**Elderly Woman with Cerebrovascular Accident and Refractory Arrhythmias**

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**Abstract**

Fatal bilateral cerebro-vascular accident with variable atrio-ventricular blocks, atrial fibrillation and refractory tachy-arrhythmias in a previously healthy 75-years-old hypertensive female is presented.

**Introduction**

It is well known that the patients who have cerebro-vascular accident have an increased incidence of electrocardiographic abnormalities due mainly to the association of arteriosclerotic cerebral and coronary artery disease, which have common risk factors. Those patients who had massive stroke particularly intracerebral or subarachnoid haemorrhage not only show electrocardiographic evidence of arrhythmias and coronary disease but also mild to moderate rise of serum cardiac enzymes and post mortem evidence of myocardial damage.1-4

**Case Report**

A 75-years-old female, a known hypertensive with chronic duodenal ulcer, on intermittent medical treatment during last 10 years was admitted to the medical ward for giddiness, vomiting and palpitation. She was diagnosed as a case of atrial fibrillation with slow ventricular rate due to ischemic heart disease and discharged with the advice to take beta-blockers; ACE inhibitors along with antacids and proton pump inhibitors. Seven days later she was readmitted. On examination she was afebrile, conscious, nonicteric with no significant lymph node enlargement, JVP normal, carotid pulsations were equally palpable without bruit, peripheral pulsations were normal, no purpuric spots seen. Her pulse rate varied from 40-50 to 170-180 per minute and blood pressure from 95/65 to 170/120 mmHg. On CVS examination apex beat was felt at normal site, no thrill; heart sounds were irregular with no associated murmur. Her Hb was 10.9 gm/dl, TLC 10200/cm, Platelet count 2,56,000/cmm and peripheral smear was normal. Blood glucose was 110 mg/dl, urea 40 mg/dl, creatinine 1.0 mg/dl, serum sodium 130 mEq/l, and potassium 3.9 mEq/l. Her serum bilirubin was reported 1.3 mg/dl with indirect bilirubin 0.9 mg/dl and SGPT 25 u/dl, serum LDH was 465 u/dl; CK on admission was 92 iu/dl and on next day it was 325 iu/dl with CKMB 40 iu/dl. She received IV nitro-glycerine, beta-blockers, ACE inhibitors, alpha methyldopa, IV mannitol and amiodarone.

Electrocardiography and continuous cardiac monitoring revealed on different occasions during initial 2 days of hospitalisation, showed heart rate 50-60/min → junctional rhythm → complete heart block → junctional tachycardia → sinus rhythm → atrial fibrillation with fast ventricular rate → persistent supra-ventricular tachycardia (heart rate >180/min). While during her previous admission ECGs were showing slow atrial fibrillation; P wave inversion in lead II, III and avF, 1° AV block (PR interval 0.24 seconds), suggestive of low atrial rhythm (coronary sinus rhythm). 2-D electrocardiography during present and previous admissions was reported normal (no chamber hypertrophy, no structural and blood flow abnormality across valves, no clots or growths in chambers and no regional wall motion abnormality). She developed generalised tonic and clonic convulsions, left hemiparesis, and coma. Subsequently, she developed flaccidity of limb muscles; non-reacting bilateral constricted pupils and absent plantar reflexes; exhibited intermittent decerebrate posture. Fundus oculi examination was normal. T3, T4, TSH levels were within normal range.

CT head showed large cerebral infarction involving left temporal, parietal and frontal lobes, left internal capsule, basal ganglion areas and multiple sub acute areas of infarction in right frontal, temporal, parietal lobes and right internal capsule along with diffuse cerebral edema, suggestive of bilateral internal carotid artery atherosclerotic disease. Her respiration became irregular, shallow with prolonged pauses of apnoea; was kept on ventilator support. During ventilator support her blood pressure remained high (>170/120 mm Hg); heart rate was more

**Fig. 1**: Contrast cranial CT (left) showing acute large cerebral infarct in fronto-parieto-temporal lobe, internal capsule and basal ganglion region. Multiple subacute infarcts noted in right cerebral hemisphere

**Fig. 3**: ECG showing low atrial rhythm (coronary sinus rhythm) and prolong PR interval (0.24 sec. 1° AV block) in leads II, III and aVF.
than 180 beats/min. She received phenytoin sodium, diuretics, antibiotics and other supportive measures during next five days. Her general conditions did not permit further investigations. She had no improvement in heart rate, systolic blood pressure fell down to <80mmHg, unresponsive to vasopressors; remained unconscious without own respiratory effort. The patient died after 7 days of intensive care with terminal event as persistent bradycardia and refractory hypotension.

**Discussion**

It is well known that patients with acute cerebro-vascular accident have a higher incidence of electrocardiographic abnormalities and arrhythmias. In a study of 100 patients of cerebro-vascular accidents (72 of cerebral thrombosis, 12 of cerebral haemorrhage, 10 of subarachnoid haemorrhage and 6 of emboli), 90 had electrocardiographic abnormalities within 72 hours after admission. Patients who died of cerebro-vascular accident had two to five fold higher incidence of electrocardiographic abnormalities, suggestive of myocardial infarction, atrial fibrillation and conduction defects than those who survived. A coincidental acute cardiac abnormality in a stroke patients is a direct consequence of neurological lesions. Oppenheimer postulated right sided dominance for sympathetic effects. Asymmetry in brain function influences the heart through ipsilateral pathways. There is association between right hemisphere stroke and supraventricular tachy-arrhythmia and left hemisphere stroke and arrhythmia of ventricular origin. Left insular cortex, amygdala and lateral hypothalamus are sites of cardiac representation and sympathetic control. Catecholamine stress is characterised by copious release of norepinephrine (from cardiac beta receptor sites) and steroids during CNS lesions like ischemic and hemorrhagic strokes or after tonic & clonic convulsions. It is responsible for neurogenic hypertension, pulmonary edema and myocardial injury; which is due to coronary vasoconstriction and cerebrogenic cardiac arrhythmias. Progressive rise in serum cardiac enzymes, variation of blood pressure, myocardial infarction, arrhythmias, conduction defects and left ventricular wall motion abnormalities are indirect observed effects.

Patients with symptomatic atherosclerotic coronary artery disease may have acute carotid artery disease and patients with symptomatic carotid disease may have acute coronary disease. After presentation with threatened stroke, patient may be at higher risk of myocardial infarction than for stroke. Moreover, the presence of coronary artery disease adversely affects survival in stroke patients.

This elderly hypertensive patient probably had atherosclerotic disease of carotid arteries along with hypertensive cardiovascular disease of non-manifesting nature. After recent episode of atrial fibrillation and variations in blood pressure, had deleterious ischemic effect on cerebro-vascular and cardiovascular systems. She presented in second admission with tachy-brady arrhythmias, fluctuating blood pressure, generalised convulsions, respiratory distress, coma and flaccid state; which ultimately led to death. Her electrocardiographic abnormalities and associated cerebral stroke may be viewed as due to the interacting processes viz; (a) underlying atherosclerotic and hypertensive cardio-vascular ischemic changes. (b) Sympathetic overflow during stroke leading to vasoconstriction, ischaemia, arrhythmia, depolarisation changes in myocardium and myocardial necrosis precipitated by (a) and (b) or both.

**Conclusion**


**Acknowledgement**

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**References**

The 3rd edition of this annual weekend course will be conducted at Lonavala from Friday 5 to Sunday 7, March 2010. Registration fees of Rs.2000/- will cover transport to and from Mumbai or Pune, 2 nights (twin-sharing) stay with full board at a 3- or 4-star resort and course material. For brochures please contact:

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**Book Review**

**Principles and Practice of Nuclear Medicine and Correlative Medical Imaging**  
by RD Lele  
Jaypee Brothers, Rs.995/-

Prof Lele is a clinician who has seen bed to benchside medicine very closely over last 5 decades. He has compiled an excellent textbook of nuclear medicine with other modern medical imaging like PET and SPECT with even MRI as well. His book has a vast array of chapters from radiopharmaceuticals, radionuclide to radioisotopes. There is chapter devoted to computer-assisted medical imaging. The core content of the book is correlative medical imaging in modern clinical specialties of neurology, psychiatry, endocrinology, thyroid, metabolic syndrome, cardiology, nephrology, pulmonary, hepatogastrointestinal as well as hemato- oncology. Infection and orthopedics is also covered. The classic mind body soul connect via nuclear imaging is an eye opener. The style is lucid and has world class case illustrations. Its a must for all physicians who keenly use imaging in diagnosis of disease pathologies.

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