# A Case of Inferior & Right Ventricular Infarction

The epidemic of cardiovascular disease, especially IHD is emerging in developing countries and the incidence continues to rise. India is no exception. It is estimated that up to three quarters of mortality in developing countries results from various non-communicable diseases and coronary artery disease tops the list surpassing even infectious diseases. As a consequence, more and more patients of IHD with their complications are hospitalised and subjected to a battery of investigations.

Pathologically, it is generally accepted that Myocardial Ischemia, and its extreme consequence – acute myocardial infarction, can result from a transient or a permanent disproportion between myocardial oxygen demand and coronary artery blood supply. Ischemic heart diseases can result from encroachment on the coronary artery lumen by disease of the arterial wall (arteritis), intraluminal obstruction (atheroma or embolism), or an excessive increase in myocardial oxygen demand exceeding the ability of the normal coronary arterial system to supply the needed blood (aortic stenosis). Of these, Atherosclerotic Coronary Artery disorders form the major proportion of cases in our OPD.

When we study the genesis of these disorders, it becomes obvious that the disease spectrum has an evolutionary dimension. It starts from a functional state and ends at an organic level (atherosclerotic plaque) with its secondary symptoms. Earlier it was accepted that the incidence of Myocardial Infarction increases rapidly with age. But today there is an emergence of Coronary artery disorders in the young (< 40 yrs). The implications of IHD in young patients go beyond prognosis, as repercussions on the entire family structure and community commonly ensue with a growing economic and social burden. It is apparent that there is a need to understand better the potential to return to work, the degree of symptoms to be expected vis-a-vis homoeopathic therapeutics in these patients.

This prompted the National Academy of Homoeopathy, India, to open a separate Homoeopathic Cardiological Cell at its Central Secretariat at Nagpur, where detailed homoeopathic study in this specific area can be undertaken.

Hereunder, I present one case of Subendocardial Inferior and right Ventricular Infarction from our records.

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**CASE:**

Ma X, 22y, studying in B Sc, was brought at Shaad at around 2 am on 14/03/02 with:

1. Chest pain – sudden onset, Retrosternal, localized dull aching.
2. Vomiting – twice, consisted of only food, non projectile, No nausea
3. Prostration ++ with cold sweat +
4. Was anxious, restless & persistently wished to sit in spite of having no breathlessness.
5. Disliked being covered (feet & hands were cold).

All complaints developed with increasing intensity within 1-2 hrs, while he was preparing for his exams. No such similar complaints reported in the past. No H/O loose motions, palpitation, vertigo, abdominal pain, etc except H/O active bleeding piles since 4 days.

O/E Well built: General condition not satisfactory; Restless & cold.

Pulse-Reg 130/min, synchronous, low volume

BP 90/60 mm Hg, No Oedema feet, JVP – Not raised but HJR positive.

No pallor/Icterus/clubbing; Central Cyanosis+. Face pale, cold with profuse sweat all over, CVS-HS 1st muffled, P2 loud, No S3 gallop RS-RR 28/min thoraco-

abdominal, Breath Sounds Vesicular. No Rales. PA – soft, Non-tender, Liver /Spleen– Not Palpable, Kidneys-not ballotable, No h/o Ascitis, sounds+ PR – Grade 2 Haemorrhoids ++. CNS-NAD

On this clinical presentation, Pt was hospitalised in the ICU. Taking his age into consideration, a possibility of Severe Gastritis/ Oesophagitis/ Acute Pancreatitis and Angina were considered. An ECG established the diagnosis -Acute Inferior Wall with Right Ventricular Subendocardial Infarct with Early Right Ventricular Failure with Hypotension. Carbo-veg, Cactus, Aconite and Actea were considered but the symptom totality pointed towards Camphor.

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|---|---|---|
| 14-03-02<br>2.10 am | ECG-ST depression (2mm) II, AvF, (4mm) rV4 with T inversion sinus tachycardia<br><br>Blood sent for investigation, TPR BP, ECG - 4 hrly | *Camphor* 30 x 10 min<br>POPosition, O2 inhalation<br>*Amyl-nitr Q* nebulizer ½ h<br>IV DNS (dose titrated) |
| 2 hrs after | Pt quiet, restlessness >, chest pain>>, Heavy feeling chest ++, passed urine 50 cc, Afebrile, P-110/m, BP 100/64 | *Camphor* 30 x 2 hrs<br>O2/*Amyl-nitr* SOS |
| 10 am | GC fair, Pt settled, Troponin T+, S CPKMB –47 I U/L, SGOT-56U/L, urea-32 mg/dl. S, Cr 1.4 mg/dl | Ct same<br>Ct. Ringer Lactate |
| 4 pm | Restlessness, prostration >, No vomit, No cyanosis. BP 110/70 mmHg, Electrolytes-WNL | Omit *Amyl-nit*<br>Rest Ct all |
| 9.10 pm | Retrosternal Chest pain relapsed < lying had to sit x 5 min, feeling of weakness++, restlessness +++, BP 80 mm Hg Systolic, No Fresh ECG changes | IV NS@ 20 Dr/min<br>*Camphor* 200 Fract Ds |
| 11.30 pm | Chest pain >, restlessness +, P-100/mm Hg, BP 100/70 mmHg | Ct all |
| Next day | Pt >, did not sleep well, No chest pain, dyspnoea Palpitation, Vomiting Urine 800 cc/24 hrs, P 100/min, Reg; BP 114/80 mm, JVP-NR ECG-IWM1 (Non Q wave) | *Sac lac* x 1 hrly<br>Omit IV fluids<br>Light diet |
| 16 March | Sleep>, GC fair, Chest pain >>, Passed soft stool 5 am, Urine 1150/24 hrs, Vitals stable, ECG-WNL | *Sac lac* TDS |
| 17 March | GC fair, no major comp, P-86/m, Reg; BP 120/76, ECG – WNL. Lipid profile-N | *Sac lac* TDS |
| 18 March | Patient>>, Stool/Urine N, Vital Stable, BP 110/80, RS-clear, HS pure, Adv ECHO/Coronary Angiography | Discharged on request<br>*SL* BD x 7 days |
| 22 March | 2D ECHO-Normal<br>Follow-up: No complaints | *SL* BD x 15 days |
| 1 mth after | Detailed life history | *Arg nit* 1M 1D |
| 13-07-02 | All Complaints much better,<br>Pt. is still under regular follow-up. | *SL* 1TDS |

**DISCUSSION**

*Camphor* was administered on the basis of exhaustion, debility, sudden onset, Restlessness, Anxiety, SIT INCLINATION to, Central Cyanosis, Face pale, cold with profuse sweat all over, did not want to be covered; *{Combined & Synthesis Repertories}*. Camph was started in low potency. Kent says: In the mental state there is anxiety and extreme fear; fear of persons,

of the dark, the patient is most troublesome patient to nurse; nobody and nothing suits. He cannot endure covering to warm his limbs though he suffers from cold. Robin Murphy gives the following Modalities:

> free discharges, sweat, thinking of it, drinking cold water.

< motion, night, contact, cold air, when half asleep, mental exertion,

*Camphor* covered the acute case totality so much so that only once after admission did he get an anginal attack with sudden hypotension. Of course since his lipid profile and coronary angiogram was normal which is a usual finding with young patients of Myocardial infarction – the basic pathologic mechanism for this attack was not atherosclerosis but a severe coronary vaso-spasm leading to an infarct (a functional manifestation indeed). This patient was given *Arg-nit* 1M 1 dose which was his constitutional remedy, to remove the hyper-irritability of the smooth muscles of the arterial wall.

2.) *Amyl-nit* nebulization was given concurrently for oppression of chest. Even IV fluids were extremely important because Inferior and Right Ventricular infarction patients, generally present with vomiting, since the Inf. Wall is close to the stomach and there is sudden hypotension due to over action of vagus nerves.

# A Case of Osteosarcoma

A 14 year-old girl, thin built, wheatish complexion and limping gait was brought to me by her parents for pain and swelling in right leg just below the knee since 2½ years. The swelling was reddish and shiny (ANGRY APPEARANCE). With my poor knowledge of clinical medicine I asked them is it a case of cancer. The parents replied that it had been diagnosed as a case of **Osteosarcoma of Rt Upper Tibia.**

The pain was constant and the girl could not give any character of the pain. Pains < exertion.

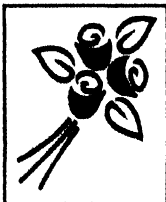
No Trauma. On repeated questioning, parents disclosed that she was very fond of dancing (Trauma all the same). She was very fond of fast paced music. Now she could not dance due to her complaints. (We can deduce that she must have suffered from minor traumas repeatedly due to dancing to fast paced music).

Althrough this time the child looked aloof and when I questioned about her behaviour, the parents replied that she was very irritable and rude to both of them and was very dominating.

Craving: sweets [3]

Both past and family history were not contributory. The intra-uterine history revealed that the mother had been under stress throughout 9 months of pregnancy and had nausea and vomiting till the end of pregnancy.

X-ray revealed multiple fractures in the affected bone.



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**ANALYSIS**

Looking back and reflecting, we can see that there is a split at the psyche level (Conceptually speaking: she was split from her family – as evidenced from her rude and irritable behaviour towards her parents) and at the same time we find fractures in the bone, splitting lesions themselves. This is a case showing a beautiful reflection of the Psychopathology. She was also very domineering.

We can find a lot of stress in the intra-uterine time, which resulted in the constant feeling of nausea and vomiting. A very marked concomitant in the case is her intense

craving for sweets.

**PRESCRIPTION**

Satisfied now that the drug picture had clearly emerged. I prescribed in this case

Rx : *Thyroidinum* 30, TDS for 3 days followed by *placebo*

**DISCUSSION**

When we look up the drug *Thyroidinum* in the Boger's "A Synoptic Key of Materia Medica', then the very first line reads thus "*splitting sensation; chest; ensiform appendix spine*". I would like to point out here what I learnt from Dr J N Majumdar that the first line of every drug in this priceless book is the soul (essence) of the drug. This splitting was evident in the case from psyche to the soma/pathology.

Though in the book Boger does not mention bones as an important sphere of action of *Thyroidinum* but when he mentions chest, ensiform appendix (or xiphoid process) and spine, we can see that they are all bony areas. So we can deduce that 'the bones' are also important sphere of action of the drug.

The knowledge of physiology shows that the thyroid is the prime gland to be affected under all the states of stress (right from conception through intra-uterine life.) We also know that this gland strongly controls the BMR of the entire body. Though we do not find in the scanty provings of this drug any mention of domineering attitude of the *Thyroidinum* personality. (If we conduct intense provings of this drug then this symptom will manifest along with many others, so for the time being we may call such symptoms as ornamental symptoms). Craving for sweets is known to be a key feature of *Thyroidinum*.

So this is the drug which covers the patient in all levels.

**FOLLOW UP**

I have been following the case now for a couple of months and she is still on *Placebo*. The intensity of pain and the swelling has decreased and she can move much more comfortably than before. The x-rays show that there has not been any increase in the size or number of fractures, which previously had been increasing steadily every month.

When we have prescribed thus (w)holistically in any case then all we have to do is to sit back and enjoy the pleasure of Prescribing for a long time as the patient marches on the path of recovery....

"The only certain means of success is to render more and better service than is expected of you, no matter what your task may be".

**- Og Mandino, 1923-1996,**

American Motivational Author, Speaker

"Hard work without talent is a shame, but talent without hard work is a tragedy".

**-Robert Half,**

American Businessman,

Founder of Robert Half & Associates

Only choose in marriage a woman whom you would choose as a friend if she were a man – Joseph Joubert

We all suffer from the preoccupation that there exists, in our loved one, perfection – Sidney Poitier