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CASE REPORT

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Acute myocardial infarction with e-cigarette or vaping-use associated lung injury in a young Filipino vape user

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Abstract

Electronic cigarettes (or e-cigarettes) and vape products have multisystemic adverse effects despite being advertised as a safer smoking alternative and cessation device. We present a 22-year-old Filipino male with sudden chest pain. He had no known comorbidities but had a two-year history of daily vape use. Work-up revealed elevated cardiac markers, anteroseptal ST-elevation myocardial infarction, hypokinesia of the anterior wall and interventricular septum, and an ejection fraction of 30%. Chest radiography showed consolidation pneumonia but culture studies and Biofire Pneumonia Panel were negative for microbial detection. Coronary angiography revealed chronic total obstruction of the mid-left anterior descending (LAD) and right coronary arteries (RCA). Percutaneous coronary angioplasty of the LAD was done. The patient eventually required mechanical ventilation for progressive respiratory distress but expired after three hospital days despite medical management. This case highlights a possible association between vape use and the development of both acute lung injury and myocardial infarction.

KEYWORDS
acute myocardial infarction, E-cigarette or vape use associated lung injury

INTRODUCTION

E-cigarettes and vaping have been shown to have adverse health outcomes including e-cigarette or vaping-use associated lung injury (EVALI) as well as elevated risk for acute myocardial infarction and stroke.[1,2] A number of case reports have documented simultaneous EVALI and cardiomyopathy while one study by Abusharekh and colleagues documented the incidence of both single vessel coronary artery disease and EVALI in a dual conventional smoker and e-cigarette user.[3–5] In this article, we present a case of simultaneous anteroseptal ST-elevation myocardial infarction, two vessel coronary artery disease and EVALI in a young, Filipino male vape user with no history of conventional cigarette smoking.

CASE REPORT

A 22-year-old Filipino male with no known comorbidities presented at the emergency room with a two-day history of sudden onset severe chest pain after sports activity, associated with dyspnea, diaphoresis, and myalgia. This was preceded by a one-week history of productive cough, hemoptysis, fever, and vomiting. He had a two-year history of daily vape use. He denied a history of cigarette smoking, alcohol intake, or illicit drug use. The patient had no prior infection with coronavirus disease 2019 (COVID-19).

On admission, heart rate was 111 beats per minute, blood pressure was 98/64 mmHg, respiratory rate was 24 cycles per minute, temperature was 36.6°C, and oxygen saturation was 87% on room air with improvement to 99%

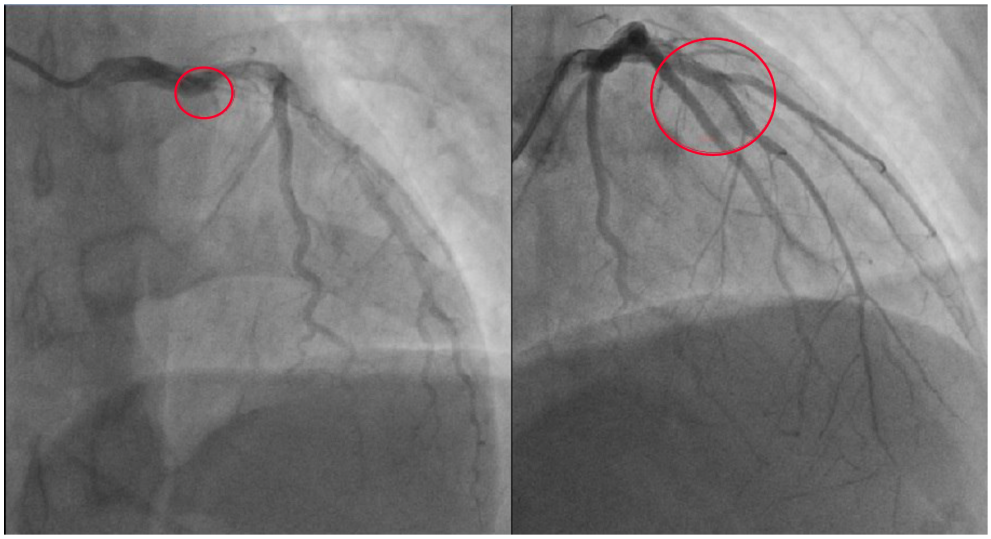

FIGURE 1 Cranial projection images showing pre (left) and post (right) percutaneous coronary angiography with reconstruction of LAD flow. Red circle on the left image denotes a blunt stump indicative of chronic total obstruction and larger red circle in the right images shows well deployed stent at the proximal to mid LAD.

on 8 litres per minute of oxygen support via face mask. Bibasal crackles were noted on auscultation. A 12-lead electrocardiogram showed ST-segment elevation in leads V1-V4, troponins were 3786 times elevated at 45,440 pg/mL (upper normal limit of 9 pg/mL), and transthoracic echocardiography (TTE) revealed a dilated left ventricle and hypokinesia of the basal to mid-interventricular septum and anterior wall, with a depressed overall systolic function (ejection fraction of 30%–35%).

Complete blood count showed leukocytosis with neutrophilic predominance (WBC count at 17.2×10^3/mL, 79% neutrophils), haemoglobin levels were at 15.1 gm/dL and platelet count was at 348000/mL. Chest radiography (CXR) showed airspace opacities with consolidation on the right upper and middle lung fields consistent with pneumonia. Procalcitonin was not elevated (0.08 ng/mL), COVID-19 was not detected on reverse transcription polymerase chain reaction, and no other infectious organisms were detected on culture studies and Biofire FilmArray Pneumonia Panel Plus. Screening tests for Human Immunodeficiency Virus, Antinuclear Antibody, urine cannabinoid and methamphetamine were negative.

Coronary angiography showed chronic total obstruction of the mid-LAD and RCA. Culprit-vessel percutaneous coronary angioplasty of the proximal to mid-LAD was done with resultant TIMI 3 flow of the LAD and improved visualization of the RCA via left to right collaterals (See Figure 1).

Six hours post-procedure, the patient developed hypotension as low as 80/60 mmHg, hemoptysis, and respiratory distress prompting invasive mechanical ventilation and initiation of norepinephrine. Repeat CXR showed progression of right sided consolidation pneumonia and computed tomography pulmonary angiography documented progressive pneumonia, bilateral pulmonary infiltrates, extensive pneumomediastinum, ground glass opacities with interlobular septal thickening, and subcutaneous

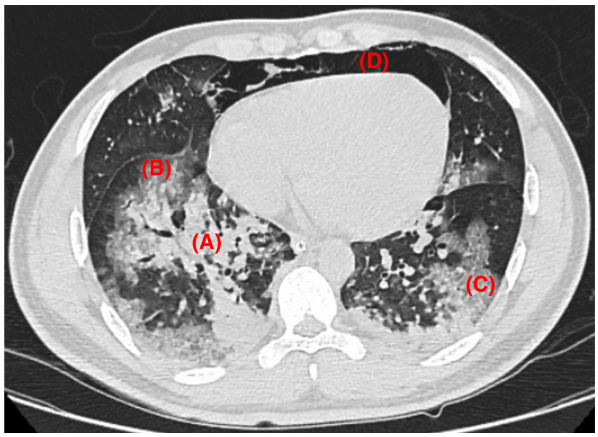

FIGURE 2 Chest computed tomography lung window axial view. (A) Extensive consolidation, (B) patches of ground glass opacities with septal thickening, (C) crazy paving pattern with peripheral distribution, and (D) extensive pneumomediastinum with pneumopericardium.

emphysema with no pulmonary embolism (See Figure 2). Repeat electrocardiogram showed no dynamic changes and repeat TTE revealed improvement in the ejection fraction to 47% with grade 1 diastolic dysfunction. Post-procedural mechanical complications such as papillary muscle rupture, ventricular septal rupture and tamponade were ruled out as well from the TTE. Left sided heart failure and cardiogenic pulmonary edema could not be ruled out given elevated NT-proBNP levels at 2373 pg/mL (upper normal limit of 35.4 pg/mL) and D-dimer at 3.24 mg/L hence loop diuretics were given.

Infectious pneumonia and sepsis were initially thought to be the cause of the patient's developing acute respiratory distress syndrome (ARDS) hence piperacillin-tazobactam, amikacin, and vancomycin were administered for empiric coverage of pneumonia. However, this aetiology was

TABLE 1 Serial arterial blood gas measurements.

Date	HD3	HD2	HD2 (post-intubation)	HD1 (pre-intubation)
FiO2	100%	100%	100%	84%
PEEP (cm H2O)	16.0	16.0	8.0	–
pH	7.30	7.31	7.29	7.45
pCO2 (mmHg)	51	42	43	32
pO2 (mmHg)	223	52	100	64
HCO3 (mmol/L)	25.1	21.1	20.7	22.2
O2sat	100%	83%	97%	93%
PFR	223	52	100	76
A-a	428.1	561.1	475.1	506.7

Abbreviations: A-a, alveolar-arterial gradient; FiO2, fraction of inspired oxygen; HCO3, bicarbonate; HD, hospital day; O2 sat, oxygen saturation; pCO2, partial pressure of carbon dioxide; PEEP, positive end expiratory pressure; PFR, PaO2/FiO2 ratio; pO2, partial pressure of oxygen.

eventually ruled out based on the negative culture results and viral panel. Other inciting factors for ARDS such as blood transfusion, aspiration, thoracic trauma, drowning, and toxic inhalation were also not present. Given the antecedent history of vape use, EVALI was then considered. High dose methylprednisolone pulse therapy was offered but next of kin did not consent. On the third hospital day, he expired after developing worsening hypoxemia requiring increasing positive end expiratory pressure (See Table 1). Postmortem autopsy was offered but the family did not consent.

DISCUSSION

As of 2020, the Centers for Disease Control has documented 2807 cases of EVALI in the United States with cases also being reported in other countries, baring the Philippines.[6] In regards to cardiovascular events, a meta-analysis by Ashraf et al in 2023 analysed data from the National Health Interview Surveys and noted an increased risk for acute myocardial injury among e-cigarette users with an odds ratio of 1.44.[1] Non-ischemic cardiomyopathy and two vessel coronary artery disease attributed to vape use has been documented in a case series by Benji et al.[7] Simultaneous occurrence of both EVALI and vape-induced cardiomyopathy has also been reported in some case studies but only the study of Abusharekh et al. has documented concomitant small to medium vessel coronary artery disease and EVALI.[3–5]

Particulates, acrolein and oxidizing chemicals in the inhaled aerosols have been proposed to cause atherosclerosis by (1) inducing airway irritation leading to vagal nerve activation, consequent flow mediated dilation impairment and endothelial dysfunction, and (2) triggering systemic inflammation leading to platelet activation, thrombosis and also endothelial dysfunction.[2,8] Nicotine in vape liquid also cases sympathetic nervous system activation leading to increased heart rate, blood pressure and myocardial demand.[2] Pathophysiologic mechanisms for EVALI involve direct damage to the respiratory tract from heated aerosol exposure as well as indirect injury by inhibiting alveolar type II cells, decreasing surfactant catabolism, and altering lipid homeostasis.[6]

The presence of simultaneous acute myocardial infarction and respiratory failure in our patient with no underlying risk factors or comorbid diseases suggests that his e-cigarette use caused both chronic total obstruction of two large vessel coronary arteries and direct lung injury, though acute myocardial infarction related ARDS and cardiogenic pulmonary edema cannot be completely excluded without pulmonary capillary wedge pressure or left atrial pressure measurement.

Current recommendations for the management of EVALI involve discontinuation of e-cigarette use and administration of empiric antibiotics, as well as antiviral therapy in some cases, while awaiting infectious work up as the initial presentation of patients may be difficult to differentiate from the more common pneumonia. The use of glucocorticoids is common practice in patients diagnosed with EVALI but is actually only recommended for patients who develop worsening symptoms or clinical deterioration.[3] Aside from the current standards of care in management of acute myocardial infarction and the discontinuation of e-cigarette use, no additional recommendations have been made regarding the treatment of vape-use associated myocardial injury.[1,6,7]

This case report is the first in the Philippines that links vape use to the development of both acute lung injury and myocardial infarction. The limitation of this study includes the lack of vape liquid analysis for further biochemical study and its qualitative nature.

In conclusion, clinicians should maintain a high index of suspicion for EVALI among patients with a history of vape use presenting with respiratory symptoms and initiate prompt workup to rule out other causes of respiratory injury. EVALI and e-cigarette associated cardiovascular disease may occur simultaneously, even in young previously healthy non-cigarette smokers, and the failure of one organ system may precipitate the further deterioration in the other.

AUTHOR CONTRIBUTIONS

Margarita Isabel C. Fernandez: Writing of article from initial draft to final revisions. **Margaret Francine Co**: Proof reading and editing from draft to revisions. **Janine Bianca Marie Rafael**: Writing of article from initial draft to final revisions, proof reading and editing from draft to revisions. **Regiel Christian Mag-usara**: Writing of article from initial draft to final revisions, proof reading and editing from draft to revisions. **Vanessa Ediza**: Proof reading and editing from draft to revisions. **Rafael Luis Gavino**: Proof reading and editing from draft to revisions, especially on antibiotic course and work up. **Jan Christian Feliciano**: Proof reading and editing from draft to revisions, acquisition and analysis representative chest computed tomography results. **Nigel Jeronimo Santos**: Proof reading and editing from draft to revisions. **Mark Andrian Yano**: Proof reading and editing from draft to revisions. **Julian Alexander Huibonhoa**: Acquisition and analysis of cardiovascular data including echocardiography and coronary angiography findings, proof reading and editing from draft to revisions. **Richard Henry Tiongco**: Acquisition and

analysis of cardiovascular data including echocardiography and coronary angiography findings, proof reading and editing from draft to revisions. **Lenora Fernandez**: Writing of article from initial draft to final revisions, acquisition and analysis of pulmonary related data.

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CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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