

**PP097 'DOWN AND  
BROKENHEARTED' A CASE OF  
CALCIUM CHANNEL BLOCKER  
TOXICITY**

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**INTRODUCTION**

Secondary myocardial infarction can be one of CCB toxicity manifestation due to profound hypotension and decrease myocardial perfusion.

**CASE DESCRIPTION**

A middle-aged man presented to ED complaining of central chest pain associated with dizziness, palpitation and profuse sweating 2 hours after ingesting 30 tablets of multiple antihypertensives (T. Felodipine 10mg, T. Valsartan 80mg, T. HCTZ 12.5mg) after had an argument with his wife.

Patient was hypotensive (BP 73/40 mmHg) however other physical examination was unremarkable. His first ECG showed ST elevation in V1-V6.

Patient was given IV Calcium Gluconate 3gm, IV drip bolus 20mls/kg/hour. High dose insulin therapy was started together with IV Noradrenaline infusion. Double antiplatelet and subcutaneous clexane was given

Patient condition improved with cessation of chest pain and repeated ECG showed resolution of ST elevation. Patient was admitted for observation and discharged well after 3 days.

**DISCUSSION**

Dihydropyridine CCBs overdose will result in reduced systemic vascular resistance due to selective effect to blood vessel. Co-ingestion with other antihypertension agents may exacerbates symptoms and lead to profound hypotension and cardiogenic

shock and may result in secondary myocardial infarction. Aggressive treatment to correct the hypotension will improve myocardial perfusion and demand.

High dose insulin therapy (HIET) is a treatment for severe CCB toxicity requiring inotropic supports. HIET may overcome metabolic starvation resulting from CCB toxicity by increase cardiac contractility and improved myocardial function without increasing oxygen demand.

**CONCLUSION**

Correction of hypotension is crucial in treatment of secondary myocardial infarction induced by CCB overdose. Early initiation of HIET together with other conventional therapy will improve the outcome.