Original Article

# Portal Hypertension: Effect of Early Splenic Artery Ligation on Platelets Count During Splenectomy

Mahendra S. Bhavsar, Hasmukh B. Vora, Lakshman S. Khiria, Venugopal H. Giriyappa

Department of Surgical Gastroenterology, Smt. NHL Municipal Medical College, Ahmedabad, India

Address for correspondence:
Dr. Venugopal H. Giriyappa, B-33, Doctors Quarters, V.S Hospital Campus, Ellis Bridge, Ahmedabad, Gujarat - 380 006, India.
E-mail: drvenugopalhg@gmail.com

## ABSTRACT

**Background/Aim:** Hypersplenism due to splenic congestion is observed in portal hypertensive patients. This study was done to know the change in platelets count following early ligation of splenic artery during splenectomy in patients with thrombocytopenia due to portal hypertension with a hypothesis that splenic decongestion results in increased platelets count; thereby platelet transfusion can be avoided. **Materials and Methods:** Patients with platelets count <100,000 per mm$^3$ due to portal hypertension were involved and we followed a protocol of ligating splenic artery first, followed by 30 minutes waiting period for splenic decongestion. Blood sample was collected at 5 and 30 minutes for the estimation of platelets count. **Results:** Significant rise in platelets was observed after 5 and 30 minutes of early ligation of splenic artery with mean rise being 23735 ± 15417 and 35085 ± 20458 per mm$^3$, respectively. The rise in platelets at 30 minutes was significant when compared with 5 minutes rise with mean platelets count being 91661 and 103070 per mm$^3$ at 5 and 30 minutes, respectively. The platelets rise was equal to 4 and 6 units of platelets concentrates, respectively. **Conclusion:** Early ligation of splenic artery during splenectomy for portal hypertension results in significant rise in platelets after 5 and 30 minutes. This method conserves platelets and avoids platelets transfusion and its complications.

Key Words: Early splenic artery ligation, platelets count, portal hypertension

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Massive splenomegaly is observed as a late complication of portal hypertension due to splenic congestion which results in features of hypersplenism.[1] Though it is more common in noncirrhotic portal fibrosis (NCPF) and extrahepatic portal venous obstruction (EHPVO) it is also seen in portal hypertension due to cirrhosis.[2] In congestive splenomegaly the spleen volume can be 10 times greater than normal, with 30-90% of platelets and 40% of erythrocytes located in the spleen.[3] This results in features of thrombocytopenia, leucopoenia and anaemia peripherally. It is known that splenectomy in these patients results in increased blood components peripherally and this is sustained for long time.[4] So in patients with platelets count <100,000 per mm$^3$ posted for surgery directed at prevention of variceal bleed we did a selective approach of ligating the splenic artery first and allowing the spleen to decongest for about 30 minutes. Blood sample was collected at 5 and 30 minutes of splenic artery ligation for the estimation of change in platelets counts which was compared with preoperative platelets count so that transfusion of platelets can be avoided which has its own side effects as with blood transfusion.

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## MATERIALS AND METHODS

From September 2009 to September 2011 about 54 patients underwent either esophagogastric devascularization with splenectomy (EGDS) or proximal splenorenal shunt (PSRS) for variceal bleed with portal hypertension due to NCPF, EHPVO or cirrhosis. All patients were operated electively. Among these, 34 patients who had features of thrombocytopenia due to portal hypertension were included in the study. Informed written consent was obtained from all patients and clearance for the study was obtained from the university board. Inclusion criteria were patients whose platelet count <100,000 per mm$^3$ before surgery for variceal

bleed with portal hypertension due to cirrhosis ($n$ = 14), EHPVO ($n$ = 11) or NCPF ($n$ = 9). Exclusion criteria were 1) patients who required blood products during the 30 minutes waiting period 2) patients with significant bleed (5 ml/kg during the 30 minutes waiting period). Intravenous fluids were given at 5 ml/kg during 30 minutes of waiting period.

5 ml of blood sample was drawn just before surgery for the estimation of prearterial ligation platelets count. We followed a protocol of locating and ligating splenic artery first, followed by a waiting period of 30 minutes to allow the spleen to decongest via splenic vein and other collaterals. Later EGDS or PSRS was conducted in the standard manner. During the 30 minute waiting period 5 ml of blood sample was collected at 5 minutes and 30 minutes to estimate the change in platelets count after arterial ligation due to splenic decongestion. During this waiting period no blood products were administered. Splenic weight was measured immediately after splenectomy.

### Statistical analysis

Statistical analysis was done using SPSS data analysis. The data was presented as mean ± standard deviation and median (range). The changes in platelets parameters before splenic artery ligation and after splenic decongestion at 5 and 30 minutes were analyzed using paired $t$ test. The correlation between splenic weight and platelets parameters was analyzed with Pearson correlation coefficient. $P$ value <0.05 was considered statistically significant.

## RESULTS

Among 34 eligible patients 21 patients underwent EGDS and 13 patients had PSRS. Mean age at surgery was 26.4 ± 13.9 years. The mean body weight of patients was 43.1 ± 12.75 kg. The weight of the spleen specimen ranged from 150 to 1200 gm with median weight being 550 gm. There was no significant correlation between the splenic weight and platelets count before surgery ($P$ > 0.05).

The mean platelet count before surgery was 68867 per mm$^3$ and after 5 minutes of arterial ligation was 91661 per mm$^3$ ($P$ < 0.001) [Table 1]. Likewise mean platelets count after 30 minutes of splenic decongestion was 103070 per mm$^3$ which was significant when compared with preoperative platelets count ($P$ < 0.002) [Table 2]. When the platelets count change at 5 minutes and 30 minutes were compared with each other, we observed the rise in platelets count at 30 minutes was significant ($P$ < 0.001) [Table 3].

The mean increase in platelets count was 23735 ± 15417 and 35085 ± 20458 per mm$^3$ at 5 and 30 minutes after arterial ligation, respectively. The median rise was 18300 (-2100 to 61100) and 30800 (-2700 to 73600) per mm$^3$ at 5 and 30 minutes after arterial ligation, respectively [Table 4].

Also significant positive correlation was observed between splenic weight and rise in platelets count both after 5 minutes and 30 minutes of splenic decongestion period with $P$ value <0.05 for both period.

## DISCUSSION

Hypersplenism is a common feature observed in late presentation of portal hypertension due to cirrhosis and also in EHPVO and NCPF. It is due to splenic congestion which results in thrombocytopenia, leucopoenia or decreased red blood cells. Sometimes all these blood components can be decreased in same patient. Jandl *et al*. and Cavalli *et al*. opined that in congestive splenomegaly, spleen volume can be 10 times greater than normal, with 30-90% of platelets and 40% of erythrocytes located in the congested spleen.[4,5]

The features of thrombocytopenia or anemia is expected to translate into a greater need for blood product transfusion, either platelets or packed red blood cells, before or at the time of surgery. Any blood products transfusion has its own risks. At the same time since the pathology is splenic congestion due to portal hypertension, whether the splenic ability of autotransfusion by decongestion can be attempted was the basis of our study. So we attempted early splenic artery ligation followed by waiting period of 30 minutes to allow spleen to decongest. Gazula *et al*. also reported similar study done on 28 patients of portal hypertension which included only EHPVO patients and they reported

**Table 1: Comparison between pre-arterial ligation platelets count and 5 minutes post-arterial ligation platelets count**

| Variable ($n$=34) | Pre-arterial ligation platelets count (mean and range) | 5 minutes Post-arterial ligation platelets count (mean and range) | $P$ value |
|---|---|---|---|
| Platelets count (per mm$^3$) | 68867 (45900-95100) | 91661 (57000-142000) | <0.001 |

**Table 2: Comparison between pre-arterial ligation platelets count and 30 minutes post-arterial ligation platelets count**

| Variable ($n$=34) | Pre-arterial ligation platelets count (mean and range) | 30 minutes Post-arterial ligation platelets count (mean and range) | $P$ value |
|---|---|---|---|
| Platelets count (per mm$^3$) | 68867 (45900-95100) | 103070 (69000-152000) | <0.002 |

**Table 3: Comparison between 5 minutes and 30 minutes post-arterial ligation platelets count**

| Variable (*n*=34) | 5 minutes Post-arterial ligation platelets count (mean and range) | 30 minutes Post-arterial ligation platelets count (mean and range) | *P* value |
|---|---|---|---|
| Platelets count (per mm$^3$) | 91661 (57000-142000) | 103070 (69000-152000) | <0.001 |

**Table 4: Change in platelets count at 5 and 30 minutes**

| Platelets count (per mm$^3$) | 5 minutes | 30 minutes |
|---|---|---|
| Mean (SD) | 23735 (±15417) | 35085 (±20458) |
| Median (range) | 18300 (−2100 to 61100) | 30800 (−2700 to73600) |

about the change in parameters of all blood components before and after splenic artery ligation.[6] They compared prearterial ligation parameters with the change in blood components after 30 minutes. Here in our study, patients with portal hypertension due to cirrhosis and NCPF were also involved. We observed for the change in platelets count after 5 minutes and 30 minutes of splenic artery ligation. Gazula *et al*. reported significant rise in platelets at the end of 30 minutes. They observed the median rise in platelet count of 19500 per mm$^3$ at the end of 30 minutes which is equal to platelet transfusion of at least 4 units of platelet concentrates in an adult. They also recorded a positive correlation between the splenic weight and platelet gain. We observed the rise in platelets count irrespective of the etiology of portal hypertension. But in this study we did not randomize the patients into cirrhotic, EHPVO and NPCF because of less number of patients in each group. There are no previous studies regarding the rise in platelets during the operative period without early splenic artery ligation to compare and we did not have a control arm to assess the same. Though our study was initially undertaken to ascertain the change in platelets count, we also observed changes in other blood components like hemoglobin and white blood cells. Furthermore, we are currently studying the change in blood components like red blood cells, white blood cells, platelets, hemoglobin and hematocrit after early splenic artery ligation in each subgroup of portal hypertensive patients at our institute, which needs more number of patients to be reported.

Here in this study, we observed a median rise of 18300 per mm$^3$ of platelets after 5 minutes of splenic artery ligation which was statistically significant and almost equal to platelet transfusion of 4 unit of platelet concentrates in an adult. Median rise at 30 minutes was 30800 per mm$^3$ which was also statistically significant and equal to nearly 6 units of platelet concentrates transfusion in an adult. Further, when the platelet parameters at 5 and 30 minutes were compared there was statistically significant platelet gain at 30 minutes which shows that waiting longer after splenic artery ligation allows more platelets to be autotransfused into the systemic circulation.

Other than the above study by Gazula *et al*. we were unable to find any other study on portal hypertension patients to know the effect of early splenic arterial ligation on platelets parameters. In their study on rats Sahin *et al*, reported that splenic vein ligation resulted in secondary hypersplenism and after two weeks one group of rats underwent splenic artery ligation, causing a rise in platelets compared to the control group where the splenic artery was not ligated.[7] In another study Bakovic *et al*, did repeated breath-hold apnoeas in trained divers, normal controls, and splenectomised patients to know the contribution of splenic contraction in circulating RBCs, leucocytes and platelets.[8] However, although there was significant increase in RBCs and leucocytes in the trained divers and intact subjects, there was no significant change in platelets concentrations in any of the groups. This may indicate the importance of blocking the splenic inflow, which results in increase in platelets count in blood.

This study demonstrated that early ligation of splenic artery and allowing the spleen to decongest for 30 minutes results in significant gain in platelets thereby avoiding the transfusion of platelets concentrates. Patients with platelets transfusion face the same risk of any other blood product transfusion and thereby risks of allergic reactions, viral or bacterial transmission, etc. can be avoided. In many centres, including our institute, patients with thrombocytopenia are transfused with at least 4 units of platelets before the beginning of surgery. In our study, platelets transfusion was avoided in all 34 patients with thrombocytopenia. However, this waiting period of 30 minutes extends the duration of anesthesia. As such, in high risk patients, particularly cirrhotic individuals, a minimum of 5 minutes waiting period allowing for splenic decongestion after early splenic artery ligation, could be justified. Though this risk is to be balanced, we did not find any complications among any of our patients including cirrhotics, either during surgery or in the postoperative period.

## CONCLUSIONS

Early ligation of splenic artery and splenic decongestion during splenectomy for portal hypertension results in significant increase in platelets count by autotransfusion. The rise in platelets count has significant positive correlation with the splenic weight. Though 5 minutes of splenic decongestion also results in significant rise in platelets, allowing 30 minutes of splenic decongestion results in maximum gain of platelets. This method avoids platelet

transfusion and its possible side effects. Further, randomized studies are required to demonstrate the change in other blood components in portal hypertension patients due to different etiology.

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