ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

A Case of Post Thrombolytic Intracerebral Haemorrhage.

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ABSTRACT

Intracerebral hemorrhage is the most feared complication of thrombolysis. In this case we have seen how thrombolysis for myocardial infarction has led to hemorrhage. Fear of intracranial hemorrhage is deterrent to the uptake of thrombolytic therapy. 50 year old male presented with headache and giddiness for the past 7 days. The patient was diagnosed with inferior wall myocardial infarction 7 days back and was thrombolysed following which he developed headache and giddiness. The patient is a known case of diabetes and hypertension and on regular treatment. On examination the patient was drowsy and disoriented and blood pressure was 170/100mmhg. CT brain was advised which revealed intra cerebral hemorrhage originating from caudate nucleus. Bilateral frontoparietal subarachnoid hemorrhage. The patient was given antihypertensives, statins, neurotropic vitamins and oral hyoglycemic agents. Coagulation profile was monitored. Marked improvement was seen in the patient following treatment.

Keywords: STEMI (ST Segment elevated myocardial infarction, TPA (tissue plasminogen activator.

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ISSN: 0975-8585

INTRODUCTION

50 year old male presented to the casualty with complains of headache and giddiness for the past 7 days. The patient complained of left sided chest pain since last night he had history of palpitation and dyspnea. There was no history of radiation or fits. There was no history of blurring of vision. The patient was diagnosed with ischemic heart disease a week back. The patient was admitted to Meenakshi Medical College and Hospital on 20/8/12 complaining of left sided chest pain which started at 3 a.m. early morning. ECG revealed an inferior wall myocardial infarction for which he was thrombolysed. The next day following thrombolysis, the patient developed severe headache and giddiness. There was no history of vomiting or loss of consciousness. CT scan was advised which revealed intra cerebral hemorrhage originating from caudate nucleus. Bilateral fronto parietal subarachnoid hemorrhage. The patient is a known case of diabetes and hypertension for the past 3 years and on regular treatment. There is no history of bronchial asthma /drug allergy /epilepsy. The patient bowel and bladder habits are normal. The patient has a history of tobacco snuffing since childhood. There are no other adverse habits. On examination the patient was drowsy, disoriented, febrile, 101°F. There is no pallor, icterus cyanosis, clubbing, lymphadenopathy, pedal edema. His blood pressure was 170/100mmhg and pulse rate was 96/min. His capillary blood sugar was 220mgs/dl. His systemic examination was normal. Central nervous system was difficult to examine the case since the patient was drowsy, disoriented and not cooperative.

The patient was followed up and the necessary treatment and investigations were done. Complete blood count, renal function test, lipid profile, coagulation profile all were within normal limits. The patient was given antihypertensives, statins, neurotropic vitamins and oral hypoglygemic agents. Coagulation profile was monitored. On day 3, the patient improved, patient was conscious, oriented and afebrile. He was able to move all 4 limbs. Patients fall towards the right side on eye closure. Sensory system examination revealed all modality of sensation diminished below the thigh. There were no cerebellar sign. Romberg positive, sways on the opposite side. Ataxic gait. Plantar could not be elicited. On day 5, the patient was able to walk. Modality of sensation was recovered. Plantar was bilateral flexors.

DISCUSSIONS

A hemorrhagic transformation can happen spontaneously. This hemorrhagic risk is clearly increased by administration of heparin or thrombolytic agent. Hemorrhage is the most important complication of thrombolysis. Intracerebral hemorrhage is especially important with approximately 0.5% risk following STEMI thrombolysis. Given the short half-life of thrombolytic agents, by the time the diagnosis is made the biological effect of the drug may have abated. 40% of patients with follow-up [1]. CT scans showed evidence of ICH expansion and ongoing bleeding, suggesting a window of opportunity for treatment [2]. Bleeding following thrombolysis is complicated by numerous other agents that also contribute to a bleeding diathesis. Symptomatic ICHs usually occur within 24–36 h after thrombolysis, and ICHs that occur after 36 h are considered unrelated to tissue plasminogen activator (tPA) [3,4]. Post-thrombolysis ICH can be classified based on radiographic criteria alone or on the combination of a clinical change in a patient's neurological status in conjunction with evidence of ICH on brain imaging studies. Both approaches have strengths and weaknesses. Use of a classification scheme incorporating clinical changes (i.e. symptomatic versus asymptomatic ICH) is subject to imprecision given the fact that observed neurological changes may or may not be causally related to visualized ICH, and the criteria used to establish a change in neurological status may be variable. On the other hand, ICH associated with neurological deterioration (SICH) may be most relevant to patients and physicians as this is directly related to an observable clinical change. Radiographic criteria for defining ICH may be more objective and reliable, but may have less direct clinical relevance [5,6].

CONCLUSION

ICH after thrombolysis is a complex and heterogeneous phenomenon, which involves multiple demographic, clinical, biological and haemodynamic parameters. In order to minimise the risk of thrombolysis related symptomatic ICH, careful attention must be given, and a strict protocol for the control of elevated blood pressure is needed during the first 24 h following thrombolysis.

ISSN: 0975-8585

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