

# Concurrent Venous and Arterial Thromboembolic Phenomena in a Patient: A Case Report

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**Abstract:** Thrombo-embolic phenomena are a major cause of morbidity and mortality in the western countries<sup>(5)</sup>. The same is not true in Zambia for some reason we do not see that many patients with this problem. It could be that we actually do not diagnose these cases<sup>(4)</sup>.

Common and serious disorders in which Thrombo-embolic mechanisms participate include: pulmonary embolism, myocardial infarction, cerebral infarction, and infarction of abdominal viscera, which conditions though seen are not common in our setting<sup>(5)</sup>.

Thrombo embolic phenomena usually occur either on the venous side or on the arterial side. It is rare to have it on both the venous and arterial side at the same time.

We present the case of an eighteen year old female patient who presented with symptoms and physical findings of concurrent venous and arterial thrombo embolic phenomena.

**Keywords:** Arterial, Thromboembolic, Venous.

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## 1. INTRODUCTION

We present a patient who developed symptoms of a pulmonary embolism including: chest pain, breathlessness, cough and haemoptysis. She had a history of having periods of breathlessness for a long time as a result she was labeled asthmatic, however throughout her illness there was no strong evidence to show that she had typical symptoms of Asthma there was no family history of asthma and she was not atopic. We suspect the breathless attacks she used to have could have been manifestations of a chronic pulmonary embolic phenomenon.

We postulate that she had venous thrombo embolic phenomenon for some time resulting in pulmonary embolism manifesting as periods of breathlessness labeled as Asthma. This resulted in cardiac strain with notable ECG changes. At one time she went into cardiac failure which caused stasis in the cardiac out flow resulting in thrombosis on the Arterial side of the vasculature which had the fatal result of thrombo- embolic blockade of the blood supply to the stomach and most of the small intestines. The history and the laboratory finding led us to believe that this was a case of reactive thrombocytosis however the possibility of essential thrombocytosis was strong<sup>(5),(6)</sup>. Polyarteritis Nodosa was an unlikely cause of this patient's illness.

### 1.1. Case Report:

We present a young lady who was 18 years. She was admitted complaining of cough which was productive of blood stained sputum, difficulties in breathing, fever and right sided chest pains for four days. She was initially treated with anti malarial drugs and a cephalosporin for four days before the admission.

The past medical history revealed that she was a known asthmatic on aminophylline and was known to get attacks from time to time. There was no history of similar illness in her siblings or any close relatives including her parents. History of contraceptive use or hormone replacement therapy was denied.

On examination, she was ill looking and in respiratory distress. Blood Pressure was 134/93mmHg, Pulse was 130/min, Respiratory rate was 24 breathes /minute, the temperature was 36.2°C. She was unable to complete a sentence without taking a breath. She was not pale. The respiratory system revealed rhonchi especially the right side. Other systems were normal.

A diagnosis of severe asthmatic attack was made and patient received nebulised salbutamol and one dose of hydrocortisone was given. Chest X-Ray showed heterogenous opacities on the right lower lobe (Fig.1) but a closer look at the opacity suggested a triangular lesion more in keeping with an infarct. The attending physician diagnosed the patient as a case of Pneumonia and treated her with cefotaxime and Oxygen therapy.

Full blood count report showed the following findings: Hemoglobin of 13.3g/dl, a total White Cell Count of  $22.7 \times 10^9/l$ . Platelets were  $811 \times 10^9/l$ . Differential count showed eosinophilia. MCV was 64fL and The ESR was 77mm/hr.

The patient did not respond to the above treatment and so Prednisolone was added to the treatment after which she improved and was discharged from hospital after one week.

The patient returned one month later and was still coughing and had developed swelling of legs up to the ankle joint and was producing less urine than normal. Blood pressure at the time was 105/51mmHg, Pulse- 115/min and temp 38°C.

Examination revealed bilateral basal crackles with rhonchi and a gallop rhythm on heart auscultation. There was also bilateral pedal oedema.

Her Hemoglobin was 14.4g/dl, WBC was  $16.5 \times 10^9/l$ , differential showed lymphopaenia and basophilia. Platelet count was not given. Sputum for AFB was negative. BUN and serum creatinine were normal. Ultrasound scan report showed no abdominal pathology. Retroviral test was non reactive. Sickling test was negative. ECG showed sinus tachycardia with ST changes in the form of deep T waves in leads V I to V III.

A diagnosis of congestive Cardiac Failure was made and patient was started on decongestive measures, In addition high dose Cefotaxime six hourly and salbutamol 4mg three times a day were given. A repeat chest X ray showed increased vascular markings and air under the diaphragm on the left (Fig.2).

Second day of admission she developed abdominal pains in the epigastric area. Examination revealed tenderness in the right lumbar area and the hypogastric areas. She further developed pain on passing stool and scanty vaginal bleeding. Clinically this was not a menstrual period.

Treatment continued but the abdominal pains worsened especially after meals. The patient was discharged one week later on Cephalosporin, Frusemide, Prednisolone, paracetamol and hyoscine butyl bromide.

The patient returned the same day with worsening abdominal pain and vomiting. An endoscopy was ordered and patient started on anti acids.

Repeat ultrasound scan reported normal abdominal organs except increased hyperechoic thick bowel walls with reduced peristalsis, suggestive of colitis.

Endoscopy revealed severe erosion of the gastric mucosa and reflux oesophagitis. The patient was put on Omeprazole, Domperidone and Ranitidine.

Liver function tests: Direct bilirubin-190ummol/l, serum protein was 73g/l, ALP was 137U/L, ALT was 17U/L and AST was 86. The LDH was 3482U/L.

The abdominal pain in the patient was not improving despite the treatment. In fact she was losing weight with time. A colonoscopy was advised after the ultrasound report showed signs of colitis.

By this time the patient had started vomiting greenish bowel material and was jaundiced. Later she started having coffee grounds vomitus. A diagnosis of intestinal obstruction was made and patient booked for an exploratory laparotomy.

## 2. LAPAROTOMY FINDINGS

At laparotomy there was: Pus and stool in the abdomen. The small bowel was necrosed and perforated from an area of 40cm from the ligamentum of Treitz to 15cm from the ileo-cecal junction and all the necrosis were on the ante - mesenteric end. The large bowel was normal.

The patient was looked after in ICU until she was stable.

The histology report showed complete transmural infarction of all the layers of bowel, which after is a feature of gangrenous bowel secondary to thrombo-embolic phenomenon in the mesenteric artery.

Repeat full blood count showed a Hemoglobin of 8.3g/dl, the WBC was  $6 \times 10^9/l$ , and platelets were  $110 \times 10^9/l$ , Differential counts were normal.

On the Seventh and fourteenth post-operative days she perforated again and a re-laparotomies were done and three perforations were found in the remaining jejunum and repaired. She was continued on antibiotics (i.e. cefotaxime) and total parenteral feeding was commenced.

The patient died on the twenty fourth post operative day, on which day she had perforated again and went into septic shock. All together she had been being under medical care for four months.

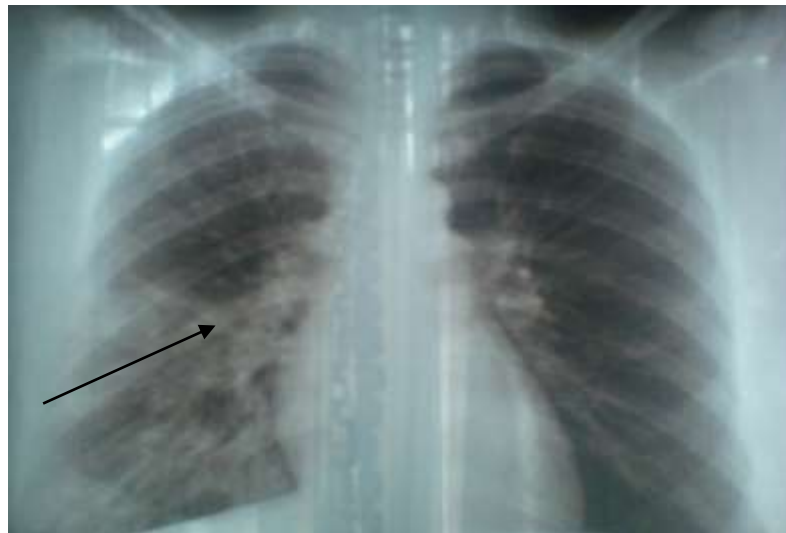


Figure 1. Infarct area



Figure 2. Air under the Diaphragm

### 3. DISCUSSION

It is important to realize that clots develop for many reasons. It is also a fact that embolism may have antecedent causes other than thrombosis. Having looked at the history, physical findings and laboratory findings of our patient, we strongly suspect that there were two probable diagnoses that could have caused the illness and demise of this patient. These were:

- Firstly, thrombocytosis which could have been essential or reactive
- Secondly, Polyarteritis Nodosa<sup>(7),(8)</sup>.

Thrombocytosis or thrombocythemia is the presence of high platelet counts in the blood, and can either be reactive or primary. The primary thrombocytosis is also termed as essential and is caused by a myeloproliferative disease<sup>(9)</sup>. Although often symptomless; particularly when it is a secondary reaction, it can predispose thrombosis in some patients.

In a healthy individual, a normal platelet count ranges from 150,000 and 450,000 per mm<sup>3</sup>, counts over 750,000 per mm<sup>3</sup> (and especially over a million) are considered serious enough to warrant investigation and intervention<sup>11</sup>. Our patient had a platelet count of 800,000 per mm<sup>3</sup> but was never investigated for Thrombosis.

Essential thrombocytosis or primary thrombocythemia, was first described by Epstein and Goedel in 1934. It is a nonreactive, chronic myeloproliferative disorder<sup>(9), (1), (10), (11)</sup>. Essential thrombocytosis is associated with sustained megakaryocyte proliferation that increases the number of circulating platelets. Traditionally, essential thrombocytosis was considered a clonal disorder that involved pluripotent stem cells; however, studies have indicated that some patients may have polyclonal hematopoiesis.

This condition is characterized by a platelet count greater than 600,000/ $\mu$ L, megakaryocytic hyperplasia, splenomegaly, and a clinical course complicated by thrombotic and/or hemorrhagic episodes. Our patient had a count of  $811 \times 10^9$ /L in the early stages of her illness. We could not establish whether these count existed before her illness or not as there were no records to that effect.

The etiology and predisposing factors for the development of essential thrombocytosis remain unclear. Genetic transmission of this disorder is rare, although reports show several families with multiple members affected by essential thrombocytosis (primary thrombocythemia). Research suggests that a thrombopoietin production or receptor abnormality can cause familial essential thrombocytosis. We did not get a positive history of similar illness in the family tree of our patient they never underwent through any test. Four years later we got a report that the girl's mother died following a similar illness suggesting to us that there could have been a primary thrombocythemia in this family.

Clinicians in the United States of America diagnose approximately 6000 cases of essential thrombocytosis (primary thrombocythemia) each year<sup>(10)</sup>. Some researchers speculate that the incidence rate may be several times higher. A study from southeastern Minnesota reported an incidence of 2.38 cases per 100,000 population per year. We have no statistics of this condition in Zambia. It is a challenge for us to think through.

Patients with essential thrombocytosis (primary thrombocythemia) have a 10-year survival rate of 64-80%, which may not be significantly different from that of the age-matched general population. Death occurs from thrombotic complications. Transformation to acute myelogenous leukemia (AML) occurs in 0.6-5% of patients and may accelerate if the patient takes chemotherapeutic agents. Although our patient had a high WBC, Eosinophilia and basophilia at certain points of her illness there was no evidence of a leukemic transformation in her<sup>(6)</sup> Thrombocytosis when caused by an underlying condition. is called reactive thrombocytosis.

Platelets are acute-phase reactants; therefore, they increase in response to various stimuli, including systemic infections, inflammatory conditions, bleeding, and tumors. The finding of a high platelet count and a raised WBC suggested a possibility of reactive thrombocytosis in our patient.

Secondary thrombocytosis (reactive thrombocytosis) may be due to the overproduction of pro inflammatory cytokines, such as interleukin (IL)-1, IL-6, and IL-11, that occurs in chronic inflammatory, infective, and malignant states. The presence of elevated IL-1, IL-6, C-reactive protein (CRP), granulocyte colony-stimulating factor (G-CSF), and granulocyte-macrophage colony-stimulating factor (GM-CSF) in individuals with these conditions suggests that these cytokines may be involved in secondary thrombocytosis (reactive thrombocytosis)<sup>(9),(10),(11)</sup>.

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The causes include the following Infections and inflammatory disorders like bacterial diseases, including Pneumonia, Sepsis, meningitis, Urinary tract infections, and septic arthritis. Other causes are Post splenectomy or hyposplenism, Malignancies like Soft tissue sarcoma and Osteosarcoma, Trauma They may also include Chronic inflammatory conditions like Kawasaki disease, Dermatitis Inflammatory bowel disease, Rheumatoid arthritis Polyarteritis Nodosa and many other conditions.

Our patient did show features of Systemic Inflammatory Response Syndrome like; a total White Cell Count of  $22.7 \times 10^9/l$ <sup>(4)</sup>, a Pulse rate of 130/min, and a respiratory rate of 24 breathes/minute.

No sex predilection exists for secondary thrombocytosis (reactive thrombocytosis), except that iron deficiency it is more prevalent in females during childbearing years<sup>(10)</sup> There was no evidence of Iron deficiency in our patient

It is known that approximately 25-33% of patients with thrombocytosis are asymptomatic at diagnosis. The remainder may experience symptoms relating to Neurologic, large vessel or micro vascular thrombosis and bleeding<sup>(10)</sup>.

Most symptomatic patients present with symptoms that relate to small- or large-vessel thrombosis. Our patient typified this picture: Thrombosis of large veins and arteries is common and may result in occlusion of, leg, coronary, and renal arteries. Other arteries may be involved like the bowel arteries. Our patient had superior mesenteric artery occlusion leading to gangrene of all but 55cm of the small bowel leading to the development of the short bowel syndrome<sup>(3),(9),(10)</sup>.

Venous thrombosis of the splenic, hepatic, or leg and pelvic veins may also develop. Pulmonary hypertension may result from pulmonary vasculature occlusion. Our patient had both cardiac and respiratory symptoms and signs of pulmonary embolism.

The gastrointestinal tract is the primary site of bleeding complications. Approximately 40% of the patients have gastric arcade thrombosis, resulting in sloughing of the gastric and duodenal mucosa, simulating gastric and duodenal ulcers. Our patient had coffee grounds vomitus at one stage suggestive upper GI bleeding The findings at Upper GI endoscopy in our patient showed severe ulceration of the gastric and duodenal mucosa

Other sites of bleeding include the skin, eyes, gums, genitor-urinary tract, joints, and brain. Our patient had vaginal bleeding at one stage. The bleeding is generally associated with a platelet count greater than 1 million/ $\mu$ L. Our patient's platelet count was  $811 \times 10^9/L$ <sup>(9),(10),(11)</sup>

She had no Neurologic symptoms.

There were some features of Polyarteritis Nodosa that we noticed: Of the ten diagnostic criteria she had one which was Weight loss of more than 4 kg. However she had a number of gastrointestinal symptoms that are found in Polyarteritis Nodosa namely;abdominal pain that was postprandial, Nausea, vomiting, Melena, Hematochezia and Diarrhea. She had Gastrointestinal signs of tender abdomen with guarding and diminished bowel sounds and she had Gastrointestinal complications of Intestinal necrosis and perforation<sup>(7),(8)</sup>.

She had Lung involvement which is rare in Polyarteritis Nodosa

Her laboratory findings included the following: white blood cell and platelet counts that were elevated. She also did have an elevated ESR of 77mm/hr but she had no anaemia unlike what pertains in Polyarteritis Nodosa<sup>(4),(7),(8)</sup>.

Urine testing did not show protein and red blood cells in the urine. Her BUN and serum creatinine were normal.

However these signs and symptoms could also have come from the disease process without being from Polyarteritis Nodosa.

#### 4. CONCLUSION

Our patient initially developed symptoms of a pulmonary embolism including: chest pain, breathlessness, cough and coughing up blood in her sputum. It is cardinal to note that she had a history of having periods of breathlessness symptoms for a long time which used to come on from time to time, she was as a result labeled as asthmatic however throughout her illness there was no strong evident to show that she had typical symptoms of Asthma. It is also worth noting that there was no family history of asthma and she was not Atopic.

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Clinically our patient had no known predisposing factors to a thrombo- embolic occurrence like having recently had major hip or knee surgery, she was not pregnant nor had she recently given birth. She had no advanced malignancy at the time she presented and was not confined to bed with a serious illness before her last illness<sup>(1),(6)</sup>. She had no past history of Myocardial Infarction, Cerebral Vascular Accident or Deep Vein Thrombosis. She was not on contraceptive pill, neither was she on hormone replacement therapy. She had not traveled a long distance in her recent past.

Despite the fact that there was no family history of thrombophilia, the occurrence a thrombocytosis during the time of her illness betrays the possibility of some form of thrombocythemia which may have been essential or reactive in addition<sup>(9),(11)</sup>, we suspect her own mother could have had a similar illness. The breathless attacks she used to have could have been manifestations of a chronic pulmonary embolic phenomenon and the clearly evident skin stretch marks showed that our patient was actually an obese person<sup>(6)</sup>.

We postulate that the progression of this young lady's illness was as follows: She has had venous thrombo embolic phenomenon for some time resulting in pulmonary embolism manifesting as periods of breathlessness labeled as Asthma. This resulted in cardiac strain with notable ECG changes<sup>(1),(5)</sup>. At one time she went into cardiac failure which caused stasis in the cardiac out flow resulting in thrombosis on the Arterial side of the vasculature which had the fetal result of thrombo embolic blockade of the blood supply to the stomach and most of the small intestines<sup>(3)</sup>. Looking at the history and the laboratory finding we believe that this was a case of reactive thrombocytosis however the possibility of essential thrombocytosis is strong. Polyarteritis Nodosa was an unlikely cause of this patient's illness<sup>(7),(8)</sup>.

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