

A Case of Acute Anterior Myocardial Infarction Complicated by Acute Traumatic Brain Injury

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ABSTRACT

A patient was admitted to Emergency Department within an hour of an episode of syncope followed by a fall. The electrocardiogram recorded on admission was suggestive of an extensive acute anterior myocardial infarction, and an emergency computed tomography (CT) scan of his brain showed a right scalp hematoma but no intracranial injury. He underwent primary percutaneous coronary intervention (PCI). Ten hours later, the patient reported sudden onset headache and vomiting. Repeat head CT showed left-sided intracranial hemorrhage, which required immediate neurosurgical decompression and reversal of antiplatelet and anticoagulant therapies. Three months later, the intracranial hematoma had been absorbed.

Keywords: Acute myocardial infarction, traumatic brain injury, treatment

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INTRODUCTION

Acute myocardial infarction with traumatic brain injury is not uncommon, given the clinical antithrombotic therapy caused great difficulties, for example in this case report. Be sure to carefully assess and reduce the risk of bleeding may bring antithrombotic therapy in the treatment of such patients. The case took a drug-eluting stent implantation in the culprit artery, can be taken bare-metal stent, PTCA or occlusive arterial thrombus aspiration opening. Of course, bivalirudin (1) should be preferred in such patients, from this case reports, we can draw some experience and inadequate.

CASE REPORT

A 59-year-old man was admitted to our Emergency Department within an hour of an episode of syncope followed by a fall. The electrocardiogram recorded on admission was suggestive of an extensive acute anterior myocardial infarction, and an emergency computed tomography (CT) scan of his brain showed a right scalp hematoma but no intracranial injury (Fig. 1). After informed consent was obtained, he underwent primary percutaneous coronary intervention (PCI). Coronary angiography showed complete occlusion of the proximal left anterior descending artery, with Thrombolysis in Myocardial Infarction (TIMI) Study Group grade 0 forward flow. After balloon dilatation (FIRE STAR 2.5 mm × 15 mm; Cordis Corporation, NJ, USA), sirolimus-eluting stents were deployed to restore coronary blood flow (EXCEL 3.5 mm × 18 mm; JW Medical, Shandong, China) (Figs. 2, 3). To facilitate PCI, 9,000 IU intravenous heparin was administered along with 500 micrograms tirofiban.

The doses of heparin and tirofiban were calculated based on the patient's weight of 80 kg, but were reduced owing to the history of trauma. After PCI, the patient reported no chest pain and there was no further bleeding from the scalp hematoma.

Ten hours later, the patient reported sudden onset headache and vomiting. Repeat head CT showed left-sided intracranial hemorrhage (Fig. 4), which required immediate neurosurgical decompression and reversal of antiplatelet and anticoagulant therapies.

Three months later, the intracranial hematoma had been absorbed (Fig. 5). As there was no further need for neurosurgical intervention, the patient was established on oral aspirin and clopidogrel once daily. The patient made an uneventful and full recovery, with no significant neurological deficit, or subsequent angina or heart failure.

DISCUSSION

In the event of an ST-segment elevation acute myocardial infarction complicated by soft tissue hematoma, priority should be given to restoring coronary blood flow by means of PCI (2). Thrombolytic therapy is contraindicated in the presence of recent bleeding (3, 4). In this case, extensive contralateral intracranial bleeding became evident 10 hours after PCI. This is likely to represent a *contrecoup* injury, which can be explained in this case by abrupt deceleration of the brain when the occiput hit a stationary object with force. Importantly, the *contrecoup* injury may only have occurred as a consequence of the anticoagulation given to facilitate the PCI procedure; consequently heparin and tirofiban (5) should be used with caution if there is direct evidence or increased risk of hemorrhage from other sites (6). In this

case, antiplatelet (6) drugs were not administered in the immediate recovery period despite the deployment of drug-eluting stents, and the patient reported no further chest pain. In complex cases such as this, it is important to balance the risk of stent stenosis and myocardial reinfarction with that of recurrent intracranial hemorrhage when determining the best strategy for secondary prevention; however, there is little empiric evidence to inform clinical practice. We chose to restart antiplatelet therapy 3 months after the initial myocardial infarction and brain injury (7, 8), and were guided by the findings of the follow-up brain CT and the clinical condition of the patient.

CONCLUSION

Acute myocardial infarction with traumatic brain injury caused great difficulties for the clinical antithrombotic therapy. Be sure to carefully assess and reduce the risk of bleeding may bring antithrombotic therapy in the treatment of such patients.

CONFLICT OF INTEREST

All authors have no conflict of interest regarding this paper.

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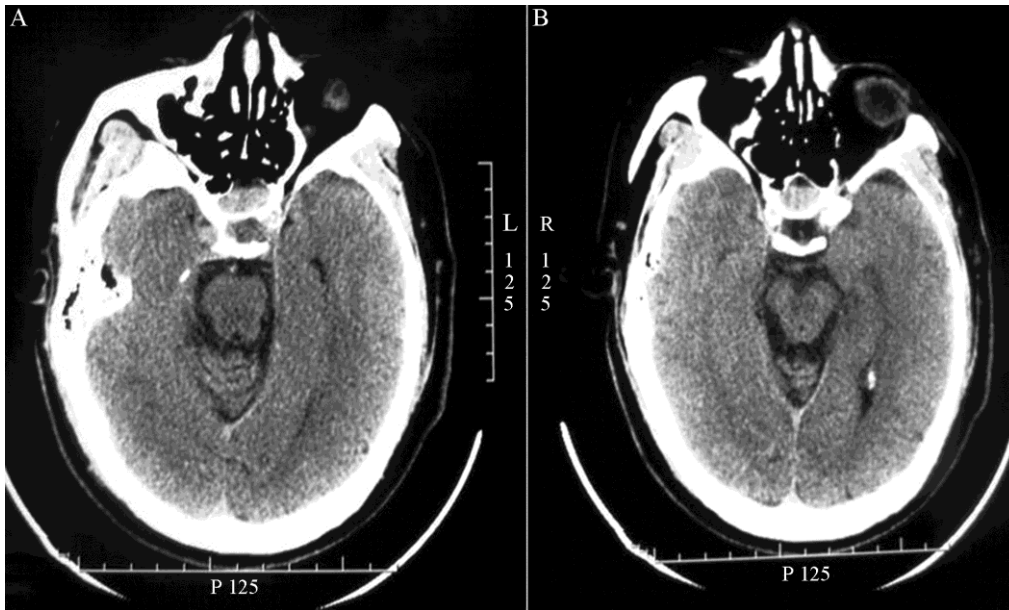


Fig.1 A, B: Emergency computed tomography examination of the head, showing a right occipital scalp hematoma, but no obvious intracranial hematoma.

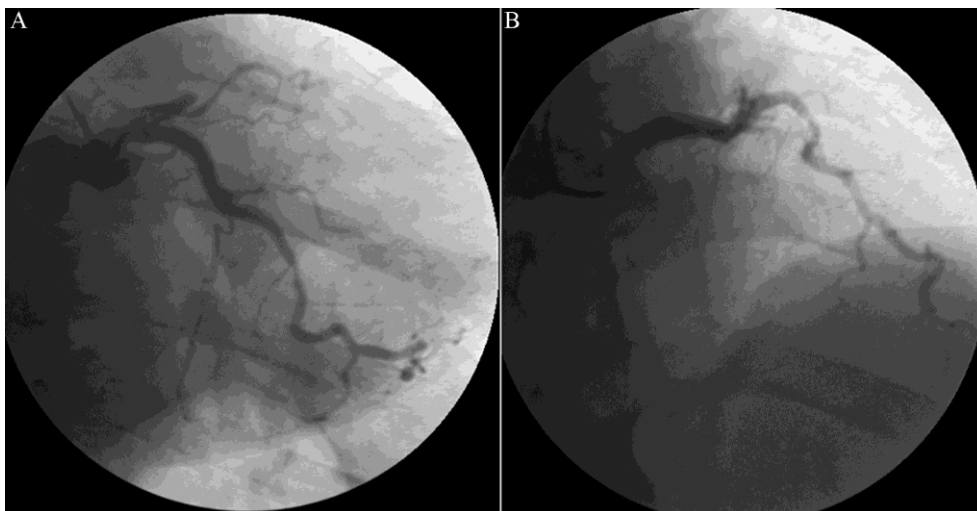


Fig.2: Emergency coronary angiography showing proximal left anterior descending artery occlusion before intervention.

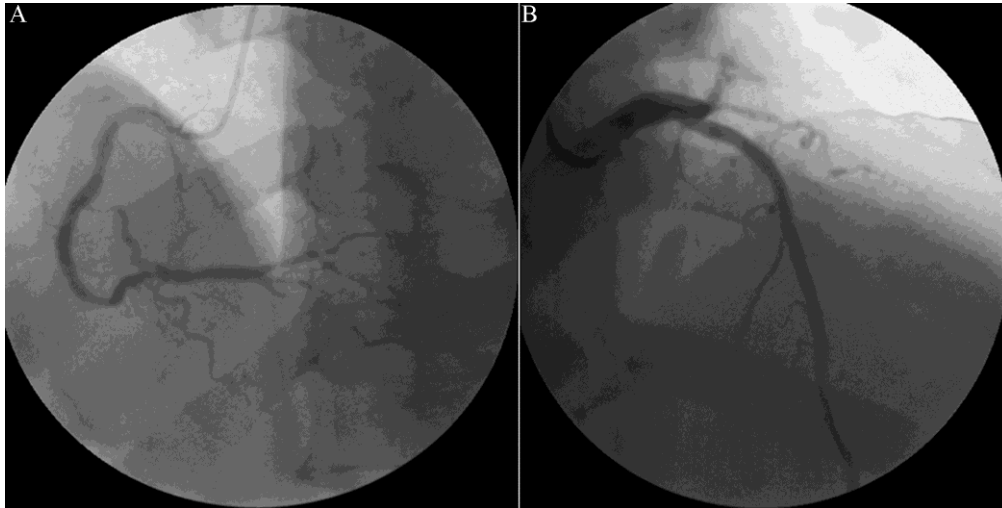


Fig.3: Left anterior descending artery PCI

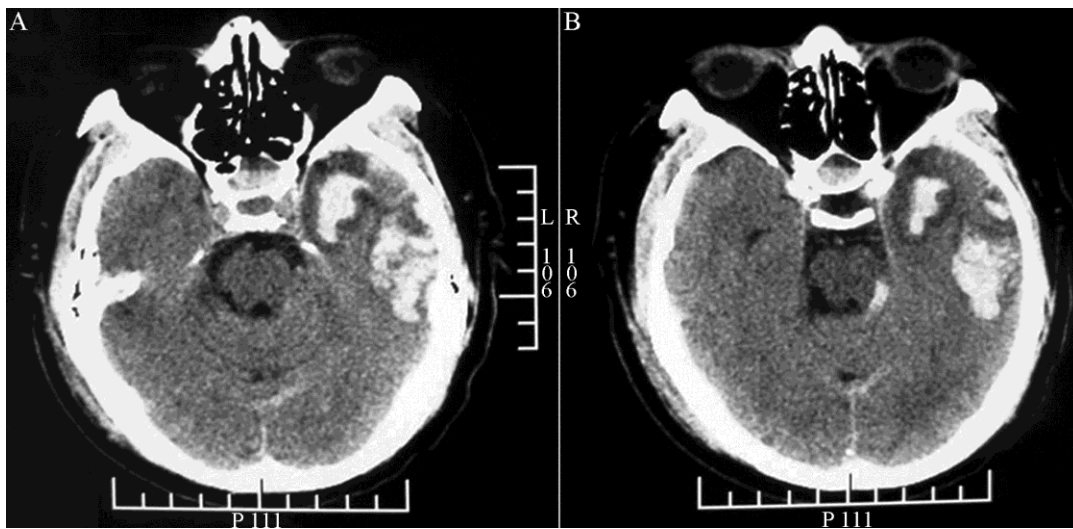


Fig.4: Left-sided intracranial hemorrhage is evident.



Fig.5: Showing absorption of the left-sided intracranial hematoma after 3 months.