of coagulation function. Given the high risk of bleeding in hemorrhagic-phase DIC, systemic anticoagulation (eg, heparin) was avoided, consistent with current DIC management guidelines [2,4,13].

Short courses of systemic corticosteroids can transiently blunt inflammatory responses and modestly affect hemostasis. However, given the approximately 2-week interval between the 4-day methylprednisolone course and surgery, and our patient's overt hemorrhagic-phase DIC characterized by severe hypofibrinogenemia and substantially elevated D-dimer levels, this brief exposure is unlikely to have been a primary driver of intraoperative coagulopathy, although a minor modulatory effect cannot be excluded.

A primary limitation of this study is the absence of preoperative data concerning microbiological cultures and inflammatory biomarkers (CRP and PCT). Although the presence of purulent discharge and substantially elevated postoperative PCT levels (>100 ng/mL) strongly support an infectious etiology, the lack of baseline data precludes definitive causal confirmation. Accordingly, infection should be interpreted as a highly probable contributing factor, rather than a proven mechanism. Future studies should incorporate standardized preoperative inflammatory screening and microbiological culture collection to better elucidate the relationship between suppurative keloids and systemic coagulopathy.

The present case emphasizes the critical role of a structured, multidisciplinary approach in managing acute intraoperative DIC. Successful outcomes depend on early recognition, targeted transfusion strategies, prompt infection control, and organ support. Routine preoperative screening of inflammatory markers (CRP and PCT), real-time intraoperative coagulation monitoring (ROTEM/TEG), and standardized transfusion protocols should be considered in the perioperative care of patients with chronic inflammatory conditions during elective surgery. Stronger collaborations among surgeons, anesthesiologists, intensivists, and hematologists are essential to improve early detection and management of acute intraoperative DIC.

Future Considerations: Can We Prevent Similar Cases?

Efforts to prevent acute intraoperative DIC in elective surgery require a high index of suspicion and proactive risk stratification. When patients present with chronic, extensive, or suppurative inflammatory lesions – not limited to keloids – preoperative screening of inflammatory biomarkers (eg, CRP and PCT) is recommended to identify occult systemic inflammation. Additionally, preoperative bacterial culture and susceptibility-guided therapy should be considered to reduce the risk of triggering DIC. Intraoperatively, particularly in cases involving complex dissection or potential for severe blood loss, the integration of real-time viscoelastic monitoring (ROTEM/TEG) warrants consideration. Such point-of-care testing may facilitate early detection of hyperfibrinolysis and guide targeted transfusion therapy more effectively than empirical approaches. Furthermore, the establishment of preemptive multidisciplinary coordination among surgeons, anesthesiologists, and hematologists in high-risk cases may enable a more rapid and effective response to unexpected bleeding. Prospective studies are needed to determine whether these standardized screening and monitoring strategies can reduce the risk of severe intraoperative coagulopathy in patients with chronic inflammatory diseases.

Clinical Implications

The present case provides a distinct clinical insight: severe, life-threatening DIC may arise from localized chronic inflammatory conditions in the absence of preexisting systemic sepsis or malignancy. It challenges the assumption that elective soft-tissue surgery in hemodynamically stable patients is inherently

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low-risk for coagulopathy. Our findings suggest that chronically infected keloids can act as a source of inflammatory priming capable of triggering overt DIC after surgical disruption.

Conclusions

This case illustrates the rare occurrence of acute intraoperative DIC associated with a suppurative keloid, suggesting a complex interaction among chronic inflammation, potential bacterial infection, and surgical trauma. Clinical experience from this case highlights the potential value of preoperative inflammatory risk assessment and intraoperative coagulation monitoring (ROTEM/TEG) in selected high-risk patients with chronic inflammatory conditions. Effective management requires a coordinated multidisciplinary approach integrating targeted transfusion therapy, infection control, and organ support. Future studies should explore the predictive utility of inflammatory biomarkers in preventing such life-threatening complications during elective surgery.

Acknowledgments

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Institution Where Work Was Done

Hanoi Medical University Hospital, Hanoi, Vietnam.

Patient Consent

This manuscript was written in accordance with the CARE checklist. Written informed consent was obtained from the patient to publish this manuscript and the accompanying images.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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