

Case Report

Abdominal Aortic Aneurysm (AAA) Presenting as Constipation in an Elderly Nigerian Man

Dr. Anthonia Ikpeme^{1*}, Dr. Victor Nwagbara² and Dr. Affiong Ngagi¹

Abstract

¹Radiology Department, University of Calabar Teaching Hospital, Calabar, Nigeria

²Surgery Department, University of Calabar Teaching Hospital, Calabar, Nigeria

*Corresponding Author's E-mail: ikpemeanthonia@yahoo.com

This is to report a case of huge Abdominal Aortic Aneurysm presenting as constipation in an elderly Nigerian male. This is a rare disease in Nigeria and it is hoped that the report will further arm clinicians with prompt diagnostic intervention. An eighty year old male with a history of tobacco and alcohol intake, chronic hypertension and a pulsatile abdominal swelling for one year presented at the Accident and Emergency unit because of constipation, examination revealed a normal blood pressure and a pulsatile mass at the right iliac fossa. Computerized tomography, Duplex studies of the abdominal aorta confirmed the diagnosis of AAA. Unfortunately patient succumbed within 24 hours of confirmation of diagnosis before referral to cardiovascular surgery was effected. AAA occurs in this environment, patients with history of hypertension, smoking, alcohol intake and abdominal swelling should have AAA included in the differential diagnoses, appropriately investigated for timely intervention.

Keywords: Abdominal, Aneurysm, Constipation, timely intervention.

INTRODUCTION

Abdominal Aortic Aneurysm (AAA) is a segmental dilatation of the aortic wall that causes the vessel to be larger than 1.5 times its normal diameter or that causes the distal aorta to exceed 3cm (Upchurch and Schaub, 2006). The commonest presentation of patients with AAA is low back ache (Upchurch and Schaub, 2006; Spangler et al., 2014). Other clinical presentations depend on how large it is and whether or not it has ruptured. They include abdominal pain and a pulsatile mass within the abdomen (Spangler et al., 2014; Fauci, 2008). Fewer than half of all cases of AAA present with symptoms (Spangler et al., 2014; Wittels, 2011) (25 – 30%).

It is the tenth most common cause of death in the Western world (Upchurch and Schaub, 2006), with about 10% of persons older than 65 years having AAA (Upchurch and Schaub, 2006). Aortic Aneurysms in Kenya occurs 10 to 15 years earlier than in white population with hypertension being the leading associated risk factor (Ogeng'o et al., 2010). Other risk factors include syphilitic and mycotic infections, inflammation, trauma, auto-immune disease, cystic

medial necrosis and Marfan's syndrome (Upchurch and Schaub, 2006; Fauci, 2008). AAA has a predilection for the male gender (Fauci, 2008). It is still relatively rare in Nigeria.

We presents a case of AAA occurring in an eighty year old male who despite having a pulsatile abdominal mass for one year presented for medical evaluation when he developed large bowel obstruction from mass effect.

The aim is to make medical practitioners and the general populace aware of this condition and improve diagnostic awareness.

CASE REPORT

E was an eighty year old male who was apparently well till the day he presented at the Accident and emergency unit of the University of Calabar Teaching Hospital with a history of inability to pass stool for two days. He was a known hypertensive of ten years and his blood pressure was well controlled. The patient was also a known



Figure 1A. Shows two gray scale transverse images of a huge Aortic aneurysm illustrating the true lumen size and a huge intraluminal clot. Dimensions are stated above. These are taken at the infra renal portions of the aorta and after the bifurcation. There is also an appearance of an intimal flap which is seen in dissecting aneurysms.

prostate cancer patient on 3 monthly Goserelin acetate 10mg (Zoladex®). Last review of his prostate specific antigen (PSA) was within normal limits. (4ng/l)

The patient also complained of mild to moderate lower abdominal pain and said he feels a pulsatile mass to the lower right quadrant of his abdomen. He had felt this mass for about one year and said it was gradually increasing in size. He was a medical practitioner and retired military man. There was no history of hematochezia or melaena stools but there was straining on trying to open bowel. He was not a diabetic.

On examination, an elderly man in satisfactory general status was seen. An abdominal examination revealed a palpable mass with irregular margins at the right iliac region. Pulsatile and synchronized with radial pulse, mildly tender. Pulse rate – 90bts/min, Blood pressure = 130/70mmHg.

Social history revealed significant history of alcohol consumption and tobacco smoking.

A clinical diagnosis of vascular aneurysm with constipation was made.

Abdominal Ultrasound/Doppler studies of the abdominal aorta and branches done with a 4 D coloured sonoscape 6000 ultrasound scanner revealed a huge abdomino-pelvic, irregularly shaped mass arising from the right iliac region. The mass was a complex cystic mass that measured 11.5 x 12.1 x 11.4cm. (Figure 1A) Doppler interrogation of the abdominal aorta and its branches revealed that the mass was an aneurysm of the terminal abdominal aorta and the proximal parts of the right and left iliac arteries. There was also a thrombus seen within. Figures (1a and 1b). The walls appeared thickened posteriorly and much thinner anteriorly.

A Computerized tomographic angiography done with a GE 6 slice helical CT scanner confirmed the above findings. (Figures 2a, 2b, 3 and 4). Chest x-ray revealed an unfolded aorta with arteriosclerosis but no aneurysmal dilatation of the thoracic aorta. Packed cell volume = 38% Fasting lipid profile were as follows;
 Total cholesterol: 3.0mmol/L (up to 5mmol/L)
 High density lipo proteins: 1.3mmol/L (1.1 x 1.7mmol/L)
 Low density lipo proteins: 1.9mmol/L (1.9 x 3.5mmol/L)
 Very low density lipo protein: 0.5mmol/L (0.4 – 0.6mmol/L)

SS

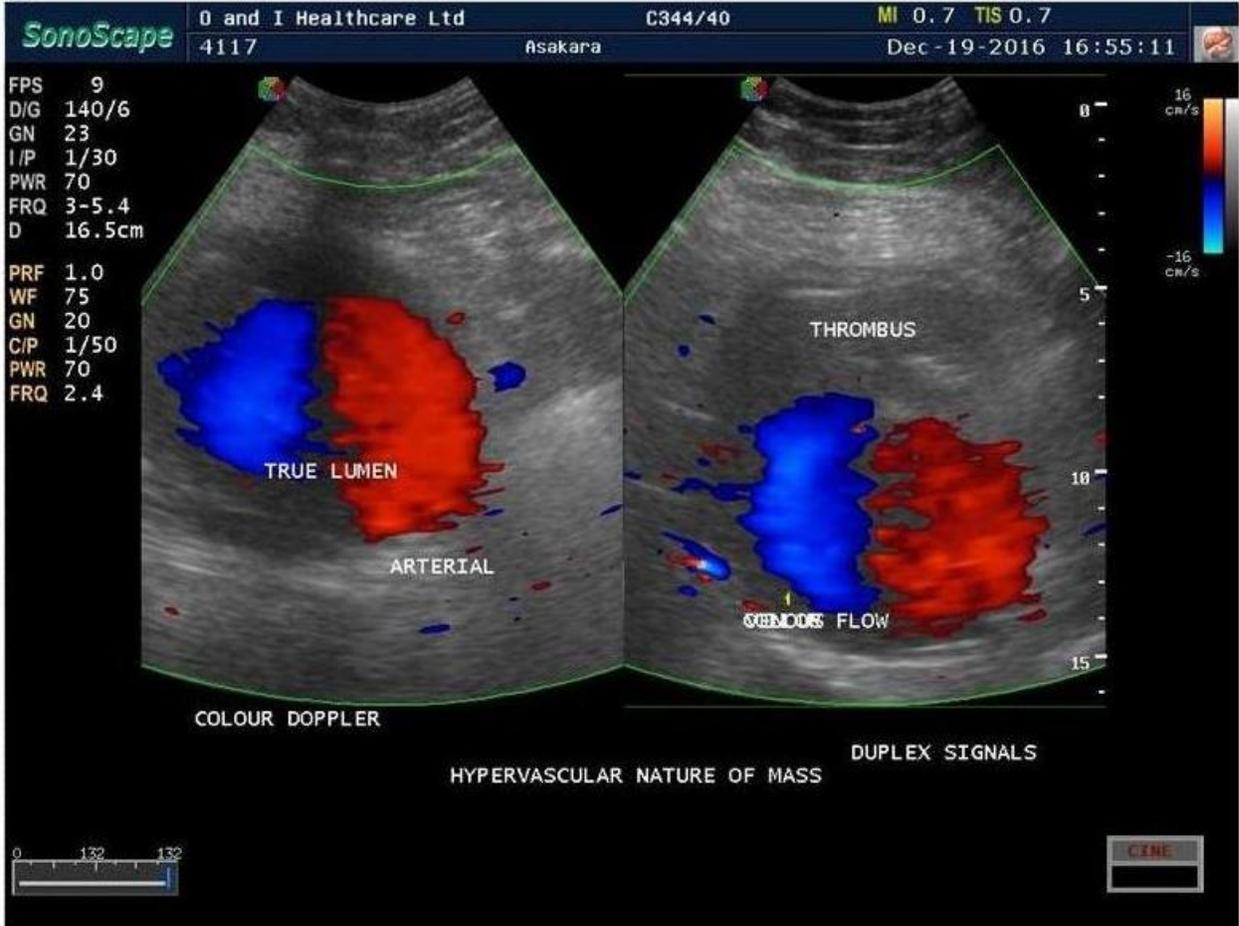


Figure 1B. Transverse colour doppler images of a huge aortic aneurysm illustrating flow only in the region of the true lumen. There is also demonstration of the forward and backward flow within the true lumen. This is suggestive of pseudo-aneurysm.



Figure 2A. Saggital reconstructed ct-scan image showing the aneurysm commencing at the superior border of third lumbar vertebra, calcifications seen within the thrombus.



Figure 2B. Coronal reconstructed ct scan image with contrast seen within the aneurysmic distal aorta and proximal right iliac artery. note also the thrombus that indicates a contained rupture.

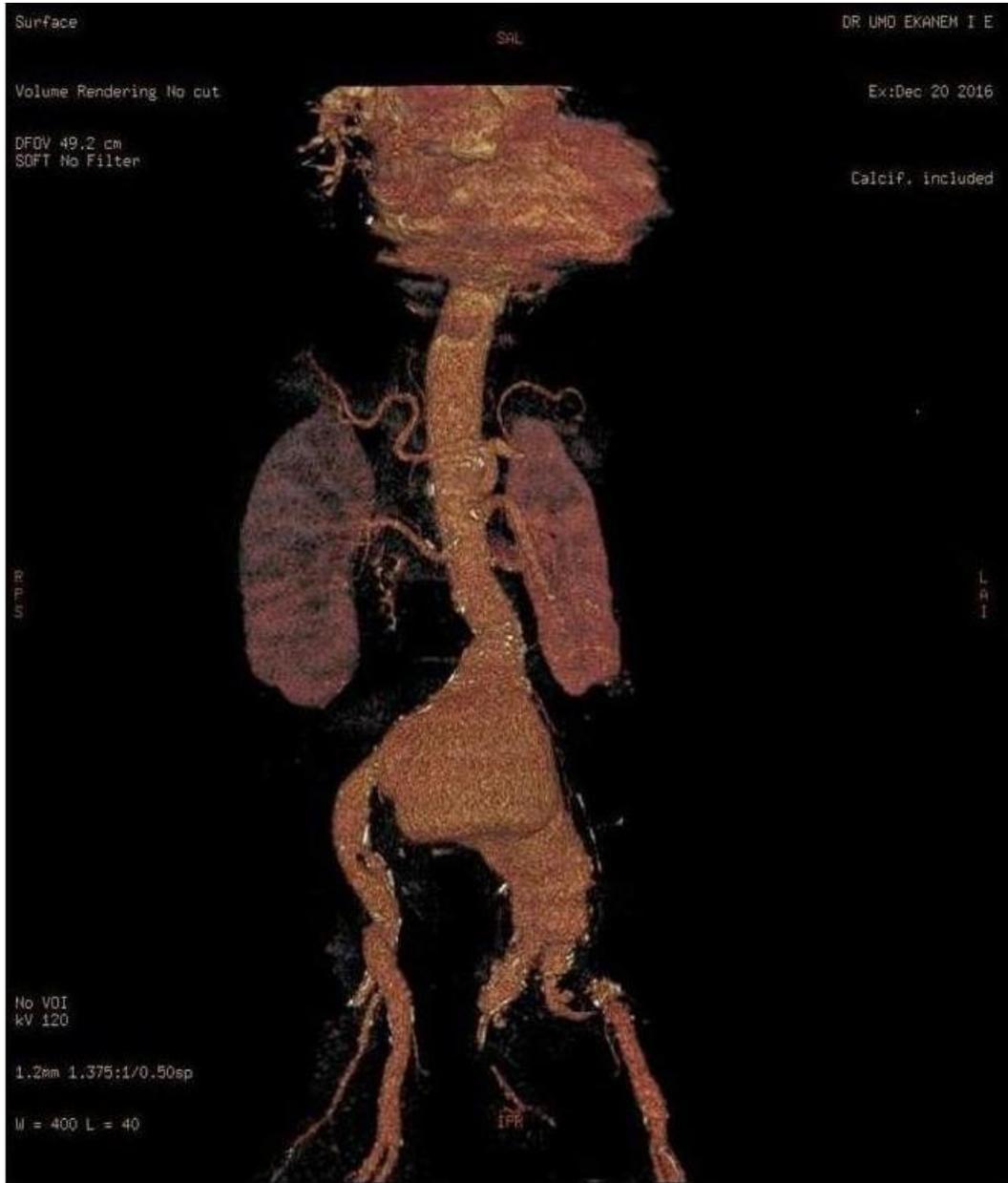


Figure 3. Volume rendering ct-scan image of the abdominal aorta, at the level of bifurcation there is a somewhat fusiform shaped aneurysm of the common iliac, right and left iliac branches.

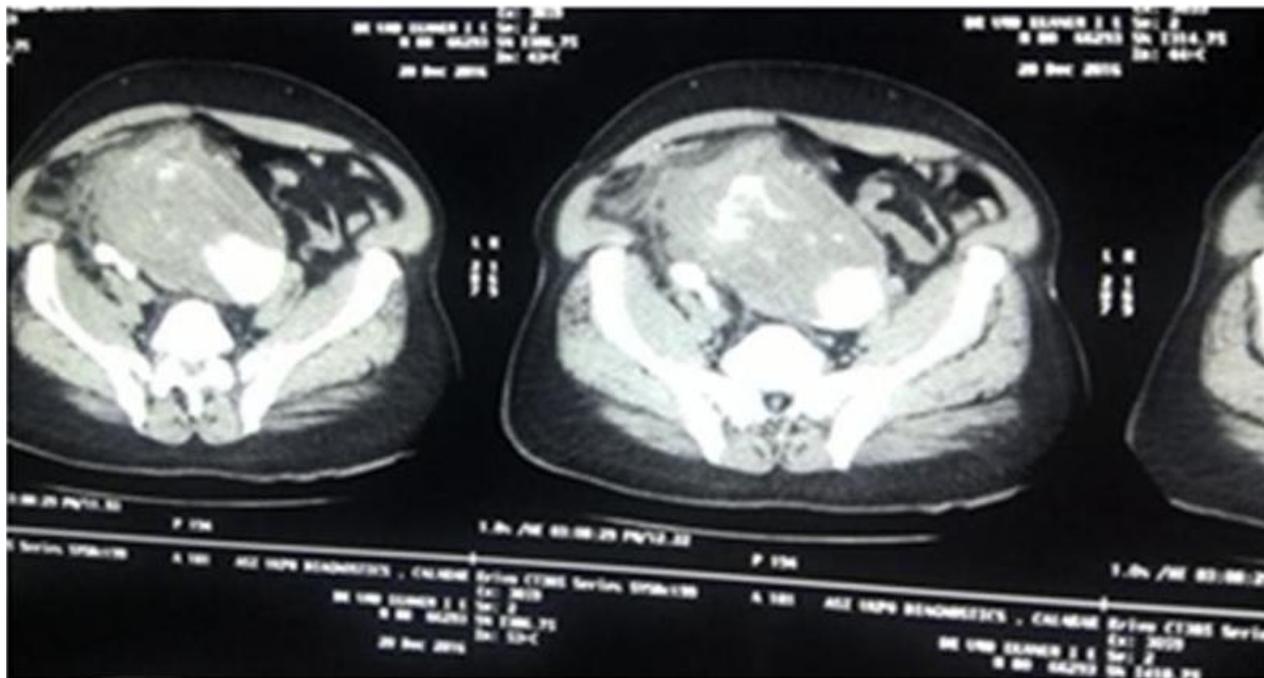


Figure 4. Contrast enhanced axial image of the AAA showing streaks of contrast within the huge thrombus.

Triglycerides: 1.0mmol/L (0.6-1.7mmol/L)

Electrocardiogram showed evidence of first degree atrio-ventricular block, intra-arterial induction delay, moderate mid and left precordial repolarization disturbance suggestive of ischaemic heart disease.

A transfer to cardio-vascular surgeon was planned. However 12 hours after a confirmation of diagnosis, patient collapsed and died.

DISCUSSION

AAA has been defined as a group of diseases characterized by thickening and loss of elasticity of the arterial walls. AAA arises as a result of a failure of the major structural proteins of the aorta (Elastin and collagen) (Mac Sweeney et al., 1994). The cause is not known but a genetic predisposition exists (vanVlijmen et al., 2002). It develops after degeneration of the media leading to widening of the vessel lumen and loss of structural integrity. AAA occurs when the lumen is said to measure 1.5 times the normal or it measures 3cm and above (Louise and Janet, 1999). The normal infra renal aortic dimension being 1.5cm and 1.7cm for women and men respectively (Louise et al., 1999). The widest diameter for the index patient was 12.1 cm and exceeded by far the limit after which a rupture is imminent. About 85% occur below the kidneys and the index patient presented this way (Louise and Janet, 1999).

Predisposing factors include prolonged systemic hypertension (Singh et al., 2001). The index patient

presented with a ten year history of systemic hypertension though it was under control with medication. There was a strong history of tobacco smoking and alcohol intake. Tobacco smoking is said to be the most important behavior related to aortic aneurysm (Singh et al., 2001; Lee et al., 1997). People with history of smoking are 3 to 8 times more likely to develop an AAA. AAA is more common in men (Wong et al., 2007) but affects white men more than blacks aged 65 years and above (Gwrgus-Blake and Wolff, 2005). The index patient was 80 years. Some cardiac abnormalities have also been implicated (Creager and Loscalzo, 2008). The patient had long standing hypertension and atherosclerotic changes depicted on chest x-ray and abdominal CT-Scan.

AAA is usually a symptomless condition (Creager and Loscalzo, 2008). The index patient felt some low back pain and mild abdominal pain not severe enough for him to seek medical advice until the day of presentation. The patient could also feel a pulsatile mass but probably because it caused him not much discomfort he stayed away from hospital for a year before presentation.

With the history of tobacco smoking and alcohol intake, ischaemic heart disease was not surprising. The mainstay of diagnosis is Computerized Angiography (Upchurch and Schaub, 2006; U.S. Preventive Services Task force, 2005). This the index patient did and it confirmed the remarkably enlarged common iliac artery and its terminal branches (Right and Left iliac arteries).

Other investigations that are useful for diagnosis include lumbo-sacral x-rays, which usually show

extensive calcifications throughout the length of the abdominal aorta and aneurysms usually at about the level of the bifurcation (Fleming et al., 2005).

Important clinical findings that suggest impending rupture include; low back ache, or lower abdominal pain and pulsatile abdominal mass (Rakita et al., 2007). The index patient presented with all three before his demise. In-addition he presented with constipation which is likely a compressive effect on the pelvic colon or rectum. Imaging signs of frank rupture include; Retroperitoneal hematoma which can be demonstrated on CT or MRI (Rakita et al., 2007; Siegel et al., 1994). Para-aortic stranding due to hemorrhage into peri renal and posterior renal space. The patient did not present with any of these. Imaging however revealed a huge thrombus within which is evidence of a contained rupture. There was also evidence of sippage of contrast into the contained thrombus.

Recognition of impending signs of rupture is important to avoid fatality of this condition which is up to 50% (Ando et al., 2003). These findings are detected intramurally. Signs of impending rupture are rapid increase in aneurysm size and draped aorta sign. These signs are evident on CT-scan or MRI (Ando et al., 2003).

AAA can be classified under Stanford classification as Stanford Type A- If it affects the ascending aorta and arch and Stanford Type B – if it affects the aorta beyond the origin of the left subclavian Artery. The Index patient therefore presented with Type B.

De Bakey's classification divides AAA into three types:

Type I – involves both ascending and descending aorta.

Type II – Involves ascending aorta alone.

Type III – is the affectation of the descending aorta.

Hence the index patient could be classified under Type III

The goal of treatment for AAA is to prevent rupture by controlling the rate of growth of the aneurysm. Treatment therefore could be surgical and non-surgical.

Non-surgical treatment are measures like; weight loss, quit tobacco smoking, control of Hypertension and diabetes as well as treatment of hyperlipidemia.

Surgical treatment includes an open repair or endovascular approach with placement of stents/grafts. The endovascular approach is minimally invasive and therefore has better prognosis.

The surgical approach is yet to be really established in Nigeria owing to the non-availability of the stents, the support care patient would require during and after the surgery. This therefore gave our index patient a very slim chance of survival since it was detected rather late when a huge thrombus due to a contained rupture had already occurred for about a year. This therefore warrants screening of elderly men at risk with simple abdominal ultrasound and close monitoring of the rate of growth of those who are found to have AAA.

This ensures prompt and proper referral of these patients. This will improve the very poor prognosis of AAA in Nigeria presently.

CONCLUSION

I have presented a case of confirmed ruptured AAA in an elderly male patient with a history of pulsatile abdominal swelling, smoking and alcohol intake and terminally, constipation. Patients with this profile should be suspected of and investigated for AAA.

ACKNOWLEDGEMENT

I wish to acknowledge Professors Emmanuel Ekanem and Ikpeme Ikpeme for reading and correcting the manuscript.

REFERENCES

- Ando M, Igari T, Yokoyama H, Hirono Satokawa, CT (2003). Features of Chronic contained rupture of an abdominal aortic aneurysm. *Ann Thorac cardio-vasc Surg.*: 9: 274-8.
- Creager MA, Loscalzo J (2008). Diseases of the aorta in Fauci AS, Braunwald E, Kasper DL, et al, editors. *Harrison's principles of Internal medicine*. 17th edition Columbus. OH: MC Graw-Hill
- Fauci, A (2008). "242" *Harrison's Principles of Internal Medicine* (17ed). McGraw-Hill professional ISBN 0-07-1466 33-9.
- Fleming C, Whitlock EP, Bell TL, Lederie FA (2005). Screening for abdominal aortic aneurysm: a best-evidence Systematic review for the U.S Preventive Services Task Force *Ann Intern Med.* 142(3): 203-11.
- Gwrgus-Blake J, Wolff TA (2005). Screening for abdominal aortic aneurysm. *Am Fam physician.*; 71: 2154-5.
- Lee AJ, Fowkes FG, Carson MN, Leng GC, Allen PL (1997). Smoking, atherosclerosis and risk of abdominal aortic aneurysm. *Eur Heart J.*; 18:671-676.
- Louise C. Brown, Janet T. Powel (1999). "Risk Factors for Aneurysms rupture in patients kept under ultrasound surveillance". *AmSurg.* 230(3): 289-96; discussion 296-7.
- Mac Sweeney ST, Powell JT, Greenhalgh RM (1994). "Patho genesis of abdominal aortic aneurysm" *B. J. Surg* 81(7): 935-41.
- Ogeng'o JA, Olabu BO, Kilonzi JP (2010). Pattern of aortic aneurysms in an African country. *J Thorac Cardiovasc surg.* 140(4): 797-800.
- Rakita D, Newatia A, Hines JJ et-al. (2007). Spectrum of CT findings in rupture and impending rupture of abdominal aortic aneurysms. *Radographics.* 27(2): 497-507.
- Siegel CL, Cohan RH, Korobkin M, Alpern MB, Courneya DL, Leder RA (1994). Abdominal aortic aneurysm morphology: CT features in patients with ruptured and unruptured aneurysms. *AJR AM J Roentgenol.* 163(5): 1123-9.
- Singh K, BonaaKtt, Jacobsen BK, Bjork L, Solbeg S (2001). Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study. *Troms Ø study Am J. Epidemiol;* 154(3): 236-244.
- Spangler R, Van Pham T, Khoujah D, Martinez JP (2014). "Abdominal emergencies in the geriatric patient". *Int J Emerg Med* 7(1):43.
- U.S. Preventive Services task free (2005). Screening for abdominal aortic aneurysm: Recommendation statement. *Ann Intern. Med;* 142:198-202.
- Upchurch GR, Schaub TA (2006). "Abdominal aortic aneurysms". *AM Fam physician.* 73(7): 1198-204.
- vanVlijmen van, keulen, CJ, Pals G, Rauwerda JA (2002). Familial abdominal aortic aneurysm. *Eur j vascEndovasc Surg.*; 24(2):105-16.
- Wittels K (2011). "Aortic emergencies" *Emerg Med Clin North Am* 29(4): 781-800.
- Wong DR, Willet WC, Rimm EB (2007). Smoking, hypertension, alcohol consumption, and risk of abdominal aortic aneurysm in men. *AM J Epidemiol.* 165(7): 838-845.