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CASE REPORT PEER REVIEWED | OPEN ACCESS

Significant cisplatin-associated carotid artery thrombosis treated and successfully rechallenged with cisplatin in a patient with testicular cancer

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ABSTRACT

Introduction: Cisplatin-based systemic chemotherapy remains the cornerstone of treatment of testicular germ cell tumors, yet it carries a significant risk of thromboembolic complications. Although venous thromboembolism is more frequently reported, arterial thrombotic events (ATEs)—including ischemic stroke—represent a serious but underrecognized complication that can occur acutely, even after one treatment cycle.

Case Report: This case report describes a 29-year-old male with metastatic mixed germ cell tumor who developed acute left-sided hemiparesis and dysarthria on day 7 of cycle one etoposide-cisplatin. Imaging revealed total right internal carotid artery occlusion with evolving middle cerebral territory infarction. The patient underwent urgent thrombolysis and thrombectomy with excellent neurological recovery and complete resolution of deficits. Potential contributing factors included a history of smoking and chronic tonsilitis. Following repeated multidisciplinary discussions, the patient was rechallenged with further three cycles of cisplatin and etoposide concurrently with anticoagulation and close monitoring. He achieved a complete biochemical response and subsequently complete radiological response after retroperitoneal lymph node dissection (RPLND).

Conclusion: This case report raises few critical points: including the importance of maintaining high index of suspicion for acute thromboembolism, particularly in patients with associated risk factors; the paramount value of various multidisciplinary team members, including stroke team, interventional radiology and neurology services; and the rechallenge with cisplatin remains the most critical decision. The use of carboplatin for cisplatin not only offers inferior results but may even be associated with higher thromboembolic risk and finally given the curative treatment intention in this young, otherwise healthy young population, continued appropriate anticoagulation therapy with vigilant monitoring represent fundamental approach. Our case adds to the sparse data on cisplatin-associated arterial thromboembolism and highlight the urgent need for universal management guidelines.

Keywords: Cisplatin, Stroke, Testicular cancer, Thrombosis

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INTRODUCTION

Acute ischemic stroke is an uncommon but increasingly recognized complication of cisplatin-based chemotherapy [1]. Testicular germ cell tumors are among the most curable solid malignancies in young men, with cisplatin-containing regimens forming the backbone of treatment and achieving excellent survival outcomes even in metastatic disease [2]. Despite its efficacy, cisplatin is associated with a broad

toxicity profile that includes significant vascular adverse events, with both venous and arterial thromboembolism increasingly reported even in patients without traditional cardiovascular risk factors [1, 3].

The mechanisms underlying cisplatin-related thromboembolism are multifactorial and may involve endothelial injury, oxidative stress, platelet activation, and alterations in coagulation pathways [4]. Cisplatin has been shown to induce endothelial apoptosis and increase circulating procoagulant factors, creating a highly thrombogenic milieu. More recent mechanistic work suggests that platinum agents may also trigger platelet pyroptosis through caspase-3–gasdermin E signaling, further amplifying thrombotic risk [4, 5].

Thromboembolic events occur in up to 18% of patients undergoing cisplatin-based chemotherapy, with arterial events representing a clinically significant subset [1]. While venous thromboembolism is more common, arterial thromboses—such as myocardial infarction and ischemic stroke—have been consistently reported in cohort studies and meta-analyses [1, 6]. Exposure to cisplatin appears to increase the risk of arterial thromboembolism by two to four times compared to regimens that do not include cisplatin [1, 3, 6].

While venous events are more common, acute arterial ischemic stroke following cisplatin therapy is rare and often overlooked. Case reports have documented such incidents occurring early in treatment; even after just the first cycle—among otherwise healthy young adults with germ cell tumors [7–9]. These findings underscore the importance of increased clinical vigilance when new neurological symptoms emerge during cisplatin-based chemotherapy. In this report, we present a case of a young adult with metastatic testicular cancer who experienced an acute thromboembolic ischemic stroke shortly after receiving cisplatin-based chemotherapy, contributing to the limited but expanding literature on this serious complication.

CASE REPORT

A 29-year-old male presented with a 4-week history of right testicular discomfort and underwent right radical orchiectomy. Histopathology demonstrated a 4 cm mixed germ cell tumor composed of embryonal carcinoma (60%), choriocarcinoma (15%), yolk sac tumor (15%), and immature teratoma (10%), with intratubular germ cell neoplasia and lymphovascular invasion present. Immunohistochemistry showed positivity for CD30, placental alkaline phosphatase (PLAP), and octamer-binding transcription factor % (OCT¾) with focal Alpha-fetoprotein (AFP) positivity. The tumor was confined to the testis with clear surgical margins.

Initial computed tomography (CT) thorax, abdomen, and pelvis showed no metastatic disease. Tumor markers demonstrated beta-human chorionic gonadotropin (β-hCG) 1577 IU/L, AFP 122 ng/mL, and lactate dehydrogenase (LDH) 246 U/L, with postoperative reduction to β-hCG 412 IU/L and AFP 67 ng/mL (Table 1). Subsequent rising tumor markers prompted repeat staging CT which demonstrated a new 3 cm aortocaval lymph node inferior to the renal vessels consistent with metastatic disease (Figure 1).

The patient had a significant smoking history of approximately 20 cigarettes daily since adolescence and chronic tonsillitis with markedly enlarged tonsils. Following multidisciplinary discussions, chemotherapy with etoposide and cisplatin (EP) begun after successful sperm banking.

On day 7 of the first cycle of chemotherapy, the patient developed acute left-sided hemiparesis with reduced power to 2/5 in upper and lower limbs, left-sided neglect (the patient was not recognizing left-sided surroundings), left-sided fascial weakness and severe dysarthria. Computed tomography of the brain and CT angiogram demonstrated total occlusion of the right internal carotid artery with evolving infarction in the right middle cerebral artery territory without hemorrhage (Figure 2). He underwent urgent thrombolysis with alteplase (rt-PA) 0.9 mg/kg intravenously over 90 minutes. Thrombolysis administered within 30 minutes of stroke onset, followed by thrombectomy after transfer to another center where radiology-guided thrombectomy was performed within less than 4 hours of stroke onset. Successful removal of a large thrombus (Figure 3) was achieved, and a rapid neurological improvement was observed; the left-sided hemiplegia, left-sided hemianopia, left fascial weakness, and dysarthria completely recovered within 48 hours. Magnetic resonance imaging and magnetic resonance angiography (MRA) performed after intervention demonstrated restoration of normal intracranial arterial caliber with a small residual mural thrombus at the origin of the right vertebral artery (Figure 4A and B). Magnetic resonance imaging of brain confirmed multifocal infarction involving the right basal ganglia and frontal lobe.

Table 1: Tumor marker dynamics pre- and postoperatively

T marker	Pre-op	Day 7 post-op	Day 28 post-op	Cycle 1 Day 10	Cycle 2 Day 10	Cycle 3 Day 10	Cycle 4 Day 10
hCG	1577	412	1154	766	0.5	0.2	≤0.2
AFP	122	67	20	20	11	3.4	3.5
LDH	246	221	231	260	202	231	217

Comprehensive work-up to rule out other causes of thromboembolism was negative, including screening for thrombophilia, transoesophageal echocardiogram for structural cardiac anomalies, lipids and HA1c, in-patient cardiac telemonitoring for arrythmias. Anticoagulation therapy commenced with complete neurological recovery.

Tumor markers expressed a progressive biochemical response during treatment, with satisfactory curve, β-hCG values of 1154, 766, 0.5, 0.2 IU/L, and finally ≤0.2 IU/L by day 10 of cycle 4. AFP values were 20, 11, 3.4, and 3.5 ng/mL, while LDH values were 231, 260, 202, 231, and 217 U/L (Table 1).

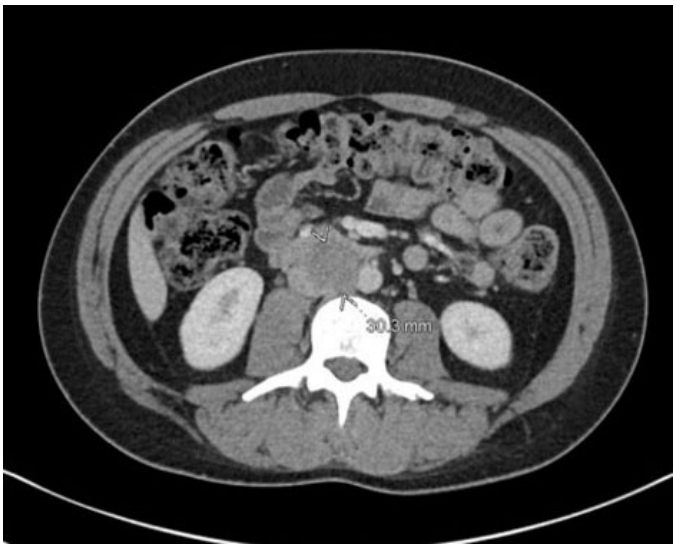

Figure 1: CT scan demonstrating 3 cm aortocaval node postoperatively and at the time of biochemical relapse.

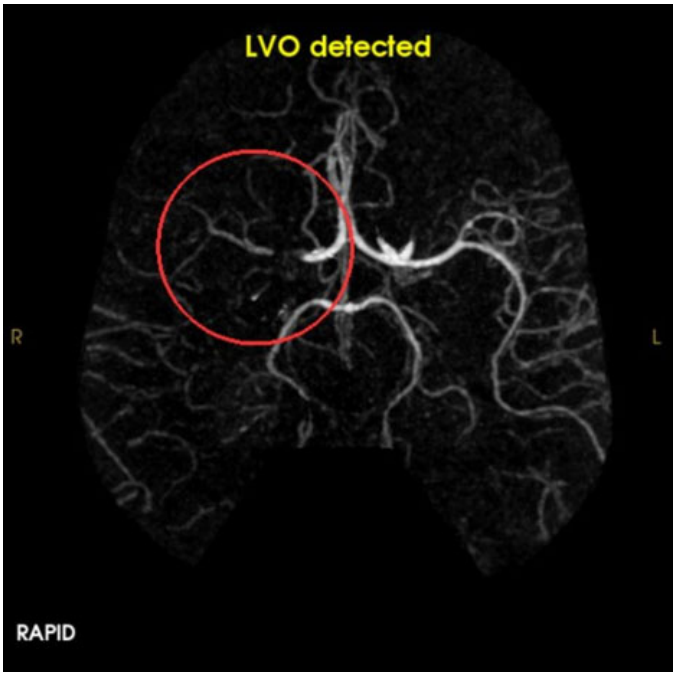

Figure 2: CT angiogram: Total occlusion of the right internal carotid artery commencing at the proximal segment with appearance concerning for dissection.

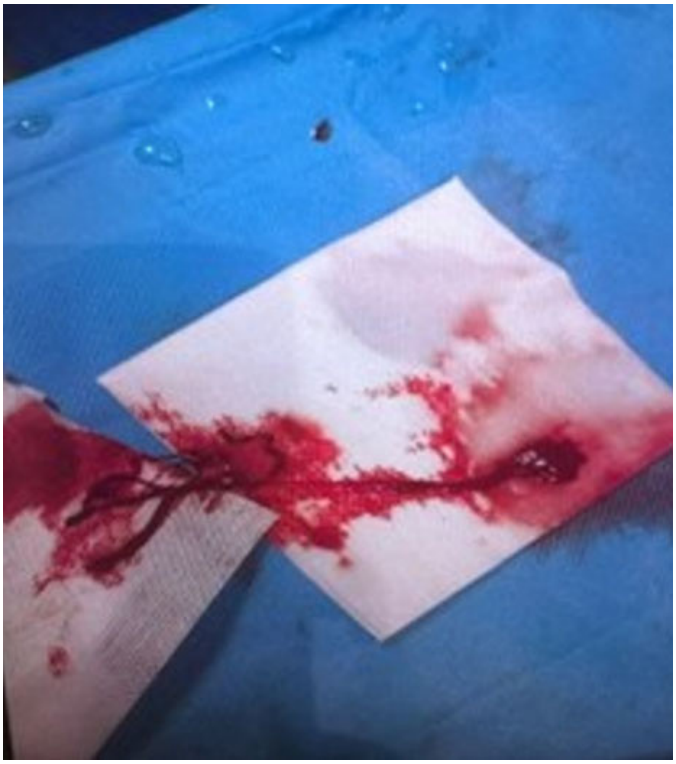

Figure 3: Large thrombus removed from internal carotid artery.

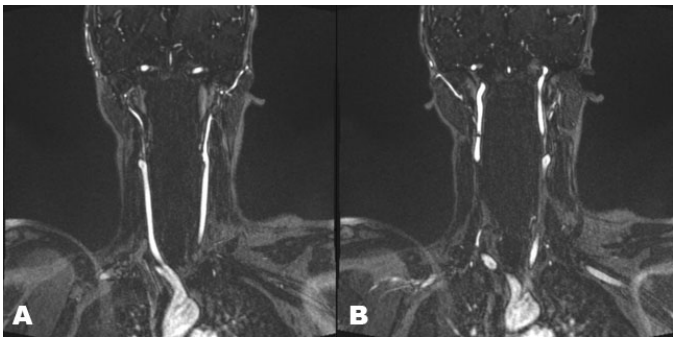

Figure 4: (A and B) 36 hours after thrombolysis and thrombectomy MRA brain/carotids showing the bilateral common carotid, internal carotid, middle cerebral, and anterior cerebral arteries are normal in caliber with no filling defect. There is a tiny volume of anterior mural thrombus within the origin of the right vertebral artery.

Treatment toxicity induced thrombocytopenia with platelet nadir of 76 × 10^9/L and neutropenia with nadir neutrophil count of 0.41 × 10^9/L during cycle 1, with subsequent recovery. Given the rare occurrence of arterial thrombosis associated with cisplatin-based chemotherapy, multidisciplinary discussion considered all options including re-challenge with cisplatin versus substitution with carboplatin. Further chemotherapy was temporarily deferred to allow neurological recovery and stabilization on anticoagulation prior to continuation of treatment. The rationale behind rechallenge decision included: the patient's complete neurological recovery; absence of underlying pathology; the patient being young

with no traditional cardiovascular risk factors; cisplatin rechallenge being initiated after successful thrombolysis, thrombectomy, and good several days of therapeutic anticoagulation, repeat angiography after stabilization on therapeutic anticoagulation confirming total resolution of the thrombus before cisplatin rechallenge was resumed; the patient being kept as an in-patient during the entire chemotherapy period; and, based on all these reasons, the risk-benefit analysis during repeated multidisciplinary meetings favored resuming the life-saving cisplatin as the most curative option, often with no available equally effective treatment. Notably our decision-making and interpretation should not be regarded as definitive as this is single patient case and there is no universally agreed consensus in such scenarios. The patient was successfully re-challenged with further 3 cisplatin-based cycles with close monitoring and appropriate anticoagulation. Six weeks post-chemotherapy CT chest, abdomen, and pelvis revealed remaining 2.1 cm residual left paraaortic lymph node with persistently normal tumor markers. The patient underwent retroperitoneal lymph node dissection (RPLND), and 49 lymph nodes were removed, with one paraaortic lymph node pathology showing mature teratoma. The patient is currently in year one of follow-up and remains in remission.

DISCUSSION

Patients with solid tumors have significantly increased risk of thromboembolic events—both arterial thrombotic events (ATEs) and venous thrombotic events (VTEs)—with seven-folds increase in comparison to general population [10].

BEP (Bleomycin, Etoposide, and Cisplatin) chemotherapy has been the cornerstone of testicular cancer management since the 1970s [11]. This has led to remarkable 5-years survival rates exceeding 90% [12] albeit for the survivors at the expense of increased multiple toxicities, including but not limited to thromboembolic events. Cisplatin, as the mainstay of the chemotherapy treatment, is associated with increased risk of thromboembolic events, mainly venous thromboembolism (VTE), but more serious and less recognized arterial thromboembolism (ATE). This significant complication can occur acutely after first treatment or long after treatment completion. The pathophysiology behind cisplatin-induced thromboembolism is multifactorial, including direct endothelial injury and activation of the prothrombotic cascade including increased von Willibrand factor and alteration in thromboxane-prostacyclin homeostasis [13, 14]. Risk factors associated with increased incidence of VTE in testicular cancer patients are not well established; however, previous reports indicates that older age, presence of cardio-vascular disease, smoking, large (>5 cm) retroperitoneal adenopathy, bulky metastasis, permanent catheters, and elevated inflammatory markers such as C-reactive protein (CRP) are all implicated [15, 16]. The only documented risk factor in our patient is smoking and possibly ongoing chronic inflammatory tonsilitis.

The incidence of venous thromboembolism in testicular cancer patients on chemotherapy ranges from 10% to 15% according to previous reports [15, 17, 18], while the incidence of arterial thromboembolic events (ATE) is much lower, with reports indicating a range of 0.2% to 2.6% during the first year of diagnosis [15, 19]; however, this small incidence tend to increase with longer follow-up and survivorship. Indeed, cases like ours sparsely reported in the literature [13, 20, 21]. Batra et al. reported a similar case of young adult who suffered a potentially fatal significant carotid artery thrombosis, also unique in that it overlapped with significant pulmonary embolism on completing first cycle of cisplatin-based chemotherapy; in their work-up and management of the case, they highlighted and stressed on the importance of early diagnosis of ATE, as it took some hours to discover the carotid artery thrombosis [8]. Overall, the incidence of cerebrovascular thromboembolism is about 1 in 2000 patients [13].

Our case highlights not only the importance of high index of suspicion to such rare and potentially disabling and fatal complication of cisplatin-based chemotherapy in this highly curable cohort of mostly young and otherwise fit patients, but it shows the excellent and rewarding results of prompt multidisciplinary management of such complicated scenario including timely valuable input from stroke team, neurology, interventional radiology and appropriate period of anticoagulation. This is critical, as urgent diagnosis of ATE independently associated with a significantly increased risk of all-cause mortality, with adjusted hazard ratio (HR) of 4.6 [22].

Thromboprophylaxis can be considered for prevention of VTE in some cancer patients; however, its role in ATE prevention remains less defined [23]. To the best of our knowledge, no available data support the routine use of thromboprophylaxis in testicular cancer patients.

Finally, our case highlights the most difficult question: what to do regarding the treatment of underlying metastatic testicular cancer, having had only one cycle and still three to go? This particularly pertinent having had life-threatening ATE successfully managed with excellent outcome and no residual neurological deficit, all achieved within the first 24–48 hours of emergency treatment. In short, and to the best of our knowledge, evidence on re-challenge with cisplatin after cisplatin-induced arterial thrombotic event (ATE) is simply very limited, if any, and no standardized guideline exist for this scenario. There is no high-level evidence to support the substitution of carboplatin for cisplatin in curative-intent treatment of testicular cancer and in fact this approach is discouraged by guidelines. However, one meta-analysis included eight studies with 1409 germ cell tumors patients (mostly of testicular origin) comparing cisplatin-based chemotherapy with carboplatin-based

chemotherapy; carboplatin-based chemotherapy had an increased treatment failure rate (odds ratio OR = 2.23), but similar overall survival rate (OR = 1.68) [24]. Furthermore, substituting carboplatin for cisplatin does not reduce the risk of thromboembolic events; indeed in one study of over 400 lung cancer patients, substituting carboplatin for cisplatin did not lower the risk of thromboembolism, while venous thromboembolism event rates were similar, carboplatin was associated with significantly higher incidence of arterial thromboembolic events (15%) compared to cisplatin (0%) [25]. Therefore, after several discussions with experts in the field and informed consultations with the patient and his family, we elected to continue with further 3 cycles of cisplatin-based chemotherapy. With close observation and appropriate anticoagulation, our patient was successfully rechallenged with no further complications.

CONCLUSION

Our case highlights the importance of early recognition of cisplatin-associated thromboembolism symptoms and signs, and equally the fundamental role of multidisciplinary team members' early involvement in this young cohort of patients, otherwise healthy and fit.

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Author Contributions

M Osman – Design of the work, Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

K Dunne – Design of the work, Acquisition of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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N Osman – Conception of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

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Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.