Cardiogenic Rhinorrhea: Evidence for an Unrecognized Heart Failure Symptom

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ABSTRACT

There have been anecdotal observations of rhinorrhea as an isolated symptom indicating volume overload and impending congestive heart failure (CHF). We present a case of apparent cardiogenic rhinorrhea presaging acute systolic CHF, with hemodynamics supported by thoracic impedance data (Medtronic OptiVol 2.0).

KEYWORDS: cardiogenic rhinorrhea, acute heart failure, intrathoracic impedance, hypervolemia

BACKGROUND

With large numbers of hospitalizations due to congestive heart failure (CHF), early identification and interventions become increasingly necessary. Understanding and recognition of the signs and symptoms portending to volume overload can allow a physician to provide care early and potentially prevent hospitalization. Rhinorrhea in the setting of CHF exacerbation has yet to be considered a clinically relevant symptom, despite anecdotal observations that have been made over the years.[1,2] In this case, we observed a direct association between a patient experiencing new onset rhinorrhea in concordance with changes in thoracic impedance and volume overload. This was followed by a CHF exacerbation episode and hospitalization, thus suggesting idiopathic rhinorrhea as a sign of volume overload.

CASE REPORT

A 77-year-old woman presented to the emergency department with acute onset of increased dyspnea and was noted to be in congestive heart failure. This patient has a history of systolic heart failure, with non-ischemic cardiomyopathy and ejection fraction of 20% as measured by echocardiogram. She had undergone biventricular intracardiac device implantation (Medtronic Claria MRI Quad CRT-D) and had mild obstructive coronary artery disease (50% stenosis of LAD by 2013 heart catheterization), stage III CKD, hypertension, depression, and GERD. Preceding her presentation, she had felt no other symptoms including orthopnea, PND, edema, weight change, dyspnea on bending or changes in diet. The only prodromal symptom that was noted was anterior as well as posterior rhinorrhea for the past 2–3 days. On exam, an audible S4 was heard and JVP was elevated, without any signs of peripheral edema. Lungs demonstrated bibasilar crackles. At presentation, she was afebrile, tachycardic without chest pain and normotensive. Her oxygen saturation on room air was 84% that increased to 100% upon administration of 2 liters of oxygen via nasal cannula. Relevant labs showed no electrolyte abnormalities, stable elevated creatinine, and BNP elevated to 505 pg/mL. There were no signs or symptoms of an upper respiratory tract infection, sinusitis or allergic rhinitis. Procalcitonin was negative and a nasal swab PCR was negative for COVID-19. She had no prior history of allergic or vasomotor rhinitis. IV diuresis was initiated.

Initial chest X-ray (**Figure 1**) taken at presentation to the ED showed cardiomegaly that was more prominent when compared to a previous study. A left-sided triple lead implantable cardiac device was noted with leads terminating in appropriate positions. Broncho-vascular markings were blurred suggesting pulmonary edema with small bilateral pleural effusions. During the patient's hospitalization, her intrathoracic impedance and volume status changes were assessed through interrogation of OptiVol 2.0 parameters on

Figure 1. Chest X-ray taken upon initial patient presentation. Biventricular Pacemaker-Defibrillator device (Medtronic Claria MRI Quad CRT-D) is visible and intrathoracic impedance monitor.

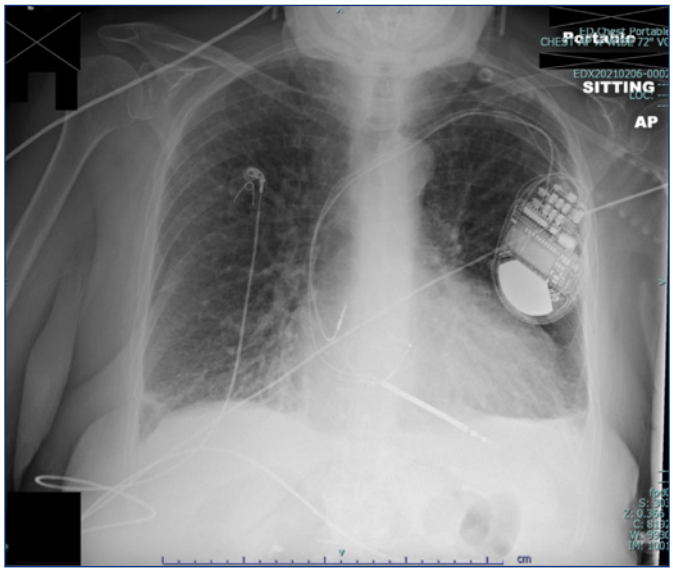
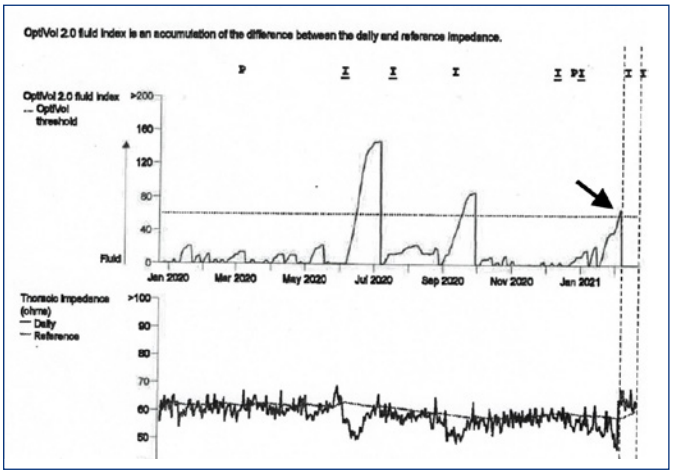


Figure 2. OptiVol 2.0 hemodynamics: three separate episodes of clinical CHF are represented on the Optivol data, two of which were prior to the presenting episode, represented by the arrow.



her Biventricular Pacemaker-Defibrillator device (Medtronic Claria MRI Quad CRT-D). Four days prior to admission, the patient's OptiVol 2.0 Fluid Index was 0. The next day it raised to 30 and on the day of admission peaked at 70, which is above the threshold indicative of volume overload (**Figure 2**).

The patient was given IV furosemide for diuresis and was able to wean off of supplemental oxygen. The patient was discharged after two days with her medication reconciled and a planned follow-up. Two days following discharge, the patient's rhinorrhea had completely resolved and her ICD measured a fluid index return to baseline of 0.

At 2-month follow-up, the patient remains free of rhinorrhea and free of all decompensated CHF symptoms, with stable Class 1 CHF. Optivol 2.0 thoracic impedance parameters remain within normal limits.

DISCUSSION

It is estimated that more than 1 million hospital admissions in America are directly related to congestive heart failure.[3] Patient education of the earliest signs and symptoms of impending exacerbation of heart failure is an important tool employed by practicing physicians in the prevention of hospital admission and readmission. Early identification and intervention is increasingly necessary, as hospital admissions account for the majority of the direct cost burden for patients with heart failure.[4] Traditional manifestations of volume overload such as weight gain, peripheral edema, orthopnea, paroxysmal nocturnal dyspnea, and abdominal bloating remain essential for its early recognition. Recent identification of a novel symptom, shortness of breath bending (termed bendopnea[5] or flexo-dyspnea),[6] has also been shown to correlate well with changes in patient hemodynamics and impending CHF exacerbation. In this case report, we present a patient whose impending exacerbation was preceded by a newly characterized clinical sign, which we propose terming "cardiogenic rhinorrhea."

Anecdotes exist positing the relationship between the onset of anterior rhinorrhea ("runny nose") and/or posterior rhinorrhea ("post nasal drip") as a preceding symptom to heart failure exacerbation in some patients, which can completely resolve with IV diuresis and appropriate CHF treatment.[1,2] This manuscript is the first, to our knowledge, to provide hemodynamic evidence suggesting the associated onset of rhinorrhea as a clinical correlate for volume overload and impending exacerbation of CHF.

We can only speculate on a possible mechanism for this association. Peripheral and generalized edema in CHF is caused by disordered intravascular volume control, in which there is disproportionate activation of vasoconstrictor-sodium retaining systems, along with failure of vasodilatory natriuretic factors, resulting in excessive salt and water balance.[7] This leads to an increase in capillary pressure,[8] causing movement of fluid from the intravascular space to the interstitium, with the net result of expanding extracellular volume as clinical edema.[9] It is feasible that a similar process causes CHF to sometimes manifest as rhinorrhea, in which an expansion of fluid volume in subepithelial capillary beds of the nasopharynx leads to fluid dripping from the nose.[10] Autonomic dysfunction also plays a role in the pathophysiology of CHF[7] as well as that of nonallergic rhinitis and rhinitis of the elderly.[11]

The OptiVol device is able to indirectly measure increases in lung fluid congestion by sensing a decrease in intrathoracic impedance. Measurements are taken throughout the day and referenced to the baseline measured upon initial placement of the device. OptiVol 2.0 has been shown in multiple trials to be a highly sensitive measure for elevated LV volume and filling pressures.[12] Increases in OptiVol 2.0 index are correlated with increases in Pulmonary Capillary Wedge Pressure (PCWP), fluid balance, and NT-pro BNP.[13,14] Measurements of reduced impedance (increased OptiVol 2.0 fluid index) typically precede symptom onset by multiple days.[15] There are two earlier episodes of clinical CHF represented on the Optivol data, during which Optivol index rose to an even higher level than it did during the present case, (155 and 95) (**Figure 2**). Clinically, these episodes involved more florid symptoms of CHF, during which the patient experienced rhinorrhea in addition to more traditional CHF symptoms, including orthopnea and weight gain. The episode of CHF documented in this case report was subjectively milder, and the only prodrome symptom was rhinorrhea.

Rhinorrhea is an extremely common and bothersome medical condition. The prevalence of non-allergic rhinitis in the United States is estimated at 19 million.[11] The cause of most cases of non-allergic rhinorrhea is unknown.[16] The prevalence of CHF is expected to increase by 25% over the next 10 years, with a particularly large increase in the

incidence of heart failure with preserved left ventricular function (HFpEF).[17,18] Asymptomatic diastolic dysfunction, a precursor to HFpEF, is characterized by elevated left ventricular filling pressures, and is highly predictive of the development of CHF.[17] We hypothesize that many cases of "asymptomatic" diastolic dysfunction are not truly devoid of symptoms. It is feasible that there is crossover between these two common conditions, and that the recognition and further investigation of cardiogenic rhinorrhea may have a significant impact on the recognition, prevention, and treatment of CHF.

CONCLUSIONS

Further clinical studies utilizing chronic hemodynamic monitoring such as Cardiomems or others will be useful to further characterize the role of traditional as well as novel symptoms of decompensated CHF, enhancing the early recognition of CHF and further reducing unnecessary hospitalizations.

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The South County Hospital Institutional Review Board (IRB) has reviewed our Case Report, and determined that it is exempt from a full IRB review according to 45 CFR 46.104. The IRB has affirmed its ethical oversight of our work.

Conflict of Interest

There are no conflicts of interest for the authors to declare.

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