# Anaesthetic Management Of A Case Of Tetrology Of Fallot For Drainage Of Brain Abscess: A Case Report

I Naqash, B Ahad, J Zargar, A Kirmani, M Wani

**Citation**

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**Abstract**

Tetrology of Fallot is a leading cause of cyanotic congenital heart disease and is responsible for as many as 10% of all cases of congenital heart diseases. Complications like brain abscess always remain a danger. We are hereby reporting the anaesthetic management of such a case with good outcome.

## CASE REPORT

A 20 year old male, weighing 40 kgs presented in the department of Neurosurgery at Sheri-i-Kashmir Institute of Medical Sciences, Srinagar with the symptoms of headache and vomiting of one month duration. He gave a history of having Tetrology of Fallot, diagnosed few years back. In the hospital, the patient was evaluated and echocardiography revealed a large sub aortic VSD and 50% aortic override with ejection fraction of 0.8. ECG showed right axis deviation with right ventricular hypertrophy. X-ray chest was suggestive of right ventricular hypertrophy with oligemic lung fields. CT scan of the brain documented an abscess in the right fronto-parietal region. Biochemical investigations were normal except for raised haematocrit (PCV 54%). Coagulation profile was within normal limits. On examination, patient was conscious, oriented but ill looking. The pulse was 88 per minute and the blood pressure 110/70 mmHg. Cyanosis and clubbing of fingers was present. Examination of cardiovascular system revealed parasternal heave with a loud pansystolic murmur and accompanying thrill in the suprasternal region. Other systemic examination was normal.

In presence of an infective focus and in view of the urgent nature of surgery the patient was posted for drainage of abscess under general anaesthesia. He was put on antibiotics, anticonvulsants and anti-edema therapy. Preoperative arterial blood gas analysis revealed desaturation with a Sao2 of 86% and Pao2 of 55 mmHg. The case was accepted for surgery under ASA physical status V and a consent was obtained.

In the operating room, an intravenous line was established with 18G medicut and the patient was connected to Datex Cardiocap monitor for monitoring of ECG, NIBP AND SpO2. A prophylactic dose of antibiotic, Ceftriaxone 1gm was given to provide prophylaxis against infective endocarditis. The patient was premedicated with glycopyrollate 0.2mg and after preoxygenation for 3 minutes anaesthesia was induced with morphine sulphate 5mg and ketamine 100mg followed by suxamethonium 100mg to facilitate endotracheal intubation. Anaesthesia was maintained with nitrous oxide in oxygen (50:50), vecuronium bromide 0.1mg per kg body weight and isoflurane (0.4–0.6%). Ventilation was controlled using a Bain's co-axial circuit to maintain slight hypocarbia. The left radial artery was cannulated for direct arterial pressure monitoring and ABG analysis intra and post operatively. CVP monitoring was done through a double lumen CVP catheter inserted into the internal jugular vein. The bladder was catheterized to monitor the urine output.

Intraoperatively, the patient received dilantin 100mg, dexamethasone 8mg, mannitol 40gm and xylocard 60mg to lower the intracranial pressure and make the brain lax for the surgeon. Right fronto-parietal craniotomy was performed by the neurosurgeon and an encapsulated abscess cavity was drained and completely removed. The surgery lasted for about two hours and throughout the procedure, the patient remained stable with SaO2 ranging between 92–96% and Paco2 between 30–33 mmHg. Normal saline and dextrose saline was used as maintenance fluid during surgery and about 300 ml of blood lost was replaced.

At the end of the surgery, the residual neuromuscular block

was antagonized with neostigmine 2.5mg and glycopyrollate 0.4mg followed by extubation, once the patient was awake and responded to verbal commands. Postoperatively, the patient was transferred to the surgical intensive care unit for overnight observation and after 24 hours was referred back to the neurosurgery ward with O2 saturation of 86%.

## DISCUSSION

Tetrology of Fallot (TOF) is a leading cause of cyanotic congenital heart disease and forms about 10% of total congenital heart diseases. It is characterized by a large ventricular septal defect (VSD), right ventricular outflow tract obstruction (Pulmonic stenosis), right ventricular hypertrophy and overriding of aorta. If left untreated about half of the affected patients die during the first year of life. Those who survive this period have reasonably good prognosis but present with the problems of preexisting hypoxia and cyanosis, polycythemia leading to thrombotic complications, coagulopathies, occasionally CHF and perioperative cyanotic spells [1]. Polycythemia associated with chronic hypoxia causes hypercoagulability and thrombosis. Right to left shunting that bypasses the filtering of pulmonary capillaries is associated with a higher incidence of systemic infections such as brain abscess. Non-cardiac surgery in these patients is extremely hazardous.

The amount of shunting of blood through VSD in TOF is determined by the ratio of the systemic vascular resistance (SVR) to pulmonary vascular resistance (PVR). As SVR is increased, right to left shunting decreases. To a smaller extent change in PVR will also have a reciprocal relationship on the pulmonary blood flow, although the primary factor governing pulmonary blood flow is the presence of infundibular or valvular pulmonary stenosis. The greatest perioperative concern is the development of cyanotic spells due to spasm of the hypertrophied pulmonary infundibulum. Both, tachycardia and increased myocardial contractility can lead to infundibular spasm. Another mechanism for such hypercyanotic episodes is decreased SVR increasing right to left shunting through VSD. Systemic blood pressures less than 60 mmHg can trigger hypercyanotic episodes [2]. Management of anaesthesia for the patient with TOF requires a thorough understanding of the events and drugs that can alter the magnitude of right to left shunt which itself alters the pharmacokinetics of both inhaled and injected drugs [3].

An anaesthesiologist's concern for a case of TOF with brain abscess include:

- Hypoxia
- Coagulopathies
- Lax brain

Hypoxia leads to hyperviscosity of blood and coagulation abnormality [4]. A haematocrit above 65% requires phlebotomy. If coagulation abnormalities are present, use of NSAID are avoided and platelet concentrates should be available perioperatively. In our patient the PCV was 54% and did not have deranged coagulation profile.

Major goals of anaesthesia in TOF are to maintain systemic vascular resistance, minimize pulmonary vascular resistance and provide mild myocardial depression. Most of the patients have a dynamic right ventricular outflow obstruction which may be worsened by sympathetic stimulation during anaesthesia [5]. The optimal induction regimen for general anaesthesia in TOF should aim to improve arterial blood oxygen saturation (Sao2) and to maintain cardiovascular stability [6]. Both, intravenous and inhalational drug regimens have been used successfully for induction of anaesthesia in TOF to maintain systemic oxygenation and haemodynamic variables. Induction of anaesthesia itself using a variety of techniques have been shown to markedly increase arterial saturation. This induction related increase in arterial saturation probably results from higher systemic venous oxygen saturation due to high oxygen concentration plus decreased oxygen consumption with induction of anaesthesia and muscle paralysis [7,8].

Systemic venous blood with higher oxygen saturation is shunted in the systemic circulation, decreasing the degree of hypoxemia seen. However, systemic vasodilatation may occur during general anaesthesia and exacerbate the left to right shunting and intensify the resultant hypoxia. With the use of opiate technique, oxygen saturation levels are well maintained and may actually improve during induction, intubation and surgical stimulation even in cyanotic children [9]. We used a combination of morphine and ketamine in our case for induction of anaesthesia which actually improved arterial oxygenation and maintained it between 92-96% intraoperatively. Ketamine has been found to be the excellent induction agent in such cases as it improves the oxygenation by decreasing the right to left shunt as a result of increase in systemic vascular resistance [10].

Lax brain is essential from surgical point of view. Hypocarbia and diuretic therapy are usually employed.

Careful use of diuretic can avoid hypovolumia and maintain right ventricular output. Mannitol in a small dose is ideal as it reduces blood viscosity also. Frusemide induces hypovolumia to a greater extent [11]. We avoided hypovolumia by maintenance of intravascular volume.

## CONCLUSION

In conclusion, aggressive perioperative management has resulted in good outcome of this patient.

## CORRESPONDENCE TO

Dr. Imtiaz Naqash
Additional Professor,
Anaesthesiology & Critical Care
Sheri-i-Kashmir Institute of Medical Sciences,
Srinagar – 190011, Kashmir, (J&K), India.

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## Author Information

**Imtiaz Naqash, MD**
Additional Professor, Department of Anaesthesiology & Critical Care, Sher-i-Kashmir Institute of Medical Sciences

**Basharat Ahad, MD**
Senior Resident, Department of Anaesthesiology & Critical Care, Sher-i-Kashmir Institute of Medical Sciences

**Javed Zargar, MD**
Associate Professor, Department of Anaesthesiology & Critical Care, Sher-i-Kashmir Institute of Medical Sciences

**Altaf Kirmani, MS, M.ch**
Additional Professor, Department of Neurosurgery, Sher-i-Kashmir Institute of Medical Sciences

**M. Afzal Wani, MS, M.ch**
Professor, Department of Neurosurgery, Sher-i-Kashmir Institute of Medical Sciences