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Incidence of Respiratory Pathogens in Naval Special Warfare Sea, Air, and Land Team Candidates With Swimming-Induced Pulmonary Edema

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BACKGROUND: Swimming-induced pulmonary edema (SIPE) is a respiratory condition frequently seen among Naval Special Warfare (NSW) trainees. The incidence of positive respiratory panel (RP) findings in trainees with a diagnosis of SIPE currently is unknown.

RESEARCH QUESTION: Does a significant difference exist in the incidence of respiratory pathogens in nasopharyngeal samples of NSW candidates with SIPE and a control group?

STUDY DESIGN AND METHODS: Retrospective analysis of clinical information from NSW Sea, Air, and Land (SEAL) team candidates with a diagnosis of SIPE over a 12-month period. Candidates who demonstrated the common signs and symptoms of SIPE underwent a nasopharyngeal swab and RP test for common respiratory pathogens. SIPE diagnoses were supported by two-view chest radiography. RP tests were obtained for a selected control group of first-phase trainees without SIPE.

RESULTS: Forty-five of 1,048 SEAL team candidates received a diagnosis of SIPE (4.3%). Five had superimposed pneumonia. Thirty-six of 45 showed positive results for at least one microorganism on the RP (80%). In the study group, human rhinovirus/enterovirus (RV/EV) was the most frequently detected organism (37.8%), followed by coronavirus OC43 (17.8%), and parainfluenza virus type 3 (17.8%). Sixteen of 68 candidates from the control group showed positive RP (24%) findings. Patients with SIPE and positive RP results reported dyspnea (94%), pink frothy sputum (44%), and hemoptysis (36%) more frequently than the control participants with positive RP results. Those who reported respiratory infection symptoms in both the study and control groups showed higher incidences of positive RP results ($P = .046$).

INTERPRETATION: We observed that 80% of trainees with a diagnosis of SIPE showed positive results on a point-of-care RP. This positivity rate was significantly higher than that of RP test results from the control cohort. These findings suggest an association between colonization with a respiratory pathogen and the development of SIPE in NSW candidates.

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KEY WORDS: chest radiograph; pulmonary edema; respiratory panel; upper respiratory infection

FOR EDITORIAL COMMENT, SEE PAGE 1009

ABBREVIATIONS: AHF = acute heart failure; BUD/S = Basic Underwater Demolition/Sea, Air, and Land; NSW = Naval Special Warfare; RP = respiratory panel; RV/EV = rhinovirus and/or enterovirus; SIPE = swimming-induced pulmonary edema; URI = upper respiratory infection

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Take-home Points

Study Question: Do significant differences exist in the incidence of respiratory pathogens in nasopharyngeal samples of Naval Special Warfare (NSW) candidates with swimming-induced pulmonary edema (SIPE) and a control group?

Results: Thirty-six of 45 NSW candidates with SIPE showed positive results for at least one microorganism on the respiratory panel (80%), which is significantly higher than respiratory panel screening results from the control group (24%).

Interpretation: These findings suggest an association between colonization with a respiratory pathogen and the development of SIPE in NSW candidates.

Thousands of candidates arrive each year in Coronado, California, at the Naval Special Warfare (NSW) Basic Training Command to participate in United States Navy Sea, Air, and Land training. Throughout this training pathway, candidates are exposed to extremes of environment and are pushed to the limits of what the human body can endure. As such, they often experience a variety of conditions and ailments that are uncommon even among other military trainees. Swimming-induced pulmonary edema (SIPE) is one such condition and typically is limited to military trainees and participants of athletic events involving heavy exertion in water. SIPE is a respiratory condition frequently seen among candidates participating in NSW Basic Underwater Demolition/Sea, Air, and Land (BUD/S) training. In a recent study of NSW trainees, the incidence of SIPE was observed to be 5.00%.[1]

Historically, SIPE has been classified as a type of cardiogenic pulmonary edema. The pathophysiologic features of SIPE involve shunting of blood volume from the peripheral circulation to the central venous system during heavy exertion while immersed in water.[2-4] Engorgement of central veins leads to an increase in cardiac preload.[4,5] The increased preload and cardiac output from strenuous exercise gives rise to elevated pulmonary artery pressures. Increased hydrostatic pressure in the pulmonary vessels results in capillary damage that causes an overload of the capillary-alveolar barrier, leading to interstitial and alveolar edema.[6,7] Furthermore, it has been postulated that in the case of acute pulmonary edema, a stroke volume imbalance occurs between the left and right ventricles that further augments the pressure within the pulmonary circulation.[8]

SIPE typically is characterized clinically by the acute onset of dyspnea and cough in the setting of intense physical exertion while immersed in cold water.[2] Oftentimes, this condition is accompanied by hypoxia and production of pink frothy sputum or frank hemoptysis. On chest radiography, SIPE is typified by airspace opacification with a predominantly interstitial pattern, increased septal and interstitial lines, perifissural thickening, enlarged cardiac silhouette, and widened azygos vein diameter.[1] Occasionally, those who experience SIPE require a short course of supplemental oxygen therapy to treat hypoxemia or urgent medical care. Reports have been made in the literature even of near drowning attributed to SIPE.[9] However, SIPE generally is considered a self-limiting condition, and the vast majority of those who experience it have near resolution of symptoms within 48 h of initial onset.

Respiratory infections also are seen frequently in NSW trainees. The incidence of pneumonia in one study of 2,117 NSW candidates was determined to be 2.08%.[1] Anecdotally, acute upper respiratory infections (URIs) also are commonplace in this population. No studies to date have examined the incidence of acute URIs among BUD/S candidates. However, prior research has shown that military personnel are more prone to respiratory infections than their civilian counterparts.[10] Furthermore, the intense nature of this training, which oftentimes involves prolonged periods of sleep deprivation, strenuous exercise, and psychological stress, has been associated with a transient depression of numerous aspects of immune function, leading to greater susceptibility to respiratory infections.[11,12]

Both SIPE and acute respiratory infections have a significant impact on NSW training. Together, these conditions represent a substantial disease burden among BUD/S candidates and within the NSW training pathway. With the emergence of the COVID-19 pandemic, obtaining respiratory panels (RPs) became a routine step in evaluating all patients with respiratory symptoms, including SIPE. In the present study, the primary objective was to examine and compare the incidence of positive RP results among all NSW candidates with a diagnosis of SIPE and a separate first-phase control group.

Study Design and Methods

For this case-control study, we reviewed data previously collected from NSW Sea, Air, and Land candidates with a diagnosis of SIPE from December 2020 through November 2021. The data included information on clinical features documented on presentation, two-view chest radiography findings, and RP results. RP tests and clinical findings for a separate first-phase cohort without a diagnosis of SIPE were examined and compared with those of the study group. This study was approved by the Naval Health Research Center Institutional Review Board in compliance with all applicable federal regulations governing the protection of human subjects on March 25, 2022 (Identifier, NHRC.2022.0002).

Electronic medical records documenting clinical features of NSW trainees with a diagnosis of SIPE were reviewed. Clinical features examined include SIPE-characteristic symptoms (eg, cough, hemoptysis, pink frothy sputum, or shortness of breath) and physical examination findings (vital signs and lung auscultation). The incidence of each presenting symptom, among all candidates with a diagnosis of SIPE, was calculated. Additionally, we determined the number of individuals reporting URI symptoms (eg, sinus congestion, sinus pressure, rhinorrhea, yellow or green sputum production, or sore throat) before the onset and diagnosis of SIPE. For comparison purposes, the incidence of reported URI symptoms was determined separately for those with positive and negative results on the RP in both the control and study groups. A χ^2 test was used to determine whether any correlation existed between reported symptoms and RP results.

Initial and follow-up (when indicated) two-view chest radiographs were analyzed retrospectively by a board-certified thoracic radiologist (G. E. B.) at the Naval Medical Center San Diego. The initial and follow-up chest imaging were used to identify the presence and subsequent resolution of pulmonary edema. The appearance of increased septal and interstitial lines, ground-glass opacification, perifissural thickening, enlarged heart diameter, and widened azygos vein diameter on plain films were used to verify the presence of pulmonary edema and to differentiate it from pneumonia (including patients in whom it was present concurrently with SIPE).

The diagnosis of SIPE was rendered when (1) patients reported the acute onset of dyspnea, frank hemoptysis, or production of pink frothy sputum during or shortly after intense exertion while immersed in water, (2) pulmonary crackles were auscultated on examination or the patient was hypoxemic (oxygen saturation $<$ 95%) on initial presentation, (3) initial chest radiography demonstrated evidence of pulmonary edema, and (4) improvement or resolution of symptoms and clinical findings within 48 h of initial presentation occurred. Based on the above criteria, the number of SIPE cases was determined and the incidence of SIPE over the studied time frame was calculated.

Each trainee who showed signs and symptoms of SIPE underwent a nasopharyngeal swab and was tested for common respiratory infectious agents via the BioFire polymerase chain reaction instrument. All biological specimens were collected, handled, stored, and processed according to instructions outlined by BioFire Defense, LLC. The incidence of positive RP results was calculated for those with a diagnosis of SIPE. Each of the positive results then were subdivided further by the identity of the microorganism(s) for which they showed positive results. The incidence of each of the individual microorganisms, among candidates with positive results, then was calculated.

On May 2 and 3, 2022, surveillance RP testing was conducted for the entire first-phase BUD/S cohort, which consisted of 68 candidates. No candidates carried a SIPE diagnosis or had an ongoing diagnostic workup to evaluate for SIPE. This group did not participate in swimming events before sampling. As part of this testing, each participant underwent a nasopharyngeal swab. Each collected specimen was processed and analyzed in the Luminex NxTAG Respiratory Pathogen Panel unit. We retrospectively examined results of these assays and determined the incidence of positive RP findings for the class. A χ^2 test was used to determine whether a significant difference existed between the proportion of positive RP results in this cohort and trainees with a diagnosis of SIPE. A P value of $<$ .05 was considered significant. In the case of statistically significant results, the probability value (P value) was given.

Results

The primary study population comprised 1,048 special operations candidates with a mean age of 24 years. All participants were without significant known medical conditions and met the stringent health standards outlined by the United States Navy Bureau of Medicine and Surgery for both Special Operations and Diving Duty. All study participants were enrollees in the first phase of BUD/S training for various intervals between December 11, 2020, and November 26, 2021. The study population was dynamic and showed significant attrition related to the nature of the training and selection process. In total, six BUD/S classes took place over the study period.

Of the 1,048 study participants, 45 individuals had a diagnosis of SIPE (4.3%). Five of the trainees with a diagnosis of SIPE were found to have superimposed pneumonia. These individuals were treated with courses of antibiotics for community-acquired pneumonia. Ten of the patients with SIPE were experiencing recurrent episodes. In four of these patients, the initial and recurrent SIPE episodes occurred during the study time frame. Sixteen of the cases of SIPE occurred during Hell Week. Most candidates (78%) began experiencing SIPE-characteristic symptoms during the 1- or 2-nautical mile open water swims. Two of the trainees who experienced SIPE were hospitalized for a short time. One of these individuals underwent a short stay in the ICU to receive bilevel positive pressure ventilation. No deaths occurred among the 1,048 study participants over the course of the study. Most NSW trainees (94%) returned to training after recovery from SIPE.

Thirty-six of 45 NSW candidates with a diagnosis of SIPE showed positive results for at least one microorganism on the polymerase chain reaction RP (80.0%) (Table 1). RP positivity rates in patients with

SIPE separated according to meteorologic season (winter, spring, summer, fall) were 87% (n = 15), 75% (n = 8), 69% (n = 13), and 89% (n = 9), respectively. Of the 45 tests conducted, the most frequently detected organisms were human RV/EV (n = 17 [37.8%]), followed by coronavirus OC43 (n = 8 [17.8%]), and parainfluenza virus type 3 (n = 8 [17.8%]). Other pathogens detected included adenovirus (n = 4 [8.9%]), *Chlamydia pneumoniae* (n = 4 [8.9%]), SARS-CoV-2 (n = 3 [6.7%]), coronavirus 229e (n = 1 [2.2%]), and respiratory syncytial virus (n = 1 [2.2%]) (Fig 1).

Of the 36 RPs with positive results, nine showed positive results for more than one pathogen (25%). One of these assays detected more than two pathogens (11.1%). Human RV/EV was detected in combination with other microorganisms in four samples, with the other pathogens being *Chlamydia pneumoniae*, coronavirus OC43, and adenovirus. Parainfluenza virus type 3 was found in three separate assays in combination with other pathogens, namely, coronavirus OC43 and adenovirus.

Of the 36 candidates with positive RP results, 25 candidates (69.4%) reported URI symptoms (fever, chills, sore throat, sinus congestion, rhinorrhea, or purulent sputum production) before the characteristic symptoms of SIPE developed (acute onset of dyspnea, hemoptysis, production of pink frothy sputum). Three of nine trainees with negative results reported URI symptoms before the characteristic symptoms of SIPE developed (33%). Those with URI symptoms in both the study ($P = .046$) and control ($P < .00001$) groups showed higher incidences of positive RP findings (Table 1).

On presentation, the most commonly reported symptoms among the study group included cough (100%), dyspnea (96%), pink frothy sputum (42%), frank hemoptysis (33%), and chest pain (27%). No candidates from the control group reported hemoptysis or production of pink frothy sputum. Individuals with SIPE reported dyspnea (96%) more frequently than those in the control cohort (1.5%) ($P < .00001$). No candidates reported an aspiration event before presentation. Among trainees with SIPE, crackles were the most common pulmonary examination finding (42%), followed by rhonchi (38%) and wheezing (20%). Twenty percent of pulmonary examination results were normal. The median peripheral oxygen saturation for patients with SIPE with positive and negative RP results was 90% and 92%, respectively (Table 2). In the control group, cough was endorsed more often by patients with positive RP findings ($P < .008$) (Table 2).

The control arm for this study comprised 68 men with a mean age of 23 years. All were enrollees in the first phase of BUD/S training and met the health standards requisite for special warfare training. Sixteen of 68 candidates from this cohort showed positive results for a microorganism on the RP (24%). Of the 16 assays with positive findings, 14 assays were identified as showing human RV/EV (88%). One candidate showed positive results for both human RV/EV and human coronavirus HKU1 (6%). One candidate showed positive results for adenovirus (6%). Of the 16 patients with positive results, 10 trainees reported respiratory symptoms on the day the nasopharyngeal specimen was collected (63%) (Table 1). Individuals with SIPE showed higher positivity rates on RPs than the control group ($P < .00001$) (Table 3).

Discussion

Various risk factors for SIPE have been proposed previously in literature. Overhydration, wetsuit use, female sex, prone horizontal position during swimming,

TABLE 1] Comparison of Reported Upper Respiratory Infection Symptoms in Patients With SIPE and Control Group Participants With Positive and Negative RP Pathogen Results

Variable	Patients With SIPE (n = 45)		Control Group (n = 68)	
	Positive PCR RP Results (n = 36)	Negative PCR RP Results (n = 9)	Positive PCR RP Results (n = 16)	Negative PCR RP Results (n = 52)
Preceding respiratory infection symptoms	25 (69)	3 (33)	10 (63)	5 (10)
No preceding respiratory infection symptoms	11 (31)	6 (67)	6 (37)	47 (90)
P value	.046[a]		< .00001[a]	

Data are presented as No. (%), unless otherwise indicated. PCR = polymerase chain reaction; RP = respiratory panel; SIPE = swimming-induced pulmonary edema.
[a]Statistically significant.

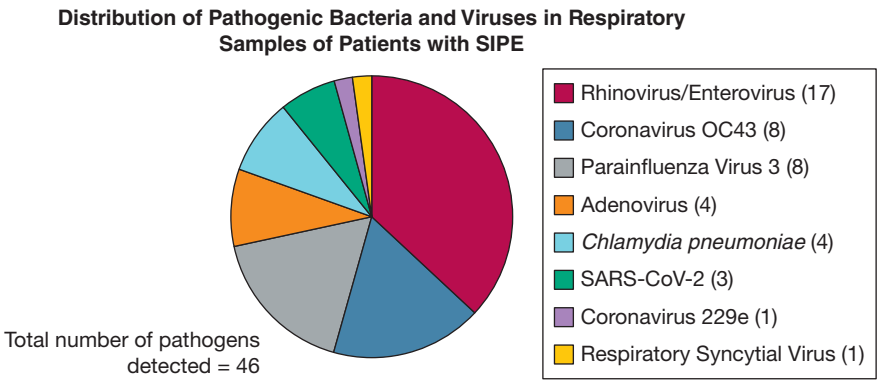

Figure 1 – *Pie chart displaying the proportion of respiratory microorganisms identified by polymerase chain reaction in patients with swimming-induced pulmonary edema. Two or more coinfecting organisms were counted in their own categories.*

and pre-existing cardiovascular disease are among those risk factors proposed.[13-17] Each risk factor involves a process that ultimately leads to an increase of the hydrostatic pressure within the pulmonary circulation, resulting in direct insult to the capillary-alveolar barrier and ensuing pulmonary edema. In this article, we report the incidence of positive RP findings and URI symptoms in NSW trainees with a diagnosis of SIPE over a

TABLE 2] Comparison of Clinical Findings Between Patients With SIPE and Control Group with Positive or Negative RP Pathogen Results

Clinical Findings	Patients With SIPE (n = 45)			Control Group (n = 68)		
	Positive PCR RP Results (n = 36)	Negative PCR RP Results (n = 9)	*P* Value[a]	Positive PCR RP Results (n = 16)	Negative PCR RP Results (n = 52)	*P* Value[b]
URI symptoms[c]						
Subjective fever/chills	6 (17)	0 (0)	NA	0 (0)	0 (0)	NA
Sinus congestion	18 (50)	2 (22)	.260	2 (13)	3 (6)	.367
Sore throat	5 (14)	1 (11)	1.00	1 (6)	0 (0)	NA
Runny nose	12 (33)	2 (22)	.456	4 (25)	2 (4)	.060
Yellow or green sputum	10 (28)	1 (11)	.416	5 (31)	0 (0)	NA
Other symptoms						
Frank hemoptysis	13 (36)	2 (22)	.695	0 (0)	0 (0)	NA
Pink frothy sputum	16 (44)	3 (33)	.712	0 (0)	0 (0)	NA
Dyspnea	34 (94)	9 (100)	NA	1 (6%)	0 (0)	NA
Chest pain or tightness	9 (25)	3 (33)	.613	0 (0)	0 (0)	NA
Cough	36 (100)	9 (100)	NA	6 (38)	4 (8)	< .008
Vital signs						
Body temperature, °C	36.5 (36.0-37.0)[d]	36.7 (36.4-37.0)	. . .	. . .	. . .	. . .
SpO_2, %	90 (88-93)	92 (90-96)	. . .	. . .	. . .	. . .
Lung auscultation findings						
Crackles	14 (39)	5 (56)	.365	. . .	. . .	. . .
Rhonchi	13 (36)	4 (44)	.711	. . .	. . .	. . .
Wheezing	8 (22)	1 (11)	.661	. . .	. . .	. . .

Data are presented as No. (%) or median (interquartile range), unless otherwise indicated. NA = not applicable because of small sample size; PCR = polymerase chain reaction; RP = respiratory panel; SIPE = swimming-induced pulmonary edema; SpO_2 = peripheral oxygen saturation; URI = upper respiratory infection.

[a]Comparison of clinical findings for individuals with SIPE with positive and negative RP results; χ^2 test or Fisher exact test for categorical variables.

[b]Comparison of clinical findings for control participants with positive and negative RP results; χ^2 test or Fisher exact test for categorical variables.

[c]Data missing for one trainee.

[d]All upper respiratory infection symptoms were present before and at the time of SIPE diagnosis for study group.

12-month period. We compared these RP results and clinical features with those of a control group of first-phase candidates of similar demographic makeup, health status, and environmental exposures. The findings of this study suggest an association between the presence of infectious agents in the respiratory tract with the incidence of SIPE in NSW candidates. We propose that these findings may represent a predisposing risk factor for SIPE not previously described in literature.

In this study, 80% of BUD/S candidates with a diagnosis of SIPE showed positive results for at least one microorganism on the RP. This positivity rate was significantly higher than RP screening results from a recent first-phase class (24%) that served as the control arm for this study ($P < .00001$) (Table 3). With the exception of rhinovirus, these microorganisms are almost always considered pathogens if detected anywhere in the respiratory tract.[18] Most candidates with positive respiratory results reported URI symptoms before the SIPE diagnosis. Several possible explanations exist for this association between URIs, positive RP results, and SIPE. It may be the result of a so-called two-hit model in which a trainee experiencing symptoms of a URI goes on to demonstrate SIPE, which results in a compounding of symptoms, making the candidate more likely to seek medical care. This explanation would apply only to a portion of candidates because a number of those with positive findings for an infectious agent did not report any symptoms before SIPE developed. Furthermore, among those with positive RP findings in the control group, few endorsed SIPE-specific symptoms (eg, dyspnea, pink frothy sputum, hemoptysis) (Table 2). Alternatively, these findings may suggest that the presence of these agents may be, either by direct cytopathic effect or indirect inflammatory changes, lowering the point at which SIPE pathophysiologic features reach a symptomatic threshold.

TABLE 3] Comparison of Respiratory Pathogen Panel Results Between Patients With SIPE and Control Group

Variable	SIPE Diagnosis (n = 45)	First-Phase Control Participants (n = 68)
Positive PCR RP results	36 (80)	16 (24)
Negative PCR RP results	9 (20)	52 (76)
P value[a]	< .00001[b]	

Data are presented as No. (%), unless otherwise indicated. PCR = polymerase chain reaction; RP = respiratory panel; SIPE = swimming-induced pulmonary edema.
[a]Comparison of respiratory pathogen panel results between patients with SIPE and the control group; χ^2 test used for these categorical variables.
[b]Statistically significant.

Many of the organisms on the traditional RP are considered primarily to be culprits in URIs. However, experimental studies have demonstrated that many of these pathogens also have been detected in the lower respiratory tract and in many cases have been shown to impart deleterious effects therein. For example, rhinovirus is one such agent that has been detected in lower airway cells and has been implicated in exacerbations of both COPD and asthma.[19,20] In one study, human coronaviruses were present in sputum samples of a substantial number of pediatric patients with lower respiratory tract infections (eg, pneumonia, bronchiolitis, bronchitis).[21] Adenovirus and rhinovirus have been shown to initiate a sequence of inflammatory events in the lungs via secretion of several proinflammatory cytokines and chemokines.[22,23] One of these cytokines, tumor necrosis factor α, is of particular interest because it has been shown in mice models to impair function of the paracellular alveolar permeability barrier and in other studies to promote pulmonary edema.[24,25] Taken together, these findings at the very least highlight the role these organisms can and do play in the pathologic characteristics of lower respiratory tract infections. In the context of this study, they represent a potential physiologic link between colonization with infectious respiratory organisms and the hemodynamic mechanisms that drive SIPE.

Respiratory infections also have been implicated as a common precipitant of acute heart failure (AHF).[26,27] A single-center retrospective study examining causal factors among 435 patients hospitalized for AHF found that preceding respiratory infection was a precipitant in 16% of hospital admissions for AHF.[28] In another article, it was proposed that inflammatory and oxidative lung injury play a significant pathophysiologic role in heart failure decompensation by further damaging the alveolar-capillary barrier and increasing its permeability. As a result, the pulmonary capillary hydrostatic pressure threshold for pulmonary fluid accumulation decreases.[29] Collectively, these findings represent a plausible pathophysiologic connection among respiratory infection, lung injury and inflammation, and AHF.

Interestingly, a high incidence of preexisting respiratory infections also has been observed in pediatric cases of high-altitude pulmonary edema.[30-32] Prior research on young rats examining this trend has shown that

preexisting viral respiratory infection may augment their susceptibility to increases in lung water and in pulmonary vascular permeability associated with exposure to hypoxia.[33] Although inflammation secondary to infection is not considered to be the primary cause of high-altitude pulmonary edema, it has been suggested that this inflammation may make the microvascular endothelium more vulnerable to increased pressures. If this is indeed the case with both high-altitude pulmonary edema and AHF, then perhaps this holds true in the case of SIPE, which also is classified primarily as a type of hydrostatic pulmonary edema.

We postulate that preexisting respiratory infection, colonization, or both with a pathogenic microorganism are predisposing risk factors for SIPE developing. This may be in part the result of the infectious agents inducing changes in permeability of the alveolar-capillary barrier, which then lowers the threshold at which hydrostatic pressures give rise to pulmonary edema. However, further studies should be conducted to explore this hypothesis in more depth. From a clinical and operational perspective, these findings are of importance. If detection of a respiratory pathogen significantly increases the chances of SIPE, trends in respiratory microorganism colonization can be used to mitigate medical risk to the individual or a unit.

At the time this study was conducted, Navy policy required all individuals reporting respiratory symptoms to be removed from training until symptom resolution. Hence, it is likely that the presence of these symptoms among candidates with SIPE were underreported. Moreover, the presence of respiratory symptoms related to SIPE may have colored candidates' recollection of their URI symptoms. These represent potential limitations of the clinical symptom dataset presented in this article.

Another limitation of the present study is that specimens showing positive results in patients with SIPE were collected over 1 year, whereas control group specimens were collected on 2 consecutive days, omitting potential seasonal variation in respiratory organism frequency. Although seasonal variation in RP positivity rates occurred among the study group (69%-89%), rates were considerably higher than in the control group (24%).

Additionally, different instruments were used for analysis of respiratory pathogens in individuals with SIPE and the control group. The NxTAG Respiratory Pathogen Panel unit has a higher sample throughput than the BioFire instrument. The NxTAG Respiratory Pathogen Panel unit is able to process up to 96 samples at one time vs the BioFire, which can process one sample per run. The NxTAG thus was used for the control group screening tests to allow for more expeditious results. In a study conducted by Chen et al[34] in 2016 comparing these two instruments, complete concordance was observed in 98.9% of positive results.

Interpretation

This study showed that a large proportion of NSW trainees with a diagnosis of SIPE experience antecedent respiratory illnesses and frequently show positive results on point-of-care RPs. Candidates with SIPE showed significantly higher rates of positivity on the RPs when compared with a standard first-phase cohort without SIPE. By highlighting these findings, we have demonstrated an association between the presence of a preexisting respiratory illness and respiratory pathogen colonization, with the development of SIPE in NSW candidates.

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Author contributions: B. A. S. takes responsibility for the content of the manuscript, including the integrity of the data and data analysis. B. A. S. conducted the primary literature search. B. A. S., P. W., P. L., and C. G. V. contributed to the study design and methodology. B. A. S. and P. W. took an active part in data collection. B. A. S., P. W., P. L., and G. E. B. assisted with data interpretation and analysis. All authors participated in content development and wrote and edited the manuscript. All authors reviewed and approved the final version of the manuscript.

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