International Journal of Medical Science in Clinical Research and Review
Online ISSN: 2581-8945
Available Online at http://www.ijmscrr.in Volume 05|Issue 05 (September-October)|2022|Page: 574-584
NLM ID: 101768774 **SJIF Impact Factor:** 5.782

Original Research Paper

Visceral Artery Aneurysms – An Indian Gastroenterologists Perspective

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Article Received: 07-08-2022 **Revised:** 26-08-2022 **Accepted:** 17-09-2022

ABSTRACT:

Background and Aims: Visceral artery aneurysms (VAA) are a rare disease entity. Our study was designed to evaluate the etiology, presentation outcomes at 1 month in a tertiary care center for visceral artery aneurysms. **Methods:** We performed retrospective analysis of data from 2017-2021 at a tertiary care centre in Western India of all the patients with aneurysms. **Results:** Fifty-seven aneurysms and 48 pseudoaneurysms in 74 patients were studied. The most commonly involved vessels were splenic artery (69.5%) & gastroduodenal artery (GDA) (17.1%). About 75.6% patients were symptomatic, 51.3% had gastrointestinal bleeding (GI bleeding) & 24.3% abdominal pain. The most common etiologies noted were - Chronic Pancreatitis (35.1%) & decompensated cirrhosis (20.1%). Almost all patients with pseudoaneurysms were symptomatic ($p< 0.0001$). True aneurysms were more likely to be multiple. ($p=0.009$). There was no significant difference in mortality and rebleed at one month between aneurysms and pseudoaneurysms ($p=0.4887$ & $p=0.873$). Male patients were found to have a higher risk of GI bleeding, irrespective of etiology ($p=0.006$), whereas female patients were more likely to have complications post intervention ($p=0.04$). Conservative treatment was given to 32.4% of patients who had a mean size of aneurysm being 1.2 ± 0.77cm. Interventional radiology guided treatment was offered in the form of angiographic coiling (40.5%), glue injection (17.6%) and combined (5.4%). Around 8.1% patients had complications post intervention, most common being Gram negative septicemia; **Conclusions:** Pseudoaneurysms have a high risk of rupture,. Endovascular intervention in is safe and effective.

Keywords: *Aneurysms, Endovascular coiling, Gastrointestinal bleeding*

INTRODUCTION:

Visceral artery aneurysms (VAA) are a rare disease entity, with a general incidence of approximately 0.01-2%. [1] The available literature reports cases of VAA from 1985 to 1995 in the English literature, reporting a mortality rate of 21% for ruptured hepatic artery aneurysms and 100% for ruptured celiac artery aneurysms.[2] The increasing detection rates of asymptomatic, incidental aneurysms are attributed to the ever increasing use of high resolution imaging technology.[3]Our study was designed to describe the demographic profile, including the modes of presentation, etiologies, possible risk factors for rupture, and various endovascular modalities available for their management at a tertiary care institute in India.

METHODS:

This was a single centre retrospective observational study from January, 2017 –August, 2021. Institutional ethics committee approval was obtained.

Study Population*:* The discharge records of all patients admitted in the medical Gastroenterology department of our institution from year 2017 to 2021, were screened for keywords "aneurysm" or "pseudoaneurysm" of the visceral arteries. Visceral arteries encompassed the celiac, superior mesenteric, inferior mesenteric artery and their branches. Abdominal aorta and renal artery aneurysms were excluded. As an institutional protocol, all patients who underwent splenic artery intervention underwent vaccination. A total of approximately 3634 patients and discharge records were screened. A waiver of consent was granted by the Institutional ethics committee. Demographic data along with details of imaging findings, details of intervention if performed

and outcomes of mortality and rebleeding at one month were obtained.

Clinical Assessment& Definitions: Seventy four patients matched our keyword search. Segregation of true aneurysms from false aneurysms was done on the basis of computed tomography reporting by a senior radiologist with at-least five years of experience. Clinically, the patients with visceral artery pseudoaneurysms (VAPA), typically present with an antecedent history of arterial trauma, intra-abdominal or retroperitoneal inflammatory disorders, malignancy, or biliary tract manipulation.[2]As histopathology is the only definitive way of differentiating between the two, imaging was used as the standard for our study. Aneurysms/Pseudoaneurysms were considered to be incidental if symptoms of Gastrointestinal bleeding, blood loss anemiaor abdominal pain couldn't be attributed to them on clinical grounds. Symptomatic VAA/VAPAs presented with either any one or combination of the following: Abdominal pain or gastrointestinal bleeding including occult blood loss, hematochezia, hematemesis, melena, hemosuccuspancreatic us, hemobilia, hemoperitoneum. Etiologywas broadly divided into liver disorders, pancreatic disorders and others. Liver disorders consisted of extrahepatic portal vein obstruction, non cirrhotic portal fibrosis and cirrhosis (compensated and decompensated). Pancreatic disorders were divided into acute &chronic pancreatitis. Miscellaneous associations like vasculitis, mitral stenosis, associated HIV were also included. Hemodynamic stability was defined as systolic blood pressure of 90mmHg or more without inotropic support. Significant Alcohol consumption was defined as consumption of $\geq$ 60gram of alcohol for men, and $\geq$20gram of alcohol for women. Smoking history was recorded as pack years of smoking. Blood investigations like complete hemogram, liver function tests, renal function tests, viral markers for chronic hepatitis, prothrombin time & INR at the time of admission was recorded. Diagnostic modality, including a contrast enhanced CT, MR Angiography (MRA), hepatoportal doppler, conventional angiography, that first documented the presence of an aneurysm or pseudoaneurysm was noted. Choice of therapy offered was recorded. Patients were either managed conservatively, surgically with splenectomy, or with vascular interventions like N-butyl cyanoacrylate glue embolization (Endocryl, Samarth Industries, India), coil embolization using Nester coils (Cook Medical, St. Jude Medical USA) or thrombin injection Reliseal (Reliance Life Sciences, Mumbai, India) or a combination of the above as deemed necessary by the treating interventional radiologist. Intervention was offered to patients who were symptomatic with GI bleeding, abdominal pain.Aneurysms with a size of >2cm have been offered intervention based on data from several studies.[5-9]Indications for repair in our study included rupture, symptomatic aneurysms, presence of a pseudoaneurysm or mycoticaneurysm, intact celiac axis or superior mesenteric artery(SMA) and splenic aneurysms $\geq$ 2.5 cm in diameter in women of childbearing age.[10]Patients with a CTP $\geq$ 10 or end stage malignancy were treated conservatively in our study. Initial success was defined as stopping the flow within the aneurysm or successfully excluding it by the end of the procedure.[11]Details of intervention were recorded. The number& size of coils used and dose of NBCA used was recorded. VAA/VAPAs in splenic artery were either managed with complete Splenic artery embolization or aneurysm embolization. One month follow up records were obtained from out patient records maintained at the department. Complications related to intervention included recurrent Gastrointestinal hemorrhage, septicemia, splenic infarction (>50%) and systemic embolization of coils or NBCA.

Statistics: Data recording was done in MS Excel. Continuous variables were reported as Mean + SD, median (IQR) and range. Discrete variables are summarized in terms of frequencies and percentages. Differences in categorical variables with the chi square test and fisher's exact test. Mann Whitney U test was used for difference between continuous variables between two groups. All statistically analysis was performed using "R Studio version 1.4.1103." A two-tailed p value of <0.05 was considered to be statistically significant.

RESULTS:

Demographic Data

A total of 74 patients were included in the study, out of which 39.2% (n=29) were women. Table 1 shows the demographic data, baseline laboratory data of the patient population along with the number of aneurysms or pseudoaneurysms, etiologies, symptoms and treatment offered. No single biochemical marker was found that could be used to predict severity in terms of hemodynamic instability, higher rebleed, post procedural complications or mortality.

Comparison of VAA with VAPA

Out of 74 patients, 41.8% (n=31) patients had 57 VAA, and 58.1% (n=43) patients had 48 VAPA. Comparison of various clinical, biochemical and outcome parameters between the two are outlined in table 2. Almost all patients with pseudoaneurysms were symptomatic (p<0.0001). True aneurysms were more likely to bemultiple (n=1.84v/s 1.12; p-0.009). VAA were significantly more likely to have a wide mouthed neck (n=15 v/s n=8; p-0.006). There was no significant difference in mortality and rebleed at one month

between aneurysms and pseudoaneurysms (p=0.4887 & p=0.873respectively).

Anatomical location

Fifty-seven aneurysms and 48 pseudoaneurysms in 74 patients were studied. The most commonly involved vessels were splenic artery (62%) & gastroduodenal artery (GDA) (18%).Fig. 1 shows the distribution of vessel involvement in our study.

Etiology& Associations

The most common etiologies (Fig 2) noted were - Chronic Pancreatitis (35.1%) & decompensated cirrhosis (20.1%). The etiology for decompensated cirrhosis ranged from Alcohol related cirrhosis (n=4), Chronic Budd Chiari Syndrome (n=4),Autoimmune Hepatitis (n=2), hepatitis B (n=1), hepatitis C (n=1), and Wilson's related cirrhosis(n=1). Two patients developed Gastrointestinal bleeding post ERCP, and had a splenic and GDA pseudoaneurysm, detected on CECT Angiography.

Symptomatology and risk factors

More than 3/4th of the patients were symptomatic, and more than half [51.3%] had gastrointestinal bleeding (GI bleeding) &(24.3%) abdominal pain. About 38 out of 56 patients presented with Gastrointestinal bleeding, with the mean size and number of aneurysms being 1.84 ± 1.93.Eighteen patients presented with only abdominal pain, with a mean size and number of aneurysm being 1.52 ± 1.08 cm. No significant association of mesenteric venous thrombosis, wide mouthed aneurysms/pseudoaneurysms could be found with symptoms.

Figure 2 shows the various presentations of GI bleeding. Melena (68.4%) and hemetemes is (50%) were most common. Men were more likely to be symptomatic than females(n=39 vs. n=17; p-0.006). There was no association of significant alcohol consumption and smoking with symptoms. Gastrointestinal bleeding was not related to the size (>2cm) and number of aneurysms. (p-0.505)

Diagnostic Modalities

All patients underwent imaging for diagnosis of VAA/VAPA. The diagnostic modalities were Doppler Ultrasound, Contrast Enhanced Computed Tomography and Angiography, Magnetic resonance angiography or Conventional Angiogram. Almost all VAA/VAPA, [86.5%] were first diagnosed on CT (n=64), with 2.7% diagnosed only on Conventional Angiogram (n=2), and 10.8% diagnosed on MR Angiogram (n=8). The two patients with normal MR and CT angiography, underwent a Conventional angiography which was diagnostic for VAA. Conventional angiogram was performed in 62.1% (n=46) patients, out of which 56.8% (n=42) were symptomatic. Out of the patients who underwent conventional angiogram, 26.1% (n=12) patients had an active blush from the VAA/VAPA.

Mesenteric venous thrombosis

There was no association of mesenteric vein thrombosis, with mortality or rebleed, as shown in Table 3.Absence of mesenteric venous thrombosis shows significant association with symptomatic aneurysms (p-0.005). Presence of venous thrombosis had significant association with complications post intervention (p-0.008).

Treatment

Interventional radiology guided treatment was offered in the form of angiographic coiling (57.1%), angiographic glue injection (24.4%) and angiographic glue+coiling respectively (18.3%). The mean amount of N-butyl cyanoacrylate & coils used was 1.45±0.72mL & 3.89±1.87 respectively. There was no significant difference between the risk of rebleeding or mortality amongst the modes of interventional therapy as shown in table 4. One patient underwent surgical management for aneurysmal rupture. He had a splenic artery pseudoaneurysm of 2.1cm, with a splenic hematoma and underwent aneurysmal repair and splenectomy. Conservative treatment was given to about a third of patients [32.4%; N- 24]. Four percent died before an intervention could be performed. Patient population managed conservatively were either asymptomatic (52%), and patients who refused invasive treatment (16%). An additional 28% of patients, who were poor candidates for invasive management like those with CTP-C cirrhosis were initially given medical therapy for symptom control (analgesics, octreotide) and managed conservatively. Only 16.6% (n=4) that were managed conservatively were VAPAs, three of whom refused invasive treatment.

Outcomes

Complications:

Around 8.1% patients had complications post intervention. All of them had culture proven Gram negative septicemia. Out of 27 patients who underwent splenic artery aneurysm coiling, 11.1% (n=3) developed splenic infarct (>50%) with sepsis, all managed medically with no mortality. Two out of those three patients with large splenic infarcts were vaccinated at the time of emergent endovascular intervention. One asymptomatic patient with NCPF & splenic artery aneurysms, developed liquefactive infected necrosis of the spleen, requiring prolonged antibiotics and percutaneous pigtail drainage. There was no systemic embolization seen post intervention in any patients.

Rebleeding:

Three patients rebled, including two patients managed conservatively, and one managed with endovascular coiling of GDA. A patient with asymptomatic VAA, and

another with a symptomatic VAPA managed conservatively came with rebleed. The patient with VAPA, came with exacerbation of chronic pancreatitis and a pseudocyst with an episode of melena with initial CT showing an inferior pancreaticoduodenal aneurysm, later not visualized on conventional angiography. He was readmitted with hemodynamical instability and GI bleed, and a SMA pseudoaneurysm was detected, which was managed with endovascular coiling. The other patient was a CTP-C cirrhosis secondary to chronic Budd chiari syndrome with HCC with splenic artery aneurysm of 1.3cm, presented with GI bleed. He was managed conservatively due to a short anticipated median survival. A patient with Mitral stenosis, underwent GDA aneurysm coiling for bleeding VAPA, rebled in a month. A repeat GDA coiling + glue was done, in a new GDA pseudoaneurysm detected on the conventional angiography.

Mortality:

A total of 5 out of 74 died within a month of symptomatic presentation. Two patients had a VAA, and both had CTP-C cirrhosis and splenic artery aneurysms, were managed conservatively. One died of septicemia after admission, within 48 hours of admission, and the other presented with melena and hypovolemic shock with no varices on esophagogastroduodenoscopy, and bleeding presumed to be aneurysmal in nature leading to death. Three patients with VAPA, died in one month of presentation, and were all symptomatic to begin with. One patient with multiple VAPA in hepatic artery secondary to Acute necrotizing pancreatitis came with respiratory failure and active hematemesis, succumbed in 48 hours after presentation. The second patient who died, came with a GDA aneurysm, with a hemorrhage in a walled off necrosis, detected on CT angiography, underwent endovascular coiling of the aneurysm. He died off an infected walled off necrosis. The third patient refused treatment and was managed conservatively for a splenic artery VAPA.

DISCUSSION:

The present study was designed with the aim to provide deeper insight into the incidence, etiology, management and outcomes of visceral artery aneurysms and pseudoaneurysms in India. To the best of our knowledge, ours is the largest study from south east Asia, that focuses on relatively rare causes of GI bleeding like VAA. In our study, the overall incidence of visceral artery aneurysms [true or pseudo] was relatively low(0.02%), with a total of 74 patients presenting with 31 patients having true and 43 patients having pseudoaneurysms, seen over a 4-year period.[1,2] The mean age in our study was 41 years, lower than several reported studies that usually report aneurysms which are secondary to atherosclerosis, hence have an overall higher age at detection. [12,13]The mean age in another Indian study, by Madhusuan et al. was 32 years.[14]Male predominance was noted a majority of reported studies and are in harmony with our findings of a male-to-female ratio of 3:2.[2,12,13]Splenic and GDA were the two most common sites.. In other reported studies historically, splenic and hepatic artery are the two most commonly involved vessels.[1,2,12] In our study, inflammatory conditions like chronic pancreatitis and acute pancreatitis were the most common causes, which explains the higher likelihood of GDA aneurysms in our study. This is in stark contrast to previously existing literature, that touts GDA aneurysms as the least likely vessel to be involved, and points to the influence of socioeconomic milieu on the patterns of disease.[15] Portal hypertension was also a predominant association with development of aneurysms in our study. However, Pitton and colleagues report atherosclerosis and post intervention aneurysms as their most common etiology, with portal hypertension and cirrhosis being a relatively less common association.[13]The study by Pitton and colleagues included abdominal aorta and renal artery aneurysms in large numbers, which were excluded in our study.[13] Our study was performed in the gastroenterology department, which lead to a selection bias as more of our patient inflow consist of cirrhotic patients, as opposed to studies performed in a radiology setup, which may have included patients from other specialties of medicine. Over Ninety-five percent of our pseudoaneurysms and 50% of our true aneurysms were symptomatic, which is higher than the available data reported by Pitton.[13]Ours is a tertiary care centre, leading to a higher case load of referred patients. They present to us only when symptomatic, which is likely a referral bias. Although the use of a cross sectional imaging with CT and MR are increasing rapidly, an ultrasound is usually the first investigation done in most patients presenting with non-emergent unrelated complaints. Hence, most of our VAPAs and over half of our VAA were detected on ultrasound, as that was the first radiological investigation performed. The mean size of aneurysms (1.66 ± 0.81cm) and pseudoaneurysms (1.59 ± 1.34 cm) in our study was lower than the mean size in several other studies.[3,13] The mean size of a ruptured VAPA was 3.0cm in a study by Tulsyan with a predominantly Caucasian population.[3]In another case series by Gabrielli et al, symptomatic ruptured VAPA secondary to chronic pancreatitis, had a mean size of 4.0cm.[16]The size of aneurysms and pseudoaneurysms in Indian patients that could lead to symptoms could be smaller than the established size cut offs based on expert consensus. This is another novel finding in our study, highlighting the need for an unmet need for data from our part of the world in order to restructure pre-existing

norms tending to our population. Although literature suggests, that asymptomatic VAA <2cm in size do not qualify for treatment, our study confirmed that size of the aneurysm has no bearing on the risk of rupture. Similar findings have been noted in the study byPitton and colleagues.[13] The number of VAA/VAPA in each vessel had no bearing on symptoms or mortality in our study. We report a significantly higher number of VAA per patient than VAPA (1.84 ± 1.93 vs. 1.12 ± 0.32, p=0.01). However, despite having a higher mean number of VAA per patient, only half were symptomatic. Presumably, a higher number of aneurysms do not necessarily suggest a higher chance of rupture. This finding however needs prospective validation. Male patients had a significantly higher chance of presenting with rupture (39 v/s 17, p=0.006) in our study. This is a new finding, not reported in literature in prior studies. Although, a definite correlation between male gender and rupture needs to be prospectively evaluated, some reasons could explain this finding in our study. Most patients presenting with pancreatitis had pseudoaneurysms, and alcohol was a major causative factor for the pancreatitis. In India[17] and worldover[18], men are more likely to be significant alcohol consumers, and present with alcohol use disorder, and hence have a higher risk of alcohol related complications like pancreatitis, which would explain the higher number of pseudoaneurysms noted. The most common mode of presentation as GI bleeding was melena and hematemesis. We had two patients with post intervention pseudoaneurysms – one of GDA and another in the splenic artery, that presented with haemobilia (noted in PercutenaousTransbiliary Drain output) and hemosuccuspancreaticus respectively. In a study by Pitton et al, 35/233 were symptomatic due to aneurysm rupture, out of which only 1 had GI bleeding.[13]Thesefindings are dissimilar from our study, where 75.6% patients were symptomatic with 51.3% presenting with GI bleeding. Although incidental detection of aneurysms is on the rise in India, most were detected when they were symptomatic in our study. Contrast enhanced CT angiography detected 86.5% of the aneurysms, with MR angiography detecting another 10.8%. Patients with overt GI bleeding, a normal CT or MR angiography, & a high index of suspicion underwent conventional angiography and in 2.7% cases, it lead to the diagnosis. We did not find a correlation of wide mouthed aneurysms with symptoms and complications post intervention. (p-0.78). This was the first study to look for an association of mesenteric venous thrombosis with symptoms of aneurysm and complications post intervention and a statistically significant association of presence of mesenteric vein thrombosis associated with development of post intervention complications (3v/s 1; p=0.008). In our study, technical success for endovascular intervention was achieved in 100% cases. It correlates with other studies where technical success was achieved in 98-100% patients.[4,13,14] Eleven patients with VAA underwent endovascular intervention. Females were more likely to develop post intervention complications (0 v/s 4; p=0.04). Three of these patients had a splenic artery true aneurysm who underwent splenic artery coiling and developed complications. All three had massive splenomegaly with a size >18cm with more than 1 aneurysm in each case. The most major complication leading to a hospital stay of more than 45 days was seen in a female with NCPF with multiple splenic artery aneurysms who developed a splenic infarct, sepsis followed by liquefactive necrosis of the spleen. The necrotic collection secondarily developed an infection which was managed with antibiotics and pigtail drainage. The rates of major complications, rebleeding and mortality were 8.1%, 2% and 2% respectively in those who underwent intervention. There are certain important limitations in our study. Firstly, it was a retrospective study, which restricted the information available, to further evaluate and prove association of various parameters with risk of rupture. Secondly, our follow up period was shorter and less than ideal to precisely gauge the impact of intervention on the risks of arterial thrombosis, or organ dysfunction on later follow up. It also put a limit on our understanding of patient well-being with conservative management. Thirdly, our follow up included only telephonic conversation and available patient records showing absence of need for admission or further anemia and GI bleeding. Imaging records post intervention at follow up would've provided a more information about whether the intervention was actually successful in isolating the aneurysms from the circulation. Another limitation lies in our ability to distinguish between true and pseudoaneurysms consistently, as no characteristic non pathology based signs are available in literature. This put a limit on establishing clearly which disease states were merely associations, and which conditions were causative for the formation, and rupture of both aneurysms, and pseudoaneurysms. It doesn't help us solve why, some patients with seemingly inflammatory conditions develop VAA, and those with EHPVO, NCPF, cirrhosis developed VAPA.

CONCLUSION:

This is one of the largest studies dealing with Visceral arterial aneurysms from south east Asia, and the first such study from India. The decision to treat asymptomatic aneurysms must be measured against the risk of complications post intervention, as bleeding risk does not correlate with size, number or location. Female gender and presence of mesenteric venous thrombosis

were associated with a higher risk of complications. Endovascular intervention appears to be safe and effective for management of VAA/VAPA. Prospective studies are required to confirm the findings from our study.

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Figure Legends and Tables

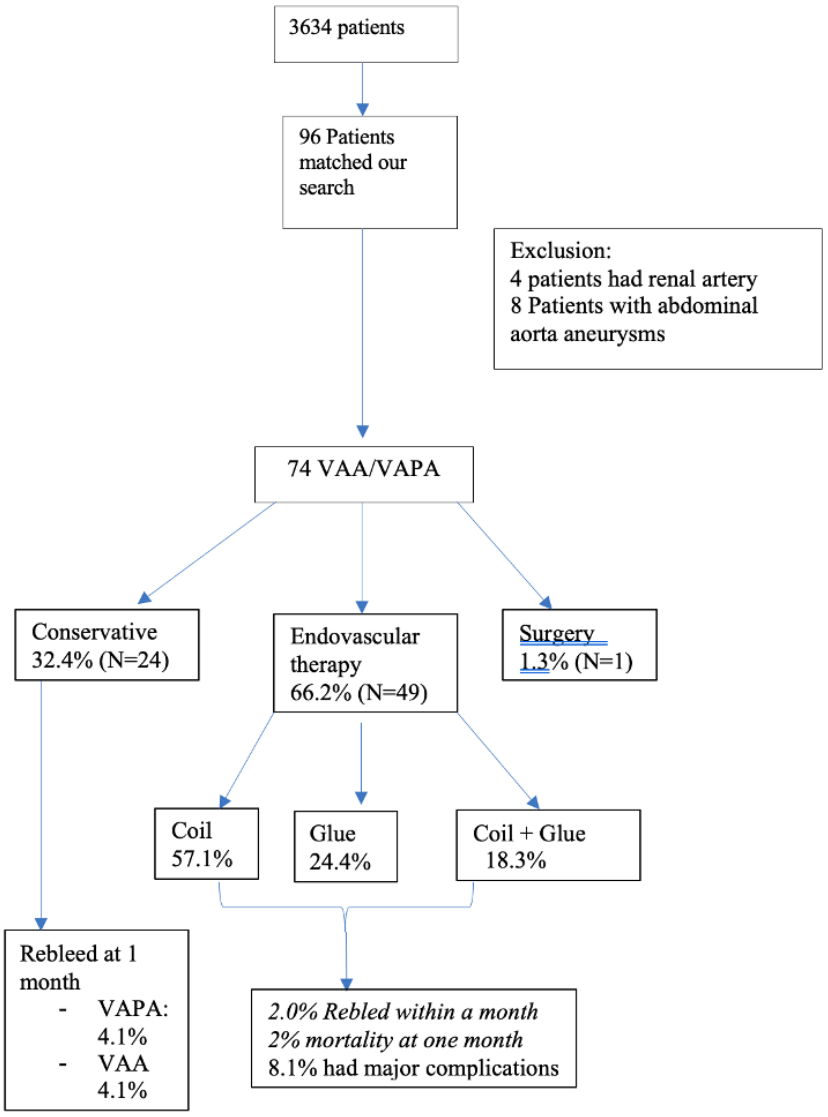
3634 patients
96 Patients matched our search
Exclusion:
4 patients had renal artery
8 Patients with abdominal aorta aneurysms
74 VAA/VAPA
Conservative 32.4% (N=24)
Endovascular therapy 66.2% (N=49)
Surgery 1.3% (N=1)
Coil 57.1%
Glue 24.4%
Coil + Glue 18.3%
Rebleed at 1 month
- VAPA: 4.1%
- VAA 4.1%
2.0% Rebled within a month
2% mortality at one month
8.1% had major complications

Figure 1a: Distribution of vessels involved, showing higher proportion of splenic artery (62%) and GDA aneurysms (18%).

Figure 1b: Etiology& Associations of VAA/VAPA

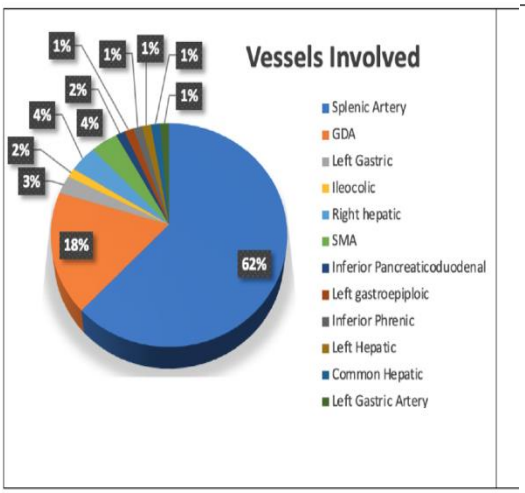

Fig. 1a: Distribution of vessels involved;

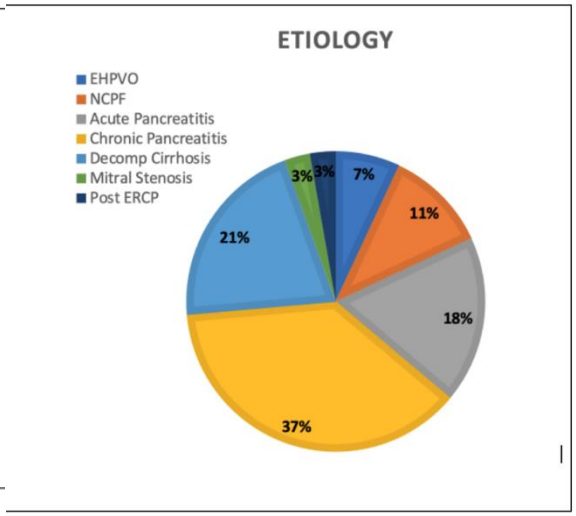

Fig. 1b : Etiology & Associations of VAA/VAPA

Figure 2: Different modes of presentation in symptomatic patients with Gastrointestinal bleeding.

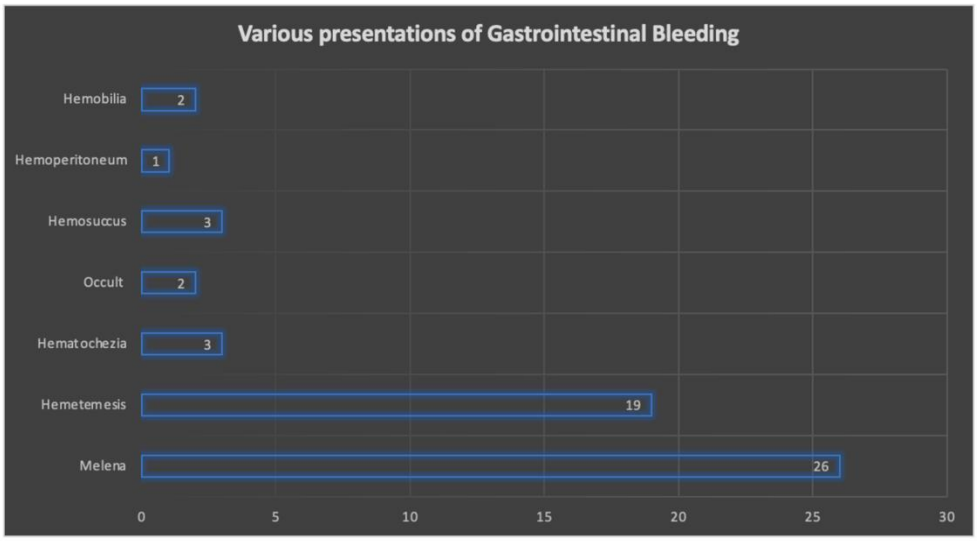

Fig. 2. Various modes of presentation in symptomatic patients with Gastrointestinal bleeding

Table 1. Baseline demographic laboratory data, vessel distribution, treatment and outcomes in true and pseudoaneurysms

Demographics			
Age in years [Mean ± SD]		41.32 ± 15.04	
Sex [No. (%)]	Female	29 (39.2)	
	Male	45 (51.8)	
BMI in kg/m2 [Mean ± SD]		21.7± 2.1	
Baseline laboratory tests			
Haemoglobin (%)		8.52±2.16	
Platelet count (/µl)		162970.27±104708.81	
SGOT (U/l)		87.71±129.69	
SGPT (U/l)		52.32±68.96	
Total protein (g/dl)		6.28± 0.79	
Albumin (g/dl)		3.10± 0.62	
Total bilirubin (mg/dl)		1.51± 1.19	
INR		1.34± 0.28	
Lipase (U/l)		132.82± 252.93	
Vessel distribution	*Patients with True aneurysm (VAA)*	*Patients with Pseudoaneurysm (VAPA) [No. (%)]*	
Splenic artery	24	22	
Gastroduodenal artery	4	9	
Right hepatic artery	0	3	
Superior mesenteric artery	0	3	
Left hepatic artery	0	1	
Common hepatic artery	1	1	
Left gastric artery	0	2	
Left gastroepiploic artery	0	1	
Inferior phrenic artery	1	0	
Inferior pancreaticoduodenal	0	1	
Ileocolic	1	0	
Etiology & Associations	*Patients with True aneurysm (VAA)*	*Patients with Pseudoaneurysm (VAPA) (No.)*	
Chronic Pancreatitis	1	26	
Acute Pancreatitis	3	10	
Decompensated Cirrhosis	13	0	
EHPVO	4	1	
NCPF	7	1	
Iatrogenic	0	3	

Mitral Stenosis	1	1	
HIV	1	1	
Vasculitis	1	0	
Clinical Presentation		**No. [%]**	
Asymptomatic		18 (VAA-16; VAPA – 2) [24.3%]	
Abdominal Pain		19 (VAA – 9; VAPA – 10) [25.6%]	
Gastrointestinal Bleeding (n=38) *	Melena	26 (68.4)	
	Hemetemesis	19 (50)	
	Hematochezia	3 (7.8)	
	Hemosuccuspancreaticus	3 (7.8)	
	Hemobilia	2 (5.3)	
	Hemoperitoneum	1 (2.6)	
	Anemia / Occult Blood loss	2 (5.3)	
Treatment			
Conservative	Total	24 (32.4)	
	Asymptomatic	12 (51.7)	
	Death within 48 hours	2 (8.3)	
	Refusal for consent	6 (28.4)	
	CTP-C Cirrhosis	4 (16.6)	
Endovascular	Total	49 (66.2)	
	Coil	28 (57.1)	
	Glue	12 (24.5)	
	Coil + Glue	9 (18.3)	
Surgery		1 (1.3)	

Table 2: Comparison of various parameters between Aneurysm and Pseudoaneurysm patients

Parameter	Status			P-value
		Aneurysm (n=31)	Pseudoaneurysm (n=43)	
Duration in months [Mean ± SD]		14.32 ± 23.31 (2.00)	8.98 ± 10.67 (8.00)	0.189*
Symptoms at presentation [No. (%)]	Asymptomatic	16 (51.6)	2 (4.6)	**< 0.0001‡**
	Symptomatic	15 (48.4)	41 (95.4)	
Number of aneurysms (CT/MR) [Mean ± SD]		1.84 ± 1.93 (1.00)	1.12 ± 0.32 (1.00)	**0.009‡**
Size of aneurysms (cm) [Mean ± SD]		1.66 ± 0.81 (1.50)	1.59 ± 1.34 (1.10)	0.782*
Number of coils [Mean ± SD]		5.00 ± 3.46 (4.00)	3.54 ± 1.93 (3.50)	0.383
Wide mouth [No. (%)]	Yes	15 (48.4)	8 (18.6)	**0.006‡**
	No	16 (51.6)	35 (81.4)	
Intervention performed	Yes	11	39	
	No	20	4	
Complications post intervention [No. (%)]	Yes	3/11 (27.3)	1/39 (2.6)	**0.007‡**
	No	8/11 (72.7)	38 /39(97.7)	

Aneurysm related Mortality at 1 month [No. (%)]	Yes	2 (6.4)	3 (6.8)	0.395‡
	No	30 (93.5)	40 (93.1)	
Rebleed at 1 month [No. (%)]	Yes	1(3.2)		0.224‡
	No	30(96.70	41 (95.3)	

Table 3. Comparison of various parameters in patients with and without Mesenteric venous thrombosis

Parameter	Status	Mesenteric V Thrombosis		P-value
		No (n=58)	Yes (n=16)	
Duration in months [Mean ± SD]		8.67 ± 10.8 (5.50)	20.44 ± 29.68 (11.50)	**0.014***
Symptoms at presentation [No. (%)]	Asymptomatic	11 (18.9)	7 (43.8)	**0.005‡**
	Symptomatic	47 (81.1)	5 (56.3)	
Number of aneurysms (CT/MR) [Mean ± SD]		1.28 ± 0.70 (1.00)	1.94 ± 2.49(1.00)	0.261‡
Size of aneurysms (mm) [Mean ± SD]		1.60 ± 1.20 (1.20)	1.68 ± 0.94 (1.60)	0.803*
Number of coils [Mean ± SD]		3.72 ± 2.31 (4.00)	5.50 ± 3.42 (5.00)	0.234‡
Wide mouth [No. (%)]	Yes	18 (31.1)	5 (31.2)	0.987‡
	No	40 (68.9)	11 (68.8)	
Complications [No. (%)]	Yes	1 (1.7)	3 (18.7)	**0.008‡**
	No	57 (98.3)	13 (81.3)	
Mortality at 1 month [No. (%)]	Yes	3 (5.2)	2 (12.5)	0.301‡
	No	55 (94.8)	14 (87.5)	
Rebleed at 1 month [No. (%)]	Yes	2 (3.4)	1 (6.2)	0.615‡
	No	56 (96.6)	15 (93.8)	

Table 4: Distribution of patient outcome according to treatment

Parameter	Status	Coil, Glue, Coil+Glue		
		Coil (n=28)	Glue (n=12)	Coil + Glue (n=9)
		No. (%)		
Mortality at 1 month	Yes	1 (3.6)	0	0
	No	27 (96.4)	12 (100)	9 (100)
Rebleed at 1 month	Yes	1 (3.6)	0	0