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Case Report

Spinal Epidural Hematoma Secondary to Tenecteplase for ST-Elevation Myocardial Infarction in the Setting of Trauma and Cervical Endplate Fracture

Christopher J. Parr, MD,[a] Weiang Yan, MD,[b] Olga Toleva, MD,[a] John Ducas, MD,[a] and Philip J. Garber, MD[a]

[a] *Section of Cardiology, Department of Internal Medicine, Max Rady College of Medicine, University of Manitoba, Winnipeg, Manitoba, Canada*

[b] *Section of Cardiac Surgery, Department of Surgery, Max Rady College of Medicine, University of Manitoba, Winnipeg, Manitoba, Canada*

ABSTRACT

A 78-year-old woman presented with an inferior ST-segment elevation myocardial infarction in the setting of a fall resulting in facial trauma causing an unrecognized C6 cervical endplate fracture. After administration of tenecteplase, she developed a spinal epidural hematoma requiring intubation for airway protection and cessation of antiplatelet therapies. The need to delay coronary intervention in this setting led to a recurrent inferolateral ST-segment elevation myocardial infarction that eventually required coronary bypass grafting. In the first report of a spinal epidural hematoma after tenecteplase for ST-segment elevation myocardial infarction, we emphasize the need for imaging after significant trauma before initiating thrombolysis.

RÉSUMÉ

Une femme de 78 ans a été vue en consultation pour un infarctus du myocarde inférieur avec élévation du segment ST, dans un contexte de trauma facial entraîné par une chute, causant une fracture du plateau vertébral de C6 non diagnostiquée. Après avoir reçu du ténectéplase, la patiente a présenté un hématome épidural rachidien ayant nécessité l'intubation pour protéger les voies respiratoires et l'arrêt des traitements antiplaquettaires. La nécessité de retarder l'intervention coronarienne dans ce contexte a entraîné un nouvel infarctus du myocarde inférolatéral avec élévation du segment ST, ayant par la suite nécessité un pontage aortocoronarien. Relativement au premier rapport d'hématome épidural rachidien survenu après l'administration de ténectéplase pour le traitement de l'infarctus du myocarde avec élévation du segment ST, nous insistons sur l'importance de procéder, avant d'instaurer la thrombolyse, à des examens d'imagerie chez les patients ayant subi un trauma important.

ST-elevation myocardial infarction (STEMI) is a serious and time-sensitive form of acute coronary syndrome. The main tenet of therapy is to rapidly reperfuse the ischemic myocardium. Although primary percutaneous coronary intervention (PCI) is the contemporary intervention of choice, most health centres in North America are not PCI capable.[1] Moreover, transfer to a PCI-capable centre within a target time frame is often not feasible. Current guidelines dictate that when a patient is more than 120 minutes away from a PCI-capable health centre, fibrinolytic therapy should be administered in the absence of any contraindications. Thus, thrombolytic therapy continues to play a critical role in early reperfusion. Thrombolysis with tenecteplase for STEMI portends a significant risk of bleeding, with an approximately 1% risk of intracranial haemorrhage and 5% risk of major bleeding.[2] Intracranial bleeding in particular has an exceptionally poor prognosis, with 60% associated mortality and 25% residual disability.[3,4] However, most bleeding is superficial, is procedure related, occurs at sites of vascular access, and is easily externally compressible.[5]

We present the case of a spinal epidural hematoma in the setting of head trauma after the administration of tenecteplase for an inferior STEMI as a unique complication of this otherwise potentially life-saving therapy.

Case Report

A 78-year-old woman presented via ambulance to a rural emergency department after syncope and an associated fall. The patient awakened with severe nausea and diaphoresis.

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Ethics Statement: Written informed consent was obtained from the patient before initiation of this Case Report.

Corresponding author: Dr Christopher J. Parr, 409 Tache Ave, Winnipeg, Manitoba R3A 1R9, Canada. Tel.: +1-204-260-5425; fax: +1-204-233-9162.

E-mail: parrc@myumanitoba.ca

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Novel Teaching Points

- Head or facial trauma often involves other unrecognized injuries, including neck fracture or basal skull fracture.
- Thrombolytics should not be administered in the setting of major trauma without appropriate imaging.
- Immediate imaging may significantly affect treatment decisions for acute ST-segment elevation myocardial infarction.
- Spinal epidural hematoma is a rare but potentially life-threatening complication of thrombolytic treatment.
- Delay in definitive therapy for myocardial infarction may lead to further ischemic coronary events.

While she was in a sitting position at the edge of her toilet, she suddenly lost consciousness and fell forward onto her face. Notably, she did not have antecedent angina. The patient's nose appeared flattened, had a laceration on the nasal bridge, and had periorbital bruising. An electrocardiogram showed an inferior STEMI. Aspirin, clopidogrel, and enoxaparin were administered. Given that the patient presented to a hospital more than 120 minutes away from a PCI-capable centre and that the patient was thought to have nonsignificant injuries, tenecteplase 40 mg was administered intravenously 81 minutes after first medical contact time. Several hours after administration of tenecteplase, the patient developed epistaxis and bleeding from the laceration on her nasal bridge. She was then transported uneventfully to a tertiary cardiac referral centre.

Upon arrival, she had dramatic electrocardiographic improvement of $>$ 50% resolution of her inferior ST elevation. While awaiting coronary angiography, she underwent assessment of her injuries. Because of the periorbital bruising, it was thought that a basilar skull fracture and other possible associated injuries needed to be excluded. Computed tomography (CT) of the brain, facial bones, and cervical spine was performed, showing acute fractures of the bilateral nasal bones, nasal septum, and anterior inferior endplate of C6. Moreover, there was a prevertebral spinal epidural hematoma with displacement of the hypopharynx (Fig. 1). Associated with this was stridorous breathing when the patient was supine. Further antithrombotic therapies, including aspirin and clopidogrel, were held. A cervical collar was applied for stabilization. In anticipation of possible airway compromise during transfer to a specialized trauma centre, the patient was taken to the operating room, and an awake fiberoptic intubation was performed. The patient was subsequently managed under the care of a multidisciplinary trauma service that included intensive care, general surgery, orthopedic surgery, neurosurgery, and oral maxillofacial surgery. Magnetic resonance imaging (MRI) of the spine showed extensive injury of the prevertebral and posterior paravertebral cervical spine soft tissues and a small epidural hematoma at the posterior aspect of C5 and C6. Transthoracic echocardiography revealed normal left ventricular size and function. After consultation with a neurosurgeon, it was recommended to forego antiplatelet therapy. Given that the patient did not display clinical signs of neurologic injury, close observation with serial examinations was elected over operative management. She was transferred 1 week later back to the rural hospital to allow her injuries to heal further before consideration for coronary angiography.

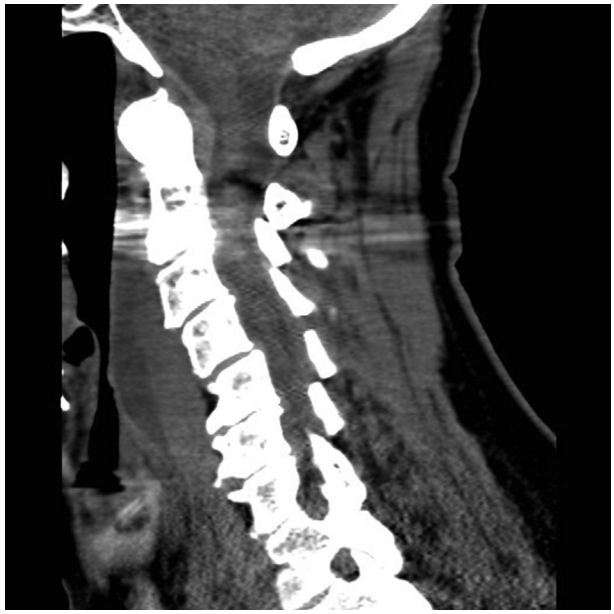

Figure 1. Sagittal computed tomography (CT) scan of the cervical spine demonstrating a prevertebral hematoma and edema extending from the mid C2 level down to the C6-7 level, anteriorly displacing and narrowing the hypopharynx, and an acute epidural hematoma along the posterior aspect of the spinal canal from C3-4 down to the C5-6 levels.

One week after discharge, the patient developed a new onset of retrosternal chest discomfort and inferolateral ST-segment elevation, and an increase in high-sensitivity serum troponin T to 8051 ng/L. She was diagnosed with a new inferolateral STEMI complicated by left ventricular dysfunction and complete heart block requiring dobutamine. This was initially managed with aspirin monotherapy and heparin. Selective coronary angiography showed severe obstructive multivessel disease, with chronic total occlusion of the left anterior descending artery, severe circumflex disease, and severe disease throughout the dominant right coronary artery. Repeat CT and MRI of the cervical spine showed interval resolution of the prevertebral soft tissue changes. Coronary artery bypass grafting was performed uneventfully on day 29 in relation to her initial presentation. The patient was discharged on day 43.

Discussion

This case is unusual in several respects. We describe the first reported case of spinal epidural hematoma after the specific administration of tenecteplase in the setting of STEMI. Likewise, the manifestation of spinal epidural hematoma as airway narrowing is uncommon, because spinal epidural hematoma typically manifests as central neurologic injury.

Spinal epidural hematoma is a rare, potentially disabling space-occupying accumulation of blood in the epidural space that mechanically compresses the spinal cord. It is an acute and rapidly progressive myelopathy that if left untreated may

result in profound sensory and motor deficits. The characteristic presentation is one of severe acute pain at the site of the lesion, followed within hours by signs of nerve root irritation, hyporeflexia, paresis, or urinary retention. Axial CT may be helpful in diagnosis spinal epidural hematoma, but MRI remains the diagnostic imaging modality of choice. Spinal epidural hematoma is a complication of thrombolysis described in both acute ischemic stroke[6] and acute coronary syndrome,[7] both spontaneous and in the setting of trauma. All cases reported thus far occurred after administration of streptokinase, urokinase, or tissue plasminogen activator.

In retrospect in relation to our case, the patient's presenting history and physical findings should have been sufficient to withhold administration of thrombolytics on the basis of major facial trauma. Society-developed practice guidelines[1,8] list significant recent (within the last 3 months) closed-head or facial trauma as absolute contraindications to fibrinolytic therapy for STEMI, among others. Within the literature, there are little data surveying the inappropriate use of thrombolytics for STEMI in the presence of trauma. Immediate imaging, by identifying the neck fracture, would have affected the decision to administer thrombolytics. Not only did inappropriate thrombolysis result in precautionary intubation, which has its own potential complications, but also thrombolytic-induced spinal epidural hematoma precluded the use of antiplatelet therapy. More important, this led to delayed coronary angiography and revascularization, because these interventions require the use of periprocedural heparin therapy and postprocedural antiplatelet therapy. Delay in definitive management contributed to the patient's second presentation with recurrent STEMI.

Conclusion

For the foreseeable future, thrombolytic agents will remain a pivotal component of treatment for STEMI. As long as this is the case, patients will be at risk of major bleeding complications, including spinal epidural hematoma. As illustrated in the described case, a focused history and physical, knowledge of the contraindications of thrombolytics, and appropriate imaging are key to deliberate and judicious use of this treatment.

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Disclosures

The authors have no conflicts of interest to disclose.

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