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Eosinophilic Pleural Effusion and Pericardial Effusion Suspected Secondary to *Paragonimus* Infection: A Case Report

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ABSTRACT

Eosinophilic pleural effusion (EPE), defined as pleural fluid containing more than 10% eosinophils, represents a diagnostic challenge due to its diverse etiologies, including infections, malignancy, trauma, pulmonary embolism, and drug reactions. Parasitic infections are an uncommon but important cause, particularly in endemic regions. Paragonimiasis, caused by lung flukes of the genus *Paragonimus*, is a food-borne parasitic disease acquired through ingestion of raw or undercooked freshwater crustaceans. Pulmonary involvement may present with cough, hemoptysis and pleural effusion, often mimicking tuberculosis or malignancy.

We report a case of an 85-year-old female who presented with fever, pleuritic chest pain, cough, and dyspnea. Imaging revealed left-sided pleural effusion and pericardial effusion. Pleural fluid analysis demonstrated an exudative eosinophilic pleural effusion with significant eosinophilia. Extensive evaluation for bacterial infection, tuberculosis, and malignancy was negative. A detailed dietary history revealed consumption of undercooked freshwater crab; however, serological testing could not be performed due to regional diagnostic limitations in laboratories across Nepal. The patient was successfully treated with praziquantel, resulting in complete clinical and radiological resolution.

This case highlights the importance of considering paragonimiasis in the differential diagnosis of eosinophilic pleural effusion, particularly in endemic areas. Early recognition and appropriate treatment can prevent misdiagnosis and unnecessary invasive investigations.

Keywords: Hypereosinophilia; Eosinophilic Pleural Effusion; *Paragonimus*; Pulmonary Tuberculosis; Pericardial Effusion; Parasitic Zoonosis; Food-Borne Trematodiases; Exudative Effusion; Praziquantel; Case Report.

1.0. Introduction

Eosinophilic pleural effusion (EPE) is defined as pleural fluid containing 10% or more eosinophils among the total nucleated cells [1]. EPE accounts for approximately 5–16% of all pleural effusions and is associated with a wide spectrum of underlying conditions. Common causes include malignancy, infections, pulmonary embolism, trauma, pneumothorax, hemothorax, and drug reactions [2].

Parasitic infections are relatively uncommon causes of EPE but should be considered in endemic regions [3]. Among these, paragonimiasis, a food-borne parasitic infection caused by lung flukes of the genus *Paragonimus*, is an important yet frequently overlooked etiology [4].

Humans acquire the infection by ingesting raw or undercooked freshwater crabs or crayfish containing infective metacercariae. After ingestion, the larvae penetrate the intestinal wall, migrate through the peritoneal cavity and diaphragm, and eventually reach the lungs where they mature into adult worms. This migration can result in pleural inflammation, pleural effusion, pneumothorax, and pulmonary parenchymal lesions [5].

Pulmonary paragonimiasis can clinically and radiologically mimic tuberculosis, bacterial pneumonia, or lung malignancy, which may lead to delayed diagnosis [6]. We report a case of eosinophilic pleural effusion and pericardial effusion due to *Paragonimus* infection that was successfully treated with praziquantel.

1.1. Study Objectives

1) To highlight *Paragonimus* infection as a critical differential diagnosis in patients presenting with concurrent peripheral hyper-eosinophilia and exudative eosinophilic pleural effusion.
2) To detail the clinical diagnostic pathway utilized in resource-limited environments where confirmatory serological and molecular assays are unavailable.
3) To emphasize the clinical relevance of gathering thorough food consumption and epidemiological histories during baseline medical evaluations.
4) To describe the unusual clinical presentation of concurrent pericardial effusion and pleural effusion within pulmonary paragonimiasis.
5) To document the therapeutic efficacy and rapid radiological resolution associated with standard weight-based praziquantel therapy.

2.0. Literature Review

The existing medical literature characterizes EPE as an indicator of an underlying pulmonary insult rather than a specific diagnosis. Historically, its discovery was considered an indicator of benign pathology, but multi-center updates demonstrate a high correlation with underlying primary lung malignancies, lymphoproliferative disorders, and pulmonary manifestations of systemic connective tissue diseases [7,8].

Among infectious vectors, *Paragonimus westermani* stands out across South and East Asia as a primary source of parasitic lung disease [9]. Recent systematic reviews emphasize that larvae migrating through host tissues cause profound localized type-2 helper T-cell responses, yielding high tracks of eosinophilic deployment within the pleural space [10]. Literature tracks emphasize that misdiagnosing this fluke infection as pulmonary tuberculosis or adenocarcinoma is remarkably common due to matching findings of chronic productive cough, hemoptysis, low-grade pyrexia, and localized exudative fluid collections on chest radiographs [11,12].

When diagnostic tracking via egg isolation in sputum or stool output fails, clinician teams rely heavily on exposure background data or serological antibody panels [13]. In geographic boundaries without mature serodiagnostic testing networks, clinical case criteria rely on standard diagnostic exclusion pathways coupled with targeted therapeutic trials using specific anthelmintics [14,15]. Recent reports from 2025 and 2026 further emphasize tracking rare extrapulmonary migrations, showing that ectopic worm localization can compromise cardiovascular or central nervous system structures, requiring high vigilance during initial system surveys [16].

3.0. Methodology

This evaluation utilizes a single-patient clinical retrospective case design following a clear diagnostic exclusion network. A complete timeline tracking clinical presentation, biochemical and metabolic updates, cytological profiles, and imaging surveys was generated from the electronic records system of Bir Hospital, Nepal. Diagnostic thoracentesis was performed under aseptic conditions to collect fluid for physical, biochemical, and microbiological evaluation. Standard automated methodologies quantified the complete blood counts and

peripheral blood differentials, while automated colorimetric assays monitored serial liver enzymes and renal function indices.

Microbiological analysis of the pleural fluid included Gram staining, aerobic bacterial cultures, Acid-Fast Bacilli (AFB) smear tracking, and molecular screening via the GeneXpert Mycobacterium tuberculosis/Rifampicin (MTB/RIF) assay. Cytological preparations evaluated the cellular differentials and screened for malignant mutations or atypical cells. Because confirmatory serological testing for *Paragonimus* antibodies was structurally unavailable within the domestic laboratory network of Nepal, a presumptive clinical diagnosis framework was executed. This protocol integrated documented hyper-eosinophilic parameters, confirmed exposures to freshwater crustaceans, exclusion of matching alternative diagnoses, and objective, quantified tracking of clinical and imaging responses to a specific therapeutic trial of praziquantel.

4.0. Results and Discussions

4.1. Case Presentation

We present an 85-year-old female from Nepal with a past medical history of Chronic Obstructive Pulmonary Disease (COPD) managed with a tiotropium inhaler, who presented with two weeks of progressive dyspnea, cough, and fever.

Eleven days before admission, she developed shortness of breath, gradual in onset, which progressed day by day. She also developed an intermittent cough, mostly dry in nature, associated with pleuritic chest pain. In addition, she developed fever with chills. On admission, she denied weight loss, night sweats, hemoptysis, and reported no known sick contacts. She did not smoke cigarettes or use vaping products recently. On further questioning, the patient reported consumption of undercooked freshwater crabs a few months ago.

On physical examination, the patient was febrile with a temperature of 38.2°C. Respiratory examination revealed decreased breath sounds and dullness to percussion over the left lower lung field. Oxygen saturation was 90% on room air. Cardiovascular examination revealed an irregularly irregular pulse and muffled heart sounds.

Initial laboratory evaluation revealed leukocytosis with significant peripheral eosinophilia and elevated systemic inflammatory markers (Tables 1-3).

Table 1. Serial hematologic profile tracking leukocytes, hemoglobin levels, and shifting peripheral eosinophil counts over the initial three days of hospitalization.

Date	White Blood Cells	Eosinophils (%)	Hemoglobin (g/dL)	Platelets	Others (%)
Day 1	13,840	50%	13.4	280,000	Neutrophils: 34%; Lymphocytes: 12%
Day 2	10,160	25%	13.0	362,000	Neutrophils: 50%; Lymphocytes: 18%
Day 3	8,900	21%	11.3	352,000	Down-trending eosinophil count
Reference Range	4.0–11.0 $\times 10^3/\mu L$	1–6%	12–18	150–450 $\times 10^3/\mu L$	—

Table 2. Comprehensive metabolic and thyroid profile monitored during the acute phase of evaluation.

Day	Sodium (mEq/L)	Potassium (mEq/L)	Creatinine (mg/dL)	Glucose (mg/dL)	Calcium (mg/dL)	AST (U/L)	ALT (U/L)	ALP (U/L)	Thyroid Function Test (TFT)
Day 1	145	4.2	1.10	72 (Fasting)	8.0	21	17	70	Free T3: 1.54 pg/mL
Day 2	136	3.5	0.92	—	—	—	—	—	Free T4: 1.15 ng/dL
Day 3	139	3.3	0.90	90	8.2	45	24	47	TSH: 2.16 IU/mL
Reference Range	135–145	3.5–5.0	0.6–1.3	70–99	8.5–10.5	10–40	7–56	44–147	fT3: 1.5–3.9; fT4: 0.7–1.48; TSH: 0.35–4.94

Key: *AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; ALP: Alkaline Phosphatase; TSH: Thyroid-Stimulating Hormone.*

Table 3. Acute inflammatory markers and cardiac biomarker parameters obtained at initial presentation.

Markers	Serum LDH (IU/L)	Serum Protein (g/dL)	CRP (mg/L)	ESR (mm/hr)	CK-MB (U/L)	Troponin
Day 1	542	7.3	75.74	33	3	Negative
Reference Range	225–450	6.0–8.0	0–5	0–20	0–24	Negative

Key: *LDH: Lactate Dehydrogenase; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; CK-MB: Creatine Kinase-MB.*

4.2. Diagnostic Imaging and Thoracentesis

Chest radiography demonstrated a left-sided mild pleural effusion and marked cardiomegaly (Figure 1). Computed Tomography (CT) of the chest showed mild bilateral pleural effusion, features consistent with right heart failure and pulmonary arterial hypertension, moderate pericardial effusion, and subsegmental atelectasis within the medial segment of the right middle lobe and inferior lingular segment of the left upper lobe (Figure 2). Transthoracic echocardiography (ECHO) confirmed the presence of a moderate pericardial effusion without immediate tamponade physiology (Figure 3).

Diagnostic thoracentesis was performed to extract fluid from the pleural compartment. Pleural fluid examination demonstrated:

- Appearance: Straw-colored

- Protein: 4.8 g/dL
- Lactate Dehydrogenase (LDH): 431 U/L
- Glucose: 69 mg/dL
- Adenosine Deaminase (ADA): 23 U/L
- Total Leukocyte Count (TLC): 2,900 cells/mm^3
- Differential Count: Eosinophils 95%, Lymphocytes 5%

According to Light's criteria, these findings were consistent with an exudative eosinophilic pleural effusion. Pleural fluid Gram stain, bacterial culture, acid-fast bacilli (AFB) staining, and GeneXpert assays were all negative. Cytological analysis revealed no malignant or atypical cells.

Given the concurrent presence of profound peripheral eosinophilia and pleural fluid hyper-eosinophilia, an active parasitic infection was highly suspected. Serological testing for *Paragonimus* species was unavailable at our institution and is not routinely performed across diagnostic centers in Nepal.

Consequently, the diagnosis was established based on the combined criteria of an exudative eosinophilic pleural effusion, peripheral eosinophilia, a documented clear history of consuming undercooked freshwater crabs, the systematic exclusion of competing common causes like tuberculosis and malignancy, and the rapid clinical and radiological improvement observed following directed antiparasitic therapy.

4.3. Imaging Documentation

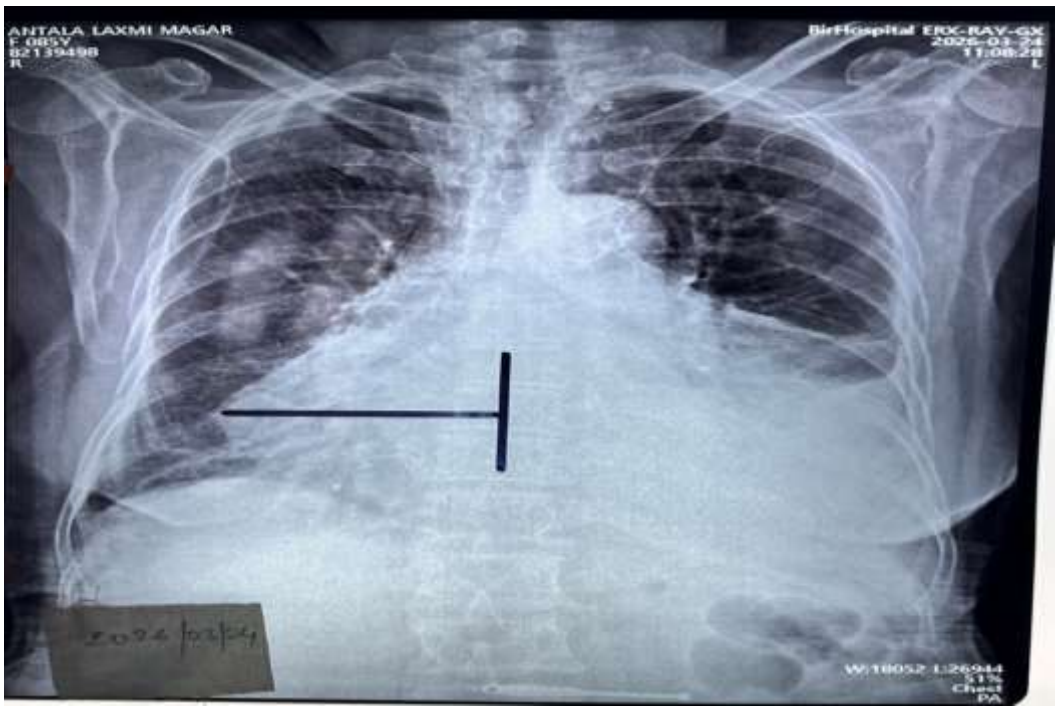

Figure 1. Posterior-anterior (PA) chest radiograph taken at presentation demonstrating a moderate left-sided pleural effusion with blunting of the left costophhenic angle, alongside marked cardiomegaly.

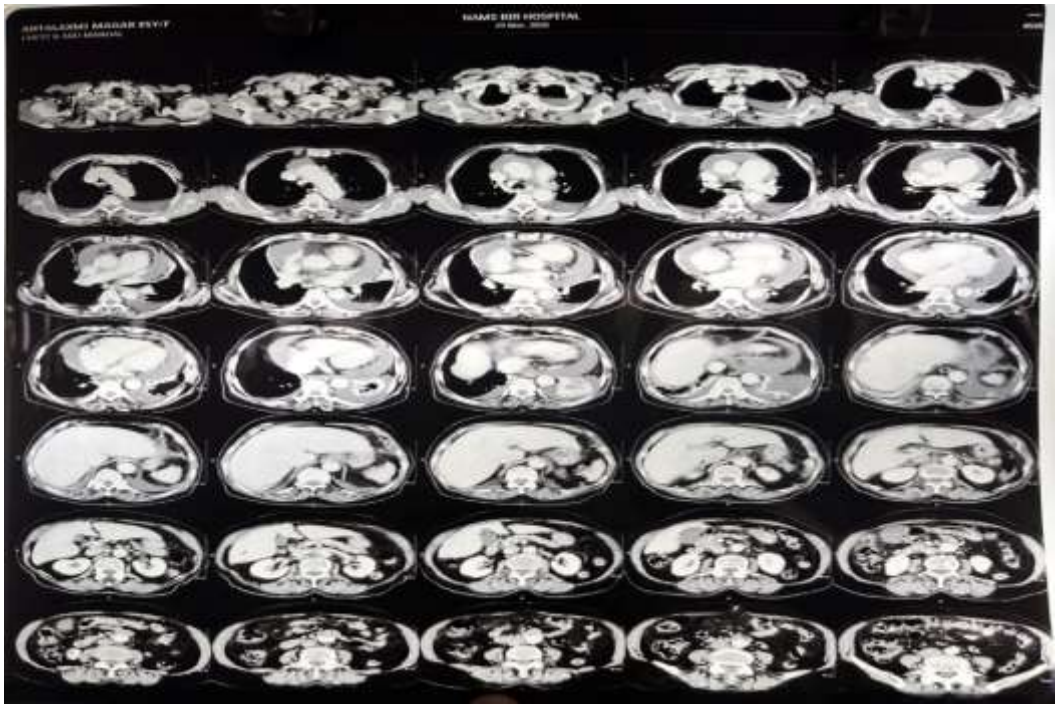

Figure 2. Contrast-enhanced computed tomography (CECT) scan of the chest and abdomen demonstrating a bilateral pleural effusion with adjacent passive lung atelectasis, severe cardiomegaly, and a gross pericardial effusion.

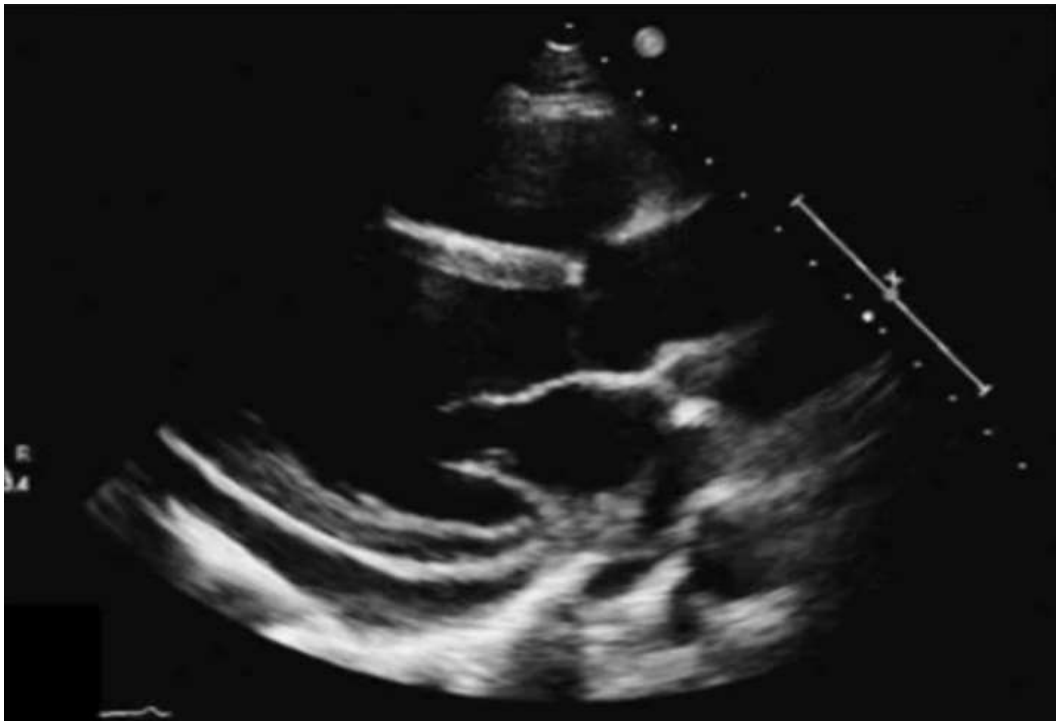

Figure 3. Transthoracic echocardiography (ECHO) report sheets verifying structural dimensions and confirming a moderate pericardial effusion (posterior pocket depth of 1.0 cm).

4.4. Discussions

Eosinophilic pleural effusion represents a significant clinical challenge due to its broad differential diagnosis [7,8]. Traditionally, the presence of elevated eosinophils within the pleural fluid was thought to suggest an

uncomplicated, benign etiology; however, modern series have shown that primary lung malignancies and metastatic disease remain common causes of EPE.

The mechanisms leading to eosinophilic pleural effusion include:

- Air or blood entering the pleural space (e.g., secondary to pneumothorax, hemothorax, or repeated thoracenteses).
- Parasitic or fungal infections.
- Hypersensitivity reactions and systemic drug-induced lung injury.
- Chronic inflammatory responses.

Parasitic infections account for a small proportion of overall EPE cases but are highly relevant in endemic areas of Asia [9]. Paragonimiasis is caused by trematodes of the genus *Paragonimus*, most commonly *Paragonimus* westermani. Humans acquire the infection through the ingestion of raw or undercooked freshwater crustaceans containing infective metacercariae. In addition, paragonimiasis can be acquired by ingesting raw meat from carnivorous animal paratenic hosts that contain young flukes (such as wild boars or deer). Transmission has also been reported via contaminated utensils, such as knives or chopping boards.

Following ingestion, the larvae migrate through the intestinal wall and diaphragm to reach the pleural cavity and the lung parenchyma. During this tissue migratory phase, patients routinely develop severe pleuritic chest pain, pleural effusion, and marked peripheral eosinophilia [10]. Pulmonary paragonimiasis may present with symptoms including chronic cough, hemoptysis, chest pain, fever, and progressive dyspnea [10]. Radiologic findings may include pleural effusion, pneumothorax, pulmonary nodules, or cavitary lesions [11]. These presentations can match pulmonary tuberculosis or bronchogenic malignancy, which often delays accurate diagnosis [12].

Diagnosis may be established by the detection of *Paragonimus* eggs in sputum or stool samples, directed serological antibody testing, specific imaging findings, detailed pleural fluid analysis, and an explicit epidemiological exposure history [13]. In many clinical scenarios, serologic testing plays an important diagnostic role when fluke eggs cannot be identified in raw excretions.

In many resource-limited settings, confirmatory diagnostic tests for paragonimiasis, such as serological enzyme-linked immunosorbent assays (ELISA) or molecular methods, are not available. Consequently, the diagnosis is often made based on clinical presentation, epidemiological exposure records, laboratory findings including hyper-eosinophilia, and a rapid response to targeted antiparasitic therapy. Several reports from endemic regions have described presumptive diagnoses of paragonimiasis using similar clinical criteria when confirmatory testing was unavailable [14].

Extrapulmonary paragonimiasis can occur when immature flukes migrate to extrapulmonary tissues, such as the brain, abdomen (including the intestinal wall, liver, spleen, pancreas, kidney, adrenal glands, peritoneal cavity, ovaries, and mesenteric lymph nodes), and subcutaneous tissues. Other ectopic localizations, such as the heart, mediastinum, striated muscle, spinal cord, parotid gland, testes, and breasts, have also been described [15]. In this

patient, the concurrent presentation of a moderate pericardial effusion alongside the eosinophilic pleural fluid highlights the potential for multi-serosal reactions or direct regional migration of the parasite. Praziquantel remains the clear treatment of choice, with cure rates exceeding 90%. Early administration typically results in rapid clinical improvement and complete resolution of pleural and parenchymal abnormalities [16].

4.5. Treatment and Clinical Outcome

The patient was treated with oral praziquantel at a dose of 25 mg/kg three times daily for three consecutive days, which is the established standard therapy for adult paragonimiasis.

The patient experienced significant clinical improvement in her primary respiratory symptoms within one week of initiating treatment. A follow-up chest radiograph performed four weeks later demonstrated complete resolution of the left-sided pleural effusion and an improvement in the overall cardiomegaly. At her one-month follow-up evaluation, the patient remained entirely asymptomatic with no clinical or radiological recurrence of effusion.

4.6. Limitations

This case has certain limitations. Confirmatory diagnostic testing for *Paragonimus* infection, including targeted serological assays and polymerase chain reaction (PCR) tests, could not be performed because these tests are not available within our regional laboratory setting. Therefore, the diagnosis in this case was presumptive, relying on clinical presentation, an exudative eosinophilic pleural effusion, a matching dietary exposure history, the exclusion of competing etiologies, and a rapid, complete response to praziquantel therapy.

5.0. Conclusion and Future Recommendations

Paragonimus infection is an uncommon but important cause of exudative eosinophilic pleural effusion. In endemic countries, clinicians should maintain a high index of suspicion for parasitic zoonoses when evaluating patients presenting with EPE, particularly when accompanied by profound peripheral eosinophilia and a clear history of consuming raw or undercooked freshwater crustaceans. Early diagnosis and prompt administration of praziquantel yield excellent outcomes and prevent unnecessary invasive procedures or toxic medications linked to misdiagnoses of tuberculosis or malignancy.

5.1. Future Recommendations

• Diagnostic Infrastructure Development: Establish specialized regional reference laboratories capable of performing enzyme-linked immunosorbent assays (ELISA) and molecular assays for parasitic infections in Nepal to reduce reliance on presumptive treatment.

• Public Health Education Campaigns: Implement community-level health literacy programs regarding the risks of eating raw or undercooked freshwater crabs and crayfish, focusing on proper food preparation techniques.

• Epidemiological Food Surveys: Conduct structured field research and vector-host profiling within local freshwater streams to identify endemic hotspots for *Paragonimus* metacercariae.

• Standardized Clinical Guidelines: Formulate and distribute national clinical diagnostic algorithms for managing unexplained hypereosinophilia and eosinophilic pleural effusions within resource-limited community hospitals.

• Routine Dietary Screenings: Mandatory inclusion of detailed dietary history checklists—specifically focusing on raw or undercooked crustacean and wild game consumption—in the baseline diagnostic workup of atypical pulmonary effusions.

Declarations

Source of Funding

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Competing Interests Statement

The authors declare that they have no competing financial or personal interests that could have influenced the work described in this paper.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this clinical case report and any accompanying images.

Authors' Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Prasanna Bahadur Amatya.

Acquisition, analysis, or interpretation of data: Prasanna Bahadur Amatya.

Drafting of manuscript: Prasanna Bahadur Amatya.

Critical review of the manuscript for important intellectual content: Milan Bajracharya.

Supervision: Shree Narayan Yadav.

Informed Consent

Informed consent was obtained directly from the patient prior to detailing the clinical case sequence and abstracting diagnostic data files.

Availability of Data and Material

The clinical data, laboratory metrics, and imaging records utilized to support the findings of this case report are included within the manuscript text and figures.

Institutional Review Board Statement

Not Applicable.

Ethical Approval

Institutional ethical approval was not required for this single-patient case report as per regional guidelines; however, full patient anonymity was preserved throughout.

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Declaration of Artificial Intelligence

Artificial intelligence tools were used solely to improve grammatical structure, proofread language clarity, and formatting alignment in accordance with the journal's editorial revision requests. No clinical data or text was fabricated.

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