# Splenic artery embolisation for portal hypertention in children

Ila V. Meisheri, Paras R. Kothari, Anil Kumar, A. Deshmukh[1]


## ABSTRACT

**Background:** Bleeding from esophageal varices is one of the most common causes of serious gastrointestinal haemorrhage in children. We analysed our experience with the use of splenic artery embolisation and variceal sclerotherapy for bleeding oesophageal varices. **Patients and Methods:** Records of all patients treated for bleeding oesophageal varices caused by portal hypertension from 1998 to 2004 were retrospectively analysed. Patients were followed up for five years. **Results:** Out of 25 patients treated, ten belonged to sclerotherapy (group A), eight to combined sclerotherapy and embolisation (group B), and seven to only embolisation (group C). The patients were selected randomly, only two patients who had active bleed recently were directly sclerosed. The splenic artery was embolised at the hilum using steel coils in 15 patients with portal hypertension and hypersplenism. Follow-up findings showed decrease in splenic mass, varices, and hyperdynamic flow.

**Conclusion:** In spite of few patients and a short period of follow-up, our results pointed out that a serious consideration should be given to this procedure, as it slowed the sequel of portal hypertension and the complications associated with it. Patients who were embolised and followed up for five years had lesser rebleeds and complications than sclerotherapy patients.


**Key words:** Children, embolisation, portal hypertension, sclerotherapy, splenic artery

**DOI:** 10.4103/0189-6725.62854

## INTRODUCTION

Bleeding from esophageal varices is one of the most common causes of serious gastrointestinal haemorrhage in children. Its treatment continues to be a real challenge for paediatric surgeons.

Department of Paediatric Surgery, B. J. Wadia Children Hospital, and [1]Department of Radiology, K.E.M. Hospital, India

**Address for correspondence:**
Dr. Paras R. Kothari, 12, New Sailesh Society, V. P. Road, Vile-Parle (west), Mumbai-400 056, India.
E-mail: drparaskothari@rediffmail.com

Sclerotherapy was first reported in 1939 in a patient with extrahepatic venous occlusion;[1] still it became popular after 1980s only. Maddison[2] did first splenic artery embolisation in 1973. It has still not become popular as an alternative to surgery, inspite of having many advantages and few complications. Some of the advantages[3–9] of embolisation of splenic artery performed to elimination of the splenic hyperdynamic component in portal hypertension are: decrease in the function of splenic mass, preservation of venous outflow, and improve in peripheral cytopenia. All these, with preservation of transhepatic portal flow, help in slowing evolution of portal hypertension which in turn delays the requirement of shunt surgery in children.

## MATERIALS AND METHODS

This was a prospective study involving a total of 25 patients suffering from extrahepatic portal hypertension with bleeding from esophageal varices.

All patients had extrahepatic portal hypertension (bleeding in all cases due to portal vein obstruction). Five patients had abnormal fundic varices.

The patients were categorised into three groups [Table 1] depending on the clinical presentation at the time of presentation and compliance on follow-up.

Group A (control group) [Table 2] had ten patients who underwent sclerotherapy who had active bleeding and who were compliant.

Eight patients from group B [Table 3] underwent sclerotherapy + embolisation (patients were sclerosed once at the time of acute variceal bleed. Due to

**Table 1: Groups**

| Groups | No. of Patients | Procedure |
|---|---|---|
| A | 10 | Sclerotherapy |
| B | 8 | Sclerotherapy + Splenic artery embolisation |
| C | 7 | Splenic artery embolisation only |

**Table 2: Group A analysis**

| Age Year | Sex | Etiology | Platelet Count | | Splenic Size (Cms) | | Sclero-therapy Sittings | Comp-lication | Endoscopy (Varices) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Pre-op | Post-op | | | | | Pre-op | | Post-op | |
| | | | | | | | | | G | C | G | C |
| 1.6 | M | EHPHT | 700,00 | 750,00 | 8.4 | 8.6 | 6 | Nil | II-III | 3 | I | 1 |
| 11 | F | EHPHT | 570,00 | 580,00 | 11 | 11 | 7 | Nil | II | 3 | II | 1 |
| 6 | M | EHPHT | 115,000 | 110,000 | 12.6 | 12 | 7 | Bleeding | II-III | 2 | I | 2 |
| 10 | F | EHPHT | 810,00 | 800,00 | 10 | 10 | 8 | Expired | II-III | 3 | III | 2 |
| 10 | M | EHPHT | 720,00 | 730,00 | 13 | 12 | 6 | Nil | II | 2 | I | 2 |
| 7 | F | EHPHT | 780,00 | 800,00 | 12 | 13 | 4 | Nil | III | 3 | III | 2 |
| 6 | M | EHPHT | 680,00 | 700,00 | 9 | 10 | 4 | Nil | II | 2 | I | 1 |
| 5 | M | EHPHT | 820,00 | 880,00 | 13 | 13 | 8 | Nil | III | 2 | II | 1 |
| 6 | M | EHPHT | 108,000 | 102,000 | 11 | 11 | 8 | Bleeding | III | 2 | II | 2 |
| 2 | M | EHPHT | 970,00 | 950,00 | 9 | 12 | 7 | Nil | III | 3 | I | 2 |

noncompliant nature to follow the sclerotherapy regimen, we planned to embolise these patients).

Seven patients from group C [Table 4] were embolised (referred patients who were medically managed for one/two episodes of variceal bleed and were stable when they presented to our institute).

There were 19 males and six females with age ranging from 3–6 years.

Sclerotherapy was performed using 3% sodium tetradecyl sulphate diluted to 1:3 by normal saline. Scleroscants were injected once a week for first two sittings and then fortnightly till varices were obliterated. In one sitting, maximum of three sites were injected. Variceal obliteration was considered complete if no varices were seen or they were thrombosed. Gastric fundic varices were not injected. Maximum of eight sittings of sclerotherapy were done in a patient.

Patients were admitted two days prior to the day of embolisation/sclerotherapy. Sonography (to know splenic size and splenic arterial blood flow) and platelet count were carried out.

Embolisation was done by angiomanometric technique in all patients. A 7F balloon catheter was introduced into the hepatic vein through femoral vein to measure the hepatic vein free pressure and wedge hepatic venous pressure (WHVP). The catheter was deflated and left *in situ*. Above procedure was followed by transfemoral catheterisation of celiac axis leading to splenic artery using a preserved (cobra shaped) catheter. A guide wire

**Table 3: Group B analysis**

| Age/sex | Etio-logy | Platelet count | | Ultrasonography splenic size | | Doppler splenic arterial flow | | WHVP | | Endoscopy (Varices) | | | | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Pre op | Post op | Pre op cms | Post op cms | Pre op ml/sec | Post op ml/sec | Pre op | Post op | Pre op | | Post op | | |
| | | | | | | | | | | G | C | G | C | |
| 8Y/M | EHP HT | 520,00 | 160,000 | 13 | 17 | 162 | 95 | 29 | 20 | III | 2 | I | 1 | Nil |
| 7½Y/M | EHPHT | 310,00 | 170,000 | 14 | 14 | 140 | 80 | 28 | 20 | II-III | 2 | I | 1 | Back-ache |
| 10Y/M | EHP HT | 280,00 | 157,000 | 20 | 14 | 150 | 39 | 30 | 19 | III | 3 | I | 1 | Re-bleed. Mesocaval shunt done. |
| 6Y/M | EHPHT | 680,00 | 155,000 | 9 | 13.5 | 175 | 47 | 29 | 23 | II | 2 | I | 1 | Nil |
| 5Y/M | EHPHT | 410,00 | 217,000 | 13 | 11 | 182 | 29 | 30 | 20 | II-IIII | 2 | I | 2 | Nil |
| 4Y/M | EHPHT | 830,00 | 197,000 | 10 | 8 | 146 | 78 | 31 | 25 | III | 3 | I | 2 | Back-ache |
| 6Y/M | EHPHT | 570,00 | 150,000 | 14.2 | 11.5 | 140 | 62 | 29 | 21 | III | 3 | I-II | 2 | Nil |
| 7Y/M | EHPHT | 810,00 | 210,000 | 17 | 7 | 135 | 47 | 27 | 19 | II | 2 | I | 1 | Nil |

**Table 4: Group C analysis**

| Age/ sex | Etio-logy | Platelet count | | Ultrasonography splenic size | | Doppler splenic arterial flow | | WHVP | | Endoscopy (Varices) | | | | Complication |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Preop | Postop | Pre op cms | Post op cms | Pre op ml/sec | Post op ml/sec | Pre op | Post op | Pre-op | | Post-op | | |
| | | | | | | | | | | G | C | G | C | |
| 7Y/F | EHPHT | 730,00 | 158,000 | 20 | 11 | 170 | 52 | 31 | 19 | II | 2 | I | 1 | Fever |
| 7Y/F | EHPHT | 110,00 | 177,000 | 16 | 11 | 150 | 33 | 28 | 20 | I1 | 3 | I | 1 | Fever |
| 4Y/M | EHPHT | 370,00 | 124,000 | 17.2 | 16 | 140 | No signal Present | 29 | 20 | III | 3 | I-II | 1 | Back-ache |
| 5Y/M | EHPHT | 320,00 | 145,000 | 15 | 8 | 162 | 30 | 30 | 21 | III | 3 | I-II | 1 | Nil |
| 3Y/M | EHPHT | 400,00 | 165,000 | 16 | 10 | 155 | 37 | 30 | 19 | III | 3 | I | 1 | Nil |
| 5Y/M | EHPHT | 890,00 | 212,000 | 14 | 11 | 152 | 50 | 30 | 21 | III | 2 | II | 1 | Nil |
| 4 1/2Y/M | EHPHT | 730,00 | 187,000 | 13 | 10 | 136 | 48 | 28 | 19 | 11 | 2 | I | 1 | Re-bleed. Mesocaval shunt done. |

G - grade. C - column.

was inserted and the catheter was withdrawn, replacing it by a balloon catheter similar to that used in the hepatic vein. After inflating the balloon in the splenic artery WHVP was remeasured. Those patients who showed marked decrease in WHVP were considered ideal for splenic artery embolisation. Patients showing less decrease in the WHVP were also embolised. A 7F polytef catheter was substituted for the catheter in the splenic artery using a transfer guide wire. Through this catheter, two-to-three metallic coils were introduced into the hilum of the splenic artery. The WHVP was remeasured after the procedure. The balloon catheter in the hepatic vein was removed.

Efficiency and characterisation of obstruction were checked by an arteriographic examination immediately. After 15 days, Doppler ultrasonographic spleen examination was done to measure splenic arterial flow. Position of the coils was also confirmed. None of the coils had migrated into the splenic parenchyma. Ultrasonography was done to see percentage of splenic infarction by comparing the volume of low-density areas in spleen with entire splenic volume. Antibiotics (cefotaxime and amikacin) were given for five days. Patients were advised absolute bed rest for one day post procedure.

Successful embolisation[1–6] resulted in a decrease in splenic arterial flow, decrease in splenic size, and esophageal varices and increased platelets count. In all patients, development of hematological patterns was followed up (especially platelet count). No liver biopsy was performed in these patients.

## RESULTS

There were a total of 25 patients who were included in the study. There were 19 males and six females with age ranging from 3−6 years. Ten patients underwent sclerotherapy alone (Group A), while eight patients received sclerotherapy and embolisation (Group B), and seven patients received only splenic artery embolisation (Groupc C) [Table 1].

Platelet count was seen as the most sensitive indicator for outcome of this procedure, as seen in Table 3. On an average, the count rose by 112,000 in embolisation group; whereas, it remained unchanged in sclerotherapy group. Even after three years, the value of platelet count reached equilibrium higher than the initial values.

Periodic ultrasonography showed splenic size decreasing uniformly and effectively in group C [Table 4], five cases in group B [Table 3] and none in group A [Table 2]. On an average, the splenic size decreased by 5 cms in group C. Sonography never showed serious phenomenal damage of the splenic parenchyma, as the coils were placed only in the splenic hilum and thus the infraction was controlled.

Doppler ultrasonography showed decrease in splenic arterial flow to an extent of 1/3$^{rd}$ to 1/5$^{th}$ (i.e., 95 cms/sec on an average) of the original flow. It also showed that the coils placed in the hilum had not migrated.

The average WHVP in group B was 29.4 cms of saline and in group C it was 29.4 cms. Following occlusion of splenic artery, there was reduction in the average WHVP by 8.5 cms of saline in group B and 9.4 cms in

group C. Massive decrease in WHVP was indicative of high hyperdynamic splenic component.

Endoscopy was another mode of confirming embolisation to be as good as or better than sclerotherapy. Endoscopy done three months after embolisation showed decrease in variceal sizes. The grade and column of varices decreased respectively from III/II to II/I and 3/2 to 2/1. It was remarked to be the most effective in group C, suggesting high splenic blood flow component in extrahepatic portal hypertension (EHPHT).

All patients were followed up for a period of five years. In group A, there were two rebleeds nil a failure rate of 20%. In group B and C, one patient in each group had rebleed; both patients underwent Mesocaval shunt. One patient from group A died due to chronic liver failure. There was no mortality in group B and C. No patients from any group had chest complications. Two patients from group C had fever which is mentioned in the Table 4.

## DISCUSSION

Bleeding from esophageal varices is one of the most common causes of serious gastrointestinal haemorrhage in children. In half of the cases, portosystemic shunt is not possible because of complete block of splenoporto axis or smaller diameter of available splenic vein. Shunt surgeries in children less than two years is difficult. Therapeutic shunt surgeries traded one cause of death (haemorrhage) for another (liver failure) with only marginal improvement in survival. Recent advances in endoscopic sclerotherapy and embolisation have helped paediatric patients a lot.

Endoscopic sclerotherapy is designed to prevent bleeding by either thrombosing the veins or thickening of overlying mucosaSclerotherapy was done as an emergency, elective and prophylactic procedure.[10-12] (where higher-grade varices were present, patient compliance for follow-up was doubtful).

Embolisation is similarly designed to decrease inflow of blood in to the portal system,[8] thus decreasing varices and bleeding episodes. Embolisation was done in elective and postsclerotherapy patients only.[2,13–15] We have not done embolisation in emergency and prophylactic conditions.

Patients in our study presented at a mean age of 5.9 years, whereas the mean age of presentation in another series was 6 years.[16]

Patients with extrahepatic portal venous occlusion have gastric varices and a risk of bleeding from them after successful obliteration of esophageal varices is present. This was observed in 5% of the patients.[15] In our series, it was 20% in sclerotherapy and 6.5% in embolisation. All embolised patients had less rebleeding episodes.[17]

There is also an increase in the platelet count[5-7,14] (double in most cases). In our study, the average increase in the platelet count was 112,000 which helped prevention of rebleed episode. Explanation of such an occurrence lies in the fact that both the direct hematopoietic activity and probable humoral splenic regulation on the medulla were initially increased.[18] No other hematological variables were measured.

An average WHVP reduction of 10 cms saline was attained.[3] In our study, WHVP was measured by balloon before and after occlusion of the splenic artery. In all patients following occlusion of splenic artery, there was significant reduction in the WHVP up to 8.5 cms of saline in group B and 9.4 cms in group C.

Colour Doppler showed decrease in the splenic arterial blood flow postembolectomy.[19] In our study, the average splenic arterial flow decreased by 95 ml/sec. These observations support that in patients with splenomegaly and an enlarged splenic artery, increased arterial flow into the portal system may be causing dominant alteration in the pathogenesis of portal hypertension.[8,20,21]

Different materials (gelatin sponge, autologous clots, polyvinyl alcohol, and butyl-2-cyanoacrylate) have been used for embolisation of splenic artery.[2,4–7,14,15,17] Control of smaller particles used angiographically is less; Therefore, gastric and pancreatic necrosis can occur with reflux of such particles.[14] The chances of complications are reduced by nonusage of such small materials for embolisation. We found steel coils effective and safe when placed in the hilum of spleen, as they could not migrate. Controlled infarction of spleen also decreases the chances of complications.[2,16] Embolisation done by Y coils[4] can minimise the infarction in spleen thus decreasing incidence of splenic abscesses and splenic rupture[2,6] (due to massive necrosis of splenic tissue with liquefaction of necrotic debris).

Preoperative use of antibiotics and utmost care during procedure limited the complications in our series to fever and pain.[5,17] In group A of our series, one patient rebleeded after total obliteration of varices and another

patient presented with continued bleeding while on sclerotherapy. The patient who continued to bleed while on sclerotherapy was taken up for embolisation. There was one rebleed in each group B and C till a follow-up period of five years.

Our aim was to reduce portal pressure in patients of portal hypertension. Most of the surgeries are palliative in nature. By embolising the splenic artery the blood supply to spleen is reduced, in turn reducing the venous drainage to portal system from splenic vein. This reduces the pressure in the portal system, which is obvious and statistically significant. It has also brought down mortality, morbidity, and rates of complications.[5,22,23]

## CONCLUSION

Although having few cases and a short period of follow-up, the results show that a serious thought should be given to this procedure, using more number of patients and a longer period of follow-up. We feel splenic artery embolisation is a good treatment for variceal bleeding for elective cases, though in emergency, initial sclerotherapy still remains the first line of treatment. Once bleeding is under control, splenic artery embolisation can be done effectively as was shown in our series.

## REFERENCES


1. Crafoord C, Frencker P. New surgical treatment of varicose veins of the oesophagus. Acta Otolaryngol 1939;36:422-9.
2. Yoshioka H, Kuroda C, Hori S, Tokunaga K, Tanaka T, Nakamura H, *et al*. Splenic embolisation for Hypersplenism using steel coils. AJR Am J Roentgenol 1985;144:1269-74.
3. Zannini G, Masciariello S, Pagano G, Sanqiuolo P, Zotti G, Iaccarino V. Percutaneous splenic artery occlusion for portal Hypertension. A new mechanical technique for hypersplenism. Arch Surg 1983;118:897-900.
4. Yamauchi T, Furui S, Irie T, Kusano S. Partial splenic embolisation with Y shaped silicone particles. Acta Radiol 1994;35:335-9.
5. Shah R, Mahour GH, Ford EG, Stanley P. Partial splenic embolisation. An effective alternative for splenectomy for hypersplenism. Am Surg 1990;56:774-7.
6. Castaneda-Zuniga WR, Hammerschmidt DE, Sanchez. R, Amplatz K. Nonsurgical splenectomy. A J Roentgenol Nov 1977;129:805-11.
7. Zannini G, Masciariello S, Pagano G, Sangiuolo P, Zotti G, Iaccarino V. Percutaneous splenic artery occlusion for portal hypertension. A new mechanical technique for hypersplenism. Arch Surg 1983;118:897-900.
8. Vorobioff J, Bredfeldt JE, Groszmann RJ. Hyperdynamic splanchic circulation in portal hypertensive rat model. A primary factor for maintenance of portal hypertension. Am J Physiol 1983;244:G52-7.
9. Maddison FE. Embolic therapy in hypersplenism. Investig Radiol 1973;8:280-1.
10. Terblanche J, Krige JE, Bornman PC. The treatment of esophageal varices. Annu Rev Med 1992;43:69-82.
11. Maksoud JG, Goncalves ME, Porta G, Miura I, Velhote MC. The Endoscopic and surgical management of portal hypertension in children. Pediatr surg 1991;26:178-81.
12. Heaton ND, Howard ER. Howard Complication and limitations of injection sclerotherapy in portal hypertension. Gut 1993;34:7-10.
13. Owman T, Lunderquist T, Alwmark A, Borjesson B. Embolisation of the spleen for the treatment of splenomegaly and hypersplenism in patients with portal hypertension. Invest Radiol 1979;14:457-64.
14. Levy JM, Wasserman P, Pitha N. Presplenectomy transcatheter occlusion of splenic artery. Arch Surg 1979;114:198-200.
15. Gianturco C, Anderson JH, Wallace S. Mechanical devices for arterial occlusion. Am J Roentgenol Radium Ther Nucl Med 1975;124:428-35.
16. Wholey MH, Chamorro HA, Rao G, Chapman W. Splenic infarction and spontaneous rupture of spleen after therapeutic embolisation. Cardio Radio 1978;1:249-53.
17. Brandt CT, Rothbarth LJ, Kumpe D, Karrer FM, LillyJR. Splenic embolisation in children: Long term efficacy. J Pediatr surg 1989;24:642-5.
18. Giorgio B, Giuseppe Z, Stephano M, *et al*. Percutaneous splenic artery occlusion for portal hypertension. Arch surg 1983; 118: 897-900.
19. Petersons A, Volrats O, Berenstein A. The first experience with non-operative treatment of hypersplenism in children with portal hypertension. Eur J Pediatr Surg 2002;12:299-303.
20. Allan E Dumont, Berman IR, Stahl WM, Ring SM. Significance of enlarged splenic artery in patients with bleeding varices. Ann Surg 1972;175:466-71.
21. Blendis LM, Banks DC, Ramboer C, William R. Spleen blood flow and splanchnic hemodynamics in blood dyscrasia and other splenomegalies. Clin Sci 1970;38:73-84.
22. Shimizu T, Onda M, Tajiri T, Yoshida H, Mamada Y, Taniai N, *et al*. Bleeding portal hypertensive gastropathy managed successfully by partial Splenic embolisation. Hepatogastroenterology 2002;49:947-9.
23. Adylova GS, Salimov ShT, Borovskii SP, Alimukhamedova DK, Tursunov KZ. Embolisation of splenic artery in children with portal hypertension. Khirurgiia 2000;6:15-7.


**Source of Support:** Nil, **Conflict of Interest:** None.

## PAPSA: Dar-es-Salaam 2010




As noted in the brochure, we are planning an exciting and full programme! This will include, among others,

- Basic Skills course in Paediatric Surgery;
- Laparoscopic training sessions;
- Workshop on the surgery of children with spina bifida and hydrocephalus;
- Part 1 examination of the European Board of Paediatric Surgery (EBPS);
- Several panels on current topics for African paediatric surgery;
- Invited guest lectures from several well-known European, North American and African experts;
- Scientific presentations and posters.

Moreover, the International Association for Spina Bifida and Hydrocephalus (IFSBH) will hold its annual workshop concurrently, and they are graciously providing some excellent faculty and speakers!

The local arrangements committee led by Dr. Petronilla Ngiloi is also preparing a social programme for participants and their families. And of course, what better starting place than Dar-es-Salaam for a holiday either before or after the congress, relaxing in Zanzibar or climbing Mount Kilimanjaro?

This promises to be the best PAPSA meeting yet, so please register early to avoid disappointment! Here's a quick checklist:

- Email the conference registration info as instructed in the brochure
- Book the hotel of your choice using the info in the brochure
- Inform both the local arrangements committee and myself if you plan to write the EBPS exam
- Prepare and email scientific abstracts using the info in the brochure

Please note however that the abstract submission deadline is June 1st, not June 15.

## PAPSA: TANZANIA 2010 AT A GLANCE

**8th CONGRESS OF THE PAN-AFRICAN PAEDIATRIC SURGICAL ASSOCIATION (PAPSA) AND ANNUAL GENERAL MEETING AND SCIENTIFIC CONFERENCE OF TANZANIA SURGICAL ASSOCIATION (TSA)**

DAR-ES-SALAAM, TANZANIA Date: 5-9 July, 2010

THEME: Trauma & disability in Africa

### DETAILS OF EVENTS

**Workshops**

1. BAPS basic skills workshop – July 5
2. Surgery of spina bifida & hydrocephalus – July 5-6
3. BAPS Laparoscopic modules – July 7-8-9

**Scientific sessions**

Presentations - July 7-8
Association meeting – July 9
Plenary sessions – subthemes

- Trauma in Africa
- Burn management
- Neurological disability in children

**Other highlights**

- The inaugural PAPSA Heinz Rode lecture
- Panel discussions
- Joint sessions with the International Federation for Spina Bifida and Hydrocephalus
- Departure - July 9

### Local Organizing Committee

Dr. P. Ngiloi- pngiloi@muhas.ac.tz (Chairperson)
Dr. Nungu- samwelnungu@yahoo.com Dr. Wandwi- wwandwi@gmail.com
Dr. Mareclle- pgmareclle@yahoo.com Dr. Ali Shabhy- aishabhay@hotmail.com
Dr. R. I. Mhina- rimhina@gmail.com Dr. John Mboya- drjohnmawolo@yahoo.com

### For More Information, please contact:

Dr Petronilla J. Ngiloi
Vice President of PAPSA, Chairman of local organizing committee
petronillangiloi@yahoo.com, pngiloi@muhas.ac.tz

### Abstract submission

Deadline for submission of abstracts:
June 15, 2010

**Abstract format:**

- Title
- Authors and Institution
- Introduction
- Methods
- Results
- Discussion

**Word limit: 400 words**

Send abstracts to
Prof. Dan Poenaru [bkkhmd.kh@kijabe.net]
With copy to [wwandwi@gmail.com]

**Registration details**

Registration fee including lunch and snacks
Participant working in Africa - $150 Participant working outside Africa – $200
Accompanying person - $100

**E-mail Registration format**

- Title
- Surname
- First name
- Institution
- Specialty/subspecialty
- Country
- Planning to attend:

• BAPS basic skills workshop – yes/no • BAPS laparoscopic modules – yes/no
• Spina bifida/hydrocephalus workshop – yes/no
• EBPS written examination – yes/no

Register Online, Visit website: via www.tsa-tz.org or
Send your registration form to Dr. Petronilla [Ngiloipetronillangiloi@yahoo.com]
You may include information on any accommodation arrangements made.

**Accommodation**

Participants are required to make their own bookings using the information below; indicate that you are attending the PAPSA conference.
The conference will be held at White Sands Hotel & Resort [marketing@hotel-whitesands.com],
Fax +255 22 264 7875
Rates: $80 single, $110 double

**Other nearby hotels with their rates:**

Eclipse Sea Breeze Resort [gmjangwani@eclipsehotelsafrica.com]
Fax +255 22 264 72069)
Rates: $68 single, $91 double
Belinda Ocean Resort [info@belindaresort.com]
Fax +255 22 264 7552
Rates $68 single, $90 double
Giraffe Ocean View Hotel [giraffe@giraffeoceanviewhotel.com]
Rates: $100 single, $115 double
Transport from the Airport will be arranged if you provide your flight itinerary
A tour agent will be available to see to any tour requirements.
For further information contact Dr. Ngiloi