

## CASE REPORT

# Spontaneous Subperiosteal Orbital Haemorrhage (SSOH): An Unusual Complication of Acute Coronary Syndrome Treatment

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### ABSTRACT

Acute coronary syndrome is a medical emergency with a high mortality and morbidity. Reperfusion therapy is widely used in its management. Ocular complications following reperfusion therapy is rare. A 51-year-old man treated with streptokinase, antithrombotic and dual antiplatelet therapy, developed mild proptosis following treatment. After four days, there was sudden worsening of the proptosis associated with orbital compartmental syndrome (OCS). The CT scan of the orbit showed a large spontaneous subperiosteal orbital hemorrhage (SSOH) occupying half of the left orbit. Despite performing an urgent lateral canthotomy and inferior cantholysis, the patient developed irreversible vision loss due to compressive optic neuropathy. While a few cases of mild SSOH have been reported in the medical literature, this is the first documented case of secondary bleed in SSOH. Early recognition and intervention by the treating physician, is crucial in preventing blindness.

**Keywords:** SSOH, proptosis, acute coronary syndrome, vision loss

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increase in the proptosis with signs of compressive optic neuropathy the CT scan done revealed a large SSOH occupying half the orbital space. Swift recognition early SSOH and intervention, may prevent its progression and devastating sequelae.

### INTRODUCTION

Thrombolytic and reperfusion therapy is widely used for acute ST elevation myocardial infarction treatment when the facilities for primary percutaneous coronary interventions is not available. While the hemorrhagic complications of reperfusion therapy, such as stroke and gastrointestinal bleeding are widely known, ocular complications are uncommon. A review of more than 40, 000 patients undergoing thrombolytic therapy, did not reveal any increase in ocular hemorrhage, even among diabetics (1). However, a few cases of mild spontaneous subperiosteal orbital haemorrhage (SSOH) have been reported with no significant visual morbidity (2). We would like to present a case of severe proptosis following an initial mild episode of SSOH. Rebleeding in SSOH has never been documented in literature. Initially, the proptosis in the left eye was mild and suggestive of orbital cellulitis due to the blood profile which supported the diagnosis of septicaemia. Immediate radiological imaging could not be performed as the patient was medically unstable to leave the intensive care unit (ICU). However, when there was a sudden

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A 51-year-old man, a heavy smoker with no significant past medical history, presented to the emergency department (ED) with retrosternal chest pain. He was diagnosed with acute myocardial infarction, as his electrocardiograph showed ST-segment elevation in multiple leads. While in ED, he developed cardiogenic shock which required resuscitation and mechanical ventilation. He was treated with double antiplatelet therapy with intravenous Streptokinase 1.5 million units, subcutaneous Fondaparinux 5mg, Aspirin 75mg and Clopidogrel 300mg.

The next day, while still under sedation and ventilatory support, the patient was referred for left eye proptosis. The attending ophthalmologist documented that there was mild non-axial proptosis of 21 mm by Hertel's exophthalmometer, with a slight displacement of the globe inferiorly. The cornea was clear, with a deep and clear anterior chamber. The pupillary reflexes were intact and fundus examination unremarkable.

There was no evidence of orbital cellulitis such as lid swelling, periocular inflammation or conjunctival chemosis. The intraocular pressure (IOP) in the right eye was mildly elevated at 22 mmHg compared to 16 mmHg in the contralateral eye. The patient was also febrile at 38.80C, his total white blood count was 19 x 10<sup>9</sup>/l, with predominant neutrophilia (82%) suggestive of septicaemia. Leucocytosis may be seen following myocardial infarction but such high white blood count is more suggestive of infection. The empirical diagnosis of orbital cellulitis to septicaemia was made and treatment with intravenous Augmentin© 1.2 gm eight hourly was started. The chest X-ray, urine examination, liver and renal profile was unremarkable. After a few doses, the fever gradually subsided despite the proptosis remaining status quo.

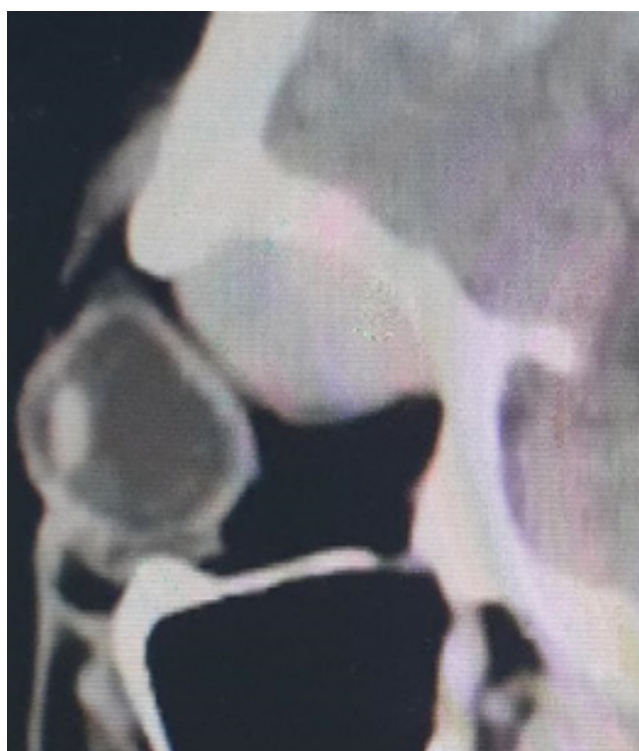
Four days after thrombolysis, there was sudden worsening of the proptosis to 28mm with significant hypotropia (Figure 1). The intraocular pressure increased to 62 mmHg and positive relative afferent pupillary defect (RAPD) was present. The fundus examination revealed that the left optic disc was swollen and the vessels were dilated and tortuous. The contrast-enhanced CT scan demonstrated a large well-defined homogenous sub-periosteal haemorrhage in the left orbit displacing the globe antero-inferiorly (Figure 2 and 3). Emergency lateral canthotomy and inferior cantholysis (LCIC) was done at the bedside under local anaesthesia, and intravenous acetazolamide 250mg six hourly and topical timolol, brimonidine and latanoprost was started at maximum dosage in an attempt to reduce the intraocular pressure and prevent compressive optic neuropathy. Over the next 3 days, the IOP remained over 40 mmHg, before gradually returning to a normal IOP. A week later, the patient was extubated and during visual assessment, the proptosis was unaltered at 28mm with positive RAPD and no perception to light in all four quadrants in the affected eye. Examination of the contralateral eye was normal with vision of 6/6 by the bedside Snellen chart. A months later, the proptosis resolved fully. There was no restriction in extraocular movement, however the impairment of vision remained.



**Figure 1: Colour photograph demonstrating proptosis and hypotropia of the left eye**



**Figure 2: Contrast CT scan in sagittal section reveals compression and deformity of the left globe as well as proptosis and inferior displacement.**



**Figure 3: Contrast CT scan in coronal section demonstrating the superior subperiosteal hematoma**

## DISCUSSION

The combination treatment strategy using thrombolytic agents and antiplatelet drugs aims at the reduction of myocardial damage and reducing MI-related mortality. This is a double edge sword which comes with the known complication of bleeding. Risk factors that predict bleeding include females, older age, renal impairment, history of hemorrhaging disease or anticoagulant utilization (3). While mild and moderate bleeding is common, and less common risk of major bleeds may be associated with stroke or MI.

The incidence of SSOH in ventilated patients has been postulated to be caused by the orbital vein congestion.

The increased intrathoracic pressure from ventilation may rupture the bridging subperiosteal vessels causing SSOH.

The most common site for SSOH is the orbital roof. This is due to the fact that it predominantly comprises of a single frontal bone. The combination of loosely adherent periosteum and a large surface area increases the risk for a large intraorbital hematoma to develop. This will then lead to OCS and optic neuropathy, following either direct trauma to the nerve or following accompanying vessels in this tightly enclosed space. The amount of proptosis can be measured using Hertel exophthalmometer and is graded as mild (21–23 mm), moderate (24–27 mm), and severe (28 mm or more). In mild and moderate proptosis, there is proptosis with no evidence of optic neuropathy or exposure keratopathy, and this can be treated with symptomatically with topical lubricant eyedrops. However, in severe proptosis there is risk of permanent visual disability due to optic neuropathy, glaucoma or corneal scarring, that requires urgent surgical decompression of the orbit. This radical surgery has been shown to successfully remove the subperiosteal hematoma (2). However, the increased risk of general anaesthesia following the recent myocardial infarction along with the increased risk for bleeding under reperfusion therapy, often means that this procedure would need to be deferred as seen in our patient.

The red herring in our patient was the raised total WBC and fever. This suggested the diagnosis of orbital cellulitis secondary to septicemia. Active smokers just like our patient have a higher WBC count, which incidentally also puts them at a greater risk for ACS (3). Elevated temperatures are also commonly seen in ACS, due to the acute phase of inflammation seen in the first three days of ACS. White blood cell counts may be increased as much as 19% in current smokers and 15% in ex-smokers (4). This is triggered by cardiomyocyte death and degradation which induces an inflammatory response from both the adaptive and innate immune systems (3). Therefore the treating physician should have a high index of suspicion, on the evolution of SSOH in a patient on thrombolytic therapy and immediately commence preemptive measures to avoid further worsening of the condition.

Ideally in all cases of reperfusion therapy with mechanical ventilation, a higher index of suspicion for SSOH should be made when there is sudden proptosis. CT scan of the orbit should be ordered as soon as possible to confirm the diagnosis, typically a superiorly located well-defined hypodense mass will be seen in SSOH. Prompt surgical evacuation of the hematoma under general anaesthesia is the definitive management for sight threatening hematomas. Our patient was not fit for general anaesthesia due to the ACS and the thrombolytic and reperfusion therapy itself runs the inherent risk of

increasing orbital bleeding during the procedure.

Another less invasive procedure maybe done at the bedside under sedation using fine needle aspiration. But this is only beneficial in the acute stage when the blood is still in the liquid form. This needle aspiration does not remove the clot and may precipitate further bleeding due to trauma to blood vessels or from sudden vasodilation of blood vessels with decompression of the orbit. Emergency lateral canthotomy and inferior cantholysis (LCIC) was done for our patient in attempt to decompress the orbital cavity, but there was no significant improvement in intraocular pressure.

The management of SSOH depends on the period of onset and severity of optic nerve involvement. In most cases, if the clot is small and there is absence of visual compromise, conservative management is sufficient with cold compression to reduce the risk for further bleeding (5). Whenever there is visual compromise and the patients is stable, surgical evacuation of the clot is the gold standard of treatment.

## CONCLUSION

SSOH is a rare complication following thrombolysis. Early recognition and intervention should be instituted immediately to prevent OCS-related complications.

## ACKNOWLEDGEMENT

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