ASCARIS LUMBRICOIDES — A CAUSE OF DVT LEADING TO PULMONARY EMBOLISM

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INTRODUCTION

Helminthic infestation, including the roundworm-Ascaris lumbricoides are very common in the tropical countries due to the poor hygienic conditions. The clinical manifestations may range from asymptomatic cases to those with features of allergic reactions, GI symptoms, and mechanical effects; depending upon factors viz., the infesting species, worm load and the host resistance. A patient with GI symptoms who developed deep vein thrombosis (DVT) of the left leg subsequently lead to pulmonary embolism was later on found to have roundworm- Ascaris lumbricoides infestation. This has not been reported so far. The case is reported for its uniqueness and keeping a high index of suspicion for similar cases, due to the high prevalence of worm infestation in our population. The clinical course, investigations and a plausible causal mechanism of pathogenesis is presented.

CASE REPORT

A young, highly educated Pakistani male, of good socioeconomic status who had recently returned home after three years stay in North America presented with frequent loose bowel movements with mucus, but no blood. He also complained of vague discomfort in the left iliac fossa. He was a smoker and had mild hypertension currently controlled with amlodipine 5mg daily. He was treated empirically with anti-amoebic drugs to which he responded. However the symptoms recurred after a few days. This time he was investigated with FBC, urinalysis, stool examination, chest and abdomen X-rays, which except for an ESR of 45-52 mm were all found to be normal. He was given another course of anti-amoebic drugs, but the symptoms reappeared after the cessation of the drugs. Stool examination repeated thrice, was negative for ova and pathogens. The patient’s symptoms continued with remissions and exacerbations after anti-amoebic drugs for about three years. In July 1995, he had an episode of continuous fever, for which he was given antibiotics with little relief. Later on he developed slight cough with left-sided pleuritic chest pain. The chest X-ray showed raised right hemi-diaphragm with evidence of basal atelectasis, a normal ultrasound excluded sub-diaphragmatic pathology. He was given a course of roxithromycin 150 mg BD for a week. A repeat chest X-ray at the
end of treatment still had raised right hemi-
diaphragm. In September, the patient de-
veloped swelling of the whole left leg with
feeling of heaviness, followed by a sudden,
sharp excruciating pain in the leg with
cardiovascular collapse, while walking. He
also had moderate central chest pain that
did not radiate, with no sweating, vomiting,
cough or haemoptysis. He was immediately
admitted to CCU, with a clinical diagnosis
of DVT with massive pulmonary embolism.
The ECG showed right axis deviation, with
an S1 Q111↓ T111 pattern pathognomonic
of pulmonary embolism. The cardiac en-
zyme were within normal limits ruling out
acute myocardial infarction or ischaemia.
The DVT was confirmed by doppler
ultrasound and CT scan which showed a
thrombus measuring 1.0×1.3 cm in the left
femoral vein, with evidence of extension
upto the transition into left external iliac
vein. A small right pleural effusion and
minimal ascites posterior to the right lobe
of the liver were also seen. Anticoagulant
therapy was started, initially with heparin;
followed by warfarin with prothrombin time
monitoring to keep the INR within range of
2-3, and was continued for 6 months. He
was also given Inj. Ceftriaxone one gm daily
for 10 days with supportive therapy. The
patient was discharged after 10 days. The
99m Tc-MAA (5 mCu) lungs scan showed
normal perfusion. Although the doppler
ultrasound showed resolution of the throm-
bus, the swelling of the limb did not subside
clinically. The GIT symptoms after this
episode remitted for about 2 months, but
gradually returned. The stools were again
negative for parasites.

In view of the repeated bouts of GIT
disturbances with negative results of inves-
tigations, he was treated as a case of irritable
bowel syndrome, but to not much benefit.
One morning while defecating, the patient
noticed the passage of a long, motile worm
per rectum, which he brought to his
physician; later on identified to be a female
Ascaris lumbricoides. After this incidence,
the patient observed steady relief from the
GIT discomfort and gradual disappearance
of swelling of the leg.

**DISCUSSION**

Ascaris is the commonest soil-transmit-
ted intestinal helminth in man. It is endemic
in many regions of Southeast Asia, Africa,
Central and South America, infecting 25% of
population and 20,000 annual mortality
globally. From the ingested eggs, the
larvae hatch in the duodenum, pass through
the intestinal wall, liver and diaphragm into
the lungs, moulting twice on the way to
mature into adult worms, crawl up the trachea,
get swallowed to reach their final
habitat in the ileum. The clinical features
depend upon the stage of life cycle, during
the tissue phase of migration, they provoke
immunological and inflammatory reactions
e.g.; transient hepatomegaly, pneumonitis,
pulmonary infiltrates and eosinophilia-
Loeffler's syndrome, bronchial asthma and
urticaria. During the intestinal phase they
cause morphological changes in the intestine
e.g., hypertrophy of the muscle layer and
physiological changes e.g., anorexia, in-
creased intestinal motility, malabsorption
(e.g. of vitamin A), lactase deficiency, vague
abdominal pain. The symptoms also
depend on worm load and heavy worm
burden may lead to malnourishment due to
spoliative action.

The triad of abdominal symptoms, DVT
and pulmonary embolism, can be explained
by the solitary Ascaris, which had aberrantly
lodged in the sigmoid colon. Here it evoked
inflammation of the gut, responsible for the
diarrhoeal symptoms. The close anatomical
proximity of the sigmoid colon to the left
external iliac vein gave rise to phlebitis and
thrombosis of the later leading to DVT,
which resulted in pulmonary embolism. The
repeated negative stool results were prob-
ably due to the solitary female being either non-gravid, low egg-count—beyond the limit of detection (due the single worm), or rapid disintegration of the unfertilized ova in the feces. The sojourn of the worm to the sigmoid colon is not surprising as Ascaris are very motile and are known to migrate spontaneously or when stimulated by anesthetics or drugs e.g., mebendazole etc., from their normal habitat to distant sites e.g., stomach where may be vomited out, passed up along the oesophagus, coming out through the mouth or nose, pass down the respiratory passages, where may block the glottis or bronchus, resulting in sudden death due to suffocation. Wandering worms may enter appendix causing appendicitis. Obstructive jaundice, acute haemorrhagic pancreatitis are known to occur if the worm traverses the pancreatico-biliary tree. It has even been reported from the brain causing brain abscess.

The negative perfusion scan is explainable by the delay (10 days), when resolution of clot is expected, moreover the sensitivity of V/Q scanning is quite low being truly diagnostic in only 30% cases. Spiral CT (sensitivity 95% specificity 97%) which is non-invasive and diagnostic power approximating the pulmonary angiography, are unfortunately unavailable, as are D-dimers in our setting.

This is presumably the first case report of Ascaris lumbricoides causing DVT leading to pulmonary embolism, as an extensive literature search could not find any similar report.

REFERENCES