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Gupta Ajay Kumar[1], Tu Rong Fnag[1], Tang Ying Mei[1]* and Tong Yu Yun[2]

[1]Department of Gastroenterology and Hepatology, Second Affiliated Hospital of Kunming Medical University, Kunming city, Yunnan Province, PR China
[2]Department of Radiology, Second Affiliated Hospital of Kunming Medical University, Kunming city, Yunnan Province, PR China

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***Corresponding author:** Dr. Tang Ying Mei, Professor, MD, PhD, Department of Gastroenterology and Hepatology, Second Affiliated Hospital of Kunming Medical University, Kunming City, Yunnan Province, PR China, E-mail: tangyingmei_med@163.com

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Review Article

Hemodynamic and cardiopulmonary structural and functional changes post TIPSS: A review

Abstract

A deep understanding of the cardiopulmonary and hemodynamic changes from acute immediate to chronic long-term in the patients undergoing TIPSS is essential for providing a base for timely intervention to decrease the associated morbidity and mortality in the patients post TIPSS . The hemodynamic status of patients has shown a specific pattern of alternations in the different time periods post TIPSS. Most of the hemodynamic parameters which increases immediately after TIPSS i.e. including the CO, MAP, PcWP, RAP seems to normalize within 2 months to 1 year duration, the SVR seems to deteriorate further whereas the PPG is decreased immediately after TIPSS and remains constant. Sudden increase in PPG could be an indication of stent occlusion. The systolic function of heart remains unchanged but the diastolic function deteriorates which is indicated by decreased E/A ratio <1 with other cardiac structural changes i.e. increased Left Atrial diameter, Left Ventricle Hypertrophy and increased PcWP . TIPSS increases the incidence of Pulmonary Hypertension which is associated with a high mortality rate. In this article the pattern of changes in hemodynamic, cardiac functional and morphological status in patient's posts TIPSS are reviewed in depth on the basis of studies done in past 22 years.

Abbreviations

TIPSS: Transjugular Intrahepatic Portosystemic Shunt; PPG: Portal Pressure Gradient; PCWP: Pulmonary Capillary Wedged Pressure; MAP: Mean Arterial Pressure; CO-Cardiac Output; RAP: Right Atrial Pressure; LAD: Left Atrial Diameter; RAD: Right Atrial Diameter; RVDD: Right Ventricle Diastolic Diameter; LVDD: Left Ventricle Diastolic Diameter; LVEF: Left Ventricle Ejection Fraction; E/A- Peak velocity blood flow in early diastole (E wave) / Peak velocity of flow in late diastole (A wave).

Introduction

Transjuugular intrahepatic portosystemic shunt (TIPSS) implantation is an endovascular procedure which relieves the pressure in the portal circulation by creating a shunt through the hepatic parenchyma and hence redirecting the blood into the systemic circulation [1].

In 1968 Josef Rosch first described TIPSS but it was 1982 when Ronald Colapinto performed it for the first time in human patient. Freiburg, M. Rossle used this procedure for the first time as a treatment of portal hypertension in the year of 1988 [2-4] .

TIPSS is done for the management of Portal Hypertension complications [5]. The Established indications of TIPSS are acute variceal bleeding refractory to medical therapy and the recurrent variceal bleeding [5-7]. It has also been effective in the treatment of refractory ascites due to cirrhosis, Budd-Chiari Syndrome, Hepatic Hydrothorax and Hepatocellular Carcinoma [8-11].

It has been well known that liver cirrhosis has been associated with structural and morphological cardiac changes due to a baseline hyper dynamic status in cirrhosis i.e. expansion of plasma volume , decreased systemic vascular resistance (vasodilation), low arterial pressure leading to increased cardiac output with increased regional blood flow [12,13].

TIPSS has been effective in treating several complications of liver cirrhosis and portal hypertension, but is also associated with many short-term and long-term complications including hemodynamic and cardiopulmonary deterioration; hence knowledge about these complications becomes essential before performing TIPSS.

This article is the systemic review of immediate and long term impact of TIPSS on hemodynamics and cardiopulmonary

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changes on the basis of studies in past 22 years from 1994 A.D to 2016 A.D.

This review is specifically going to focus on the pattern of changes in PPG , SVR , PVR , MAP , CO, PcWP , RAP , LAD,RAD , RVDD , LVDD , LVEF % , Wall thickness and E/A ratio , from immediate i.e. 5 minutes post TIPSS to long term 1 to 5 years post TIPSS .

Hemodynamic changes as per durations post TIPSS

Portal Pressure Gradient: It's the pressure difference among the portal vein and inferior vena cava. The Portal decompression by TIPSS causes about 50 % decrease in the PPG immediately i.e. within 5 minutes to 30 minutes after TIPSS [14-23] and this drop in PPG is maintained in long term follow up from 30 days to 6 months [17,18] . The maintenance of decreased PPG is mainly due to Porto systemic shunt created during TIPSS. The studies showed PPG is independent of vasoactive substances like endothelins but the experimental occlusion of shunt drastically increased the PPG. Hence the rise of PPG in patients post TIPSS should raise a question on the patency of the shunt. [22,24]

Systemic Vascular Resistance: Studies showed SVR drops immediately after TIPSS and continues to drop further. The decreased in SVR further deteriorates the preexisting hyper dynamic circulation of the patients. [14,17,22]. The decrease in SVR is mainly due to cytokines including TNF alfa inducing excessive production of vasodilators like NO in splanchnic circulation. The produced vasodilators escape the degradation by cirrhotic liver and released in systemic circulation through the shunt. Hence, causing more severe vasodilation [25,26]. NO is a potent vasodilator and is also responsible for causing decrease in cardiac contractility [27-30].

Arterial NO is also produced due to hypervolemia and increased cardiac output causing stretch pressure on arteries [4,14]. The concept of central hypervolemia inhibiting RAAS and hence decreasing Angiotensin II and inhibiting catecholamine's release causing vasodilation doesn't seems so relevant because the experimental stent occlusion didn't show any rise in levels of angiotensin II , epinephrine or norepinephrine [23].

Pulmonary Vascular Resistance: Some studies found there was significant decrease in PVR immediately following TIPSS and it remains decreased till 1 to 6 months, but after 6 months the PVR start increasing and studies showed it increases more than the baseline line pre TIPSS PVR at 12 months [14,17,20].

Whereas studies done by *Hunker et al* and *Linden et al* found that the PVR rises immediately after TIPSS implantation and declines slowly and close to baseline pre TIPSS PVR in 1 month duration [21,23]. This variation in the PVR where some patients has decreased PVR immediately after TIPSS and some patients PVR increases is not well understood yet . It could be due to underlying cardiac, hemodynamic variations and severity of liver disease of the patients pre TIPSS [21]. Overall in spite of variations in PVR in early stages shown in different studies, at about 12 months duration all studies showed PVR increases significantly compared to that of pre TIPSS PVR. [20].

Pulmonary Artery Pressure: PAP significantly rises immediately after TIPSS measured within 5 to 30 minutes [14,17,21] and slowly tends to decline at 1-3 month period [14,17,20], but in long-term 1 to 5 years PASP was significantly higher that of Pre TIPSS PAP , hence increasing the incidence of secondary Pulmonary Hypertension in cirrhotic patients Post TIPSS [19]. The patients with this secondary Pulmonary Hypertension has the higher rate of mortality compared to the patients with Idiopathic Pulmonary Hypertension [27].

Mean Arterial Pressure: It is significantly elevated immediately after TIPSS i.e. 5to 30 minutes but the MAP comes close to baseline Pre TIPSS MAP or slightly above it in 1 -2 months [14,17].

Cardiac functional alterations as per durations post TIPSS

Cardiac Output: Immediately 5 to 60 minutes after TIPSS the CO increases significantly compared to baseline Pre TIPSS cardiac output and continues to remain elevated till 30 days [17,21], but at 2 months to 6 months duration the Cardiac output declines and comes close to the baseline Pre TIPSS CO [14,18,20].

Pulmonary Capillary Wedged Pressure: PCWP increases significantly immediately after TIPSS and this elevation was persistent in every follow ups of 24 hours, 3 days, 30 days and 2 month. PcWP at 30 days and 2 months we slightly less than that of 24 hours and 3 days studies but still were significantly elevated than the pre TIPSS PcWP [14,21,22].

Right Atrial Pressure: The RAP increases significantly immediately after TIPSS (30 minutes to 3 days) and seems to normalize within 1 to 2 months [14,17,21-23].

Right Atrial Diameter: RAD shows non-significant increment in follow ups post TIPSS [20].

Right Ventricle Diastolic Diameter: RVDD doesn't shows any significant change within 3 months post TIPSS but at 6 months to 12 months duration it decrease than the baseline Pre TIPSS RVDD and again in long term 1 to 5 years duration it normalize as the Pre TIPSS RVDD [19,20].

Left Atrial Diameter: The LAD seems to increase progressively with time as seen in studies which measured LAD in duration from 1 month to 12 months and 1 to 5 years [19, 20].

Left Ventricle Diastolic Diameter: LVDD increases significantly during the 1st month post TIPSS and the increment is maintained for longer duration as seen in 1 to 5 year study [19, 20].

Left Ventricle mass: The LV mass significantly increases at 6 months post TIPSS [18].

Inter ventricular Septum and Posterior wall thickness: The inter ventricular septum thickness and the poster wall did not show any significant changes at 1 to 5 years post TIPSS [19].

Left Ventricle Ejection Fraction: The EF % slightly increases

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immediately after TIPSS during the first week. The variations exist among the patients with normal effective volume status then those with decreased effective volume status. The patients with effective normal volume status has mild decrease in EF% during the 1st month, whereas in the patients with decreased effective volume status EF% seems to increase than that of baseline Pre TIPSS EF % [15,18]. In long term i.e. form the duration of 1 to 5 years both groups EF % remains normal and doesn't seem to deteriorate [19,20].

E/A ratio: The EA ratio which could be an early indicator of diastolic function, increase significantly for all group of patients during 1st 24 hours after TIPSS [18]. Among the patients with decreased effective volume status it seems to improve more significantly and remains above 1 [15]. But for other groups (i.e. Normal effective volume status patient or hypervolemia patients) its seems to deteriorate and remains below 1 as shown in 1 month to 12 months follow studies [15].

Summary

Most hemodynamic changes occur immediately and are transient whereas cardiac morphological changes occur late and permanent. Hemodynamic changes including cardiac output, Mean arterial Pressure, PcWP and Right atrial Pressure increases immediately after TIPSS and normalizes within 2 months to 1 year of time . But the SVR keeps deteriorating with time whereas the decrease in PPG is maintained mostly due to stent Patency. The morphological changes including the Left Atrial Diameter, LV diastolic diameter along with the LV mass seems to increase in long term in TIPSS patients which could be due to the physical stress after the persistent increased preload and decreased SVR maintaining the hyper dynamic status of cirrhotic patients even after TIPSS. [21,25,26]. The inter ventricular Septum and Posterior wall thickness didn't increased in long-term as mentioned above , The E/A ratio is deteriorated in long term which could be due to the LV hypertrophy and stiffness over time [18,19,23], the exception was the pre TIPSS decreased effective volume status patients who showed improvement in E/A ratio post TIPSS over time [15].

Hence it can be concluded that TIPSS deteriorates the diastolic function of heart as shown by decrease in E/A ratio, increase in Left Atrial diameter, pulmonary capillary wedge pressure and increase in pulmonary Vascular resistance whereas the systolic function remains unaffected as shown by maintained normal ejection fraction. The cardiac morphological changes occurs later usually after 1 month and mainly affects the left side of the heart causing increase in LAD and LV hypertrophy . TIPSS also increase the incidence of Pulmonary Hypertension which has high mortality rate but the exact mechanism is not understood yet. The variations found in immediate changes in PVR where some studies showed immediate decrease in PVR and some studies showed immediate increase in PVR after TIPSS, may have some correlation with post TIPSS increased PAP in some patients in long-term follow up, this needs further detailed study to find the cause and provide the guidance for more precise and effective therapeutic option for optimizing the cardiopulmonary status in cirrhotic patients Post TIPSS. Thus, it becomes essential to exclude the underlying Pulmonary Hypertension and other occult cardiac conditions in patients planned for TIPSS for providing the better cardiopulmonary outcome and hence decreasing the morbidity and mortality in patients post TIPSS.

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