Legal Medicine 85 (2026) 102935

Contents lists available at ScienceDirect

Legal Medicine

journal homepage: www.elsevier.com/locate/legalmed

Fatal septic shock due to PVL-positive *Staphylococcus aureus* necrotizing pneumonia: autopsy findings and systematic review of the literature☆

Jessika Camatti [a,*], Anna Laura Santunione [b], Rossana Cecchi [b], Erjon Radheshi [c], Edoardo Carretto [d], Maria Paola Bonasoni [e]

[a] *University of Parma, Parma, Italy*
[b] *University of Modena and Reggio Emilia, Modena, Italy*
[c] *Local Health Authority of Reggio Emilia, Reggio Emilia, Italy*
[d] *Azienda Socio Sanitaria Territoriale "Papa Giovanni XXIII", Bergamo, Italy*
[e] *Azienda Unità Sanitaria Locale, IRCCS, Reggio Emilia, Italy*

ARTICLE INFO

Keywords:
Staphylococcus aureus
Panton-valentine leukocidin
Necrotizing pneumonia
Septic shock;
Forensic medicine
Postmortem microbiology

ABSTRACT

Panton–Valentine leukocidin (PVL)–producing *Staphylococcus aureus* is associated with highly aggressive necrotizing pneumonia characterized by rapid clinical deterioration and high mortality. However, the clinicopathological features of fatal cases, particularly in the forensic setting, remain incompletely defined.

We report a fatal case of PVL-positive *S. aureus* necrotizing pneumonia diagnosed through forensic autopsy and molecular analysis. In addition, a systematic review was conducted according to PRISMA 2020 guidelines. PubMed/MEDLINE and Scopus were searched from database inception to 12 March 2026 to identify published fatal cases of PVL-positive *S. aureus* pneumonia.

A previously healthy 58-year-old man presented with acute respiratory symptoms and rapidly developed severe respiratory failure and septic shock, resulting in death within 24 h of hospital admission. Blood cultures and bronchoalveolar lavage yielded methicillin-susceptible *S. aureus*. Autopsy revealed bilateral necrotizing hemorrhagic bronchopneumonia, and molecular analysis confirmed PVL positivity. The literature review identified 31 fatal cases. Most cases were characterized by rapid clinical deterioration, frequent septic shock, and hemoptysis or pulmonary hemorrhage. When autopsy was performed, a consistent pattern of necrotizing and hemorrhagic pulmonary infection was observed.

PVL-positive *S. aureus* pneumonia represents a rare but highly aggressive infection that may rapidly progress to respiratory failure and death. The combination of rapid clinical deterioration, hemoptysis, and necrotizing hemorrhagic pneumonia at autopsy represents a distinctive clinicopathological pattern. Integration of autopsy findings with microbiological and molecular investigations is essential for establishing the diagnosis in rapidly fatal infectious deaths.

1. Introduction

Staphylococcus aureus is a well-recognized cause of both community-acquired and hospital-acquired pneumonia, accounting for a relatively small but clinically significant proportion of severe respiratory infections [1]. Compared with other bacterial pathogens, *S. aureus* pneumonia is often associated with a more aggressive clinical course, characterized by extensive pulmonary tissue destruction, bacteremia, and systemic complications [1]. In severe cases, the infection may rapidly progress to sepsis and septic shock, resulting in significant morbidity and mortality. These severe presentations are frequently driven by specific bacterial virulence factors that contribute to the pathogenicity of the organism and the development of fulminant infections [2].

Among these virulence factors, Panton–Valentine leukocidin (PVL) has been strongly implicated in the pathogenesis of necrotizing pneumonia. PVL is a bicomponent pore-forming cytotoxin that targets and destroys leukocytes, leading to intense inflammatory responses and

☆ This article is part of a Special issue entitled: 'Proceedings Congress Forensic Science' published in Legal Medicine.

* Corresponding author.
E-mail addresses: jessika.camatti@unipr.it (J. Camatti), annalaura.santunione@unimore.it (A.L. Santunione), rossana.cecchi@unimore.it (R. Cecchi), erjon.radheshi@ausl.re.it (E. Radheshi), edoardo.carretto@asst-pg23.it (E. Carretto), MariaPaola.Bonasoni@ausl.re.it (M.P. Bonasoni).

https://doi.org/10.1016/j.legalmed.2026.102935
Received 14 May 2026; Received in revised form 4 July 2026; Accepted 20 July 2026
Available online 23 July 2026

extensive tissue necrosis [3]. Beyond its direct leukocidal activity, PVL promotes neutrophil activation and lysis, resulting in the release of proteolytic enzymes, reactive oxygen species, and pro-inflammatory mediators that contribute to extensive pulmonary tissue destruction. Furthermore, the exaggerated inflammatory response may induce endothelial injury, increased vascular permeability, and cytokine release, ultimately contributing to systemic inflammatory response syndrome, septic shock, and multiorgan failure.

Strains of *S. aureus* carrying PVL genes have been increasingly reported worldwide and are frequently associated with community-acquired infections, often affecting young and otherwise healthy individuals [4]. In particular, PVL-positive strains have been consistently linked to highly aggressive forms of necrotizing pneumonia with a rapidly progressive clinical course and high mortality rates [5].

PVL-associated pneumonia typically presents as a severe necrotizing infection characterized by rapid clinical deterioration, hemoptysis, leukopenia, and respiratory failure. Despite aggressive medical management, the disease may progress rapidly and has been associated with high mortality rates. In several reported cases, PVL-positive *S. aureus* pneumonia has evolved into severe sepsis and septic shock, sometimes leading to fatal outcomes [6–8]. Because of its fulminant course, this infection may occasionally result in sudden or rapidly fatal death, highlighting the importance of postmortem examination and microbiological investigation in establishing the cause of death.

Despite increasing recognition of PVL-positive *S. aureus* infections, the clinicopathological features of fatal cases—particularly in the forensic setting—remain incompletely characterized. In this study, we report a fatal case of PVL-positive *S. aureus* necrotizing pneumonia diagnosed at autopsy and provide a systematic review of the literature focusing on fatal cases.

2. Case-report

2.1. Case presentation

A 58-year-old previously healthy man presented to the emergency department with fever, productive cough, and hemoptysis that had started the previous day. He reported no chronic medical conditions, regular medication use, or known drug allergies.

On admission, the patient was alert, with an oxygen saturation of 91% on room air, blood pressure of 102/63 mmHg, and heart rate of 78 bpm. Physical examination revealed reduced breath sounds at the right lung base. Chest radiography demonstrated extensive consolidation of the right lung, involving the upper and middle lung fields and extending to the paramediastinal region of the lower lung field, with associated right-sided pleural opacity.

Arterial blood gas analysis performed while breathing room air revealed hypoxemia (PaO_2 63.4 mmHg), consistent with impaired gas exchange. A diagnosis of extensive right-sided pneumonia was made, and the patient was admitted for further management.

However, within hours of admission, the patient experienced rapid clinical deterioration, progressing to septic shock requiring vasopressor support with norepinephrine and subsequently adrenaline. He developed acute respiratory distress syndrome (ARDS), requiring invasive mechanical ventilation with high inspired oxygen fraction and positive end-expiratory pressure. Copious pink frothy secretions were observed in the airways, and bronchoscopic examination revealed abundant serous fluid described as “meat-washing fluid,” consistent with diffuse alveolar damage and pulmonary hemorrhage.

Repeat arterial blood gas analysis demonstrated severe metabolic acidosis and profound hypoxemia (pH 6.876, PaO_2 38.6 mmHg, oxygen saturation 52.3%), with markedly elevated lactate levels (182 mg/dL).

Blood cultures yielded methicillin-susceptible *S. aureus* (MSSA), and the same organism was isolated from bronchoalveolar lavage samples, confirming severe *S. aureus* pneumonia with bacteremia.

Despite aggressive supportive treatment, including broad-spectrum antibiotic therapy with piperacillin–tazobactam and levofloxacin, the patient's condition rapidly deteriorated, and he died within 24 h of hospital admission.

2.2. Autopsy findings

A forensic autopsy was subsequently performed approximately 24 h after death. External examination revealed no significant traumatic findings. Internal examination demonstrated right-sided fibrinopurulent pleuritis. The lungs showed marked pathological changes, including diffuse hepatization of the right lung and marked congestion of the left lung.

Histopathological examination revealed bilateral necrotizing hemorrhagic bronchopneumonia (Figs. 1–4). Extensive intra-alveolar fibrin deposition, hemorrhage, and dense neutrophilic inflammatory infiltrates were observed. No unequivocal hyaline membrane formation suggestive of diffuse alveolar damage was identified. Additional findings included acute tubular epithelial injury compatible with shock-related renal damage (Fig. 5), as well as hepatic and splenic congestion, consistent with systemic septic shock. An incidental papillary thyroid microcarcinoma (2 mm) was also identified. No microabscesses were identified in the myocardium or kidneys.

Given the autopsy findings strongly suggestive of necrotizing staphylococcal pneumonia, molecular testing for PVL was performed. Genotypic testing for the Panton–Valentine leukocidin (PVL) gene was performed on the *S. aureus* isolate obtained from blood culture at the Microbiology and Virology Laboratory of Reggio Emilia. The analysis demonstrated the presence of the PVL gene, establishing the diagnosis of PVL-positive *S. aureus* infection.

3. Methods - literature review

This study was designed as a systematic review aimed at identifying published fatal cases of pneumonia caused by PVL-positive *Staphylococcus aureus*. The review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines [9]. The objective was to investigate the clinical and pathological characteristics of fatal PVL-positive *S. aureus* pneumonia.

Studies were considered eligible if they met the following criteria: (i) original articles reporting fatal cases of pneumonia caused by PVL-positive *S. aureus*; (ii) case reports, case series, or observational studies providing sufficient clinical or pathological data; and (iii) microbiological confirmation of PVL positivity. Exclusion criteria

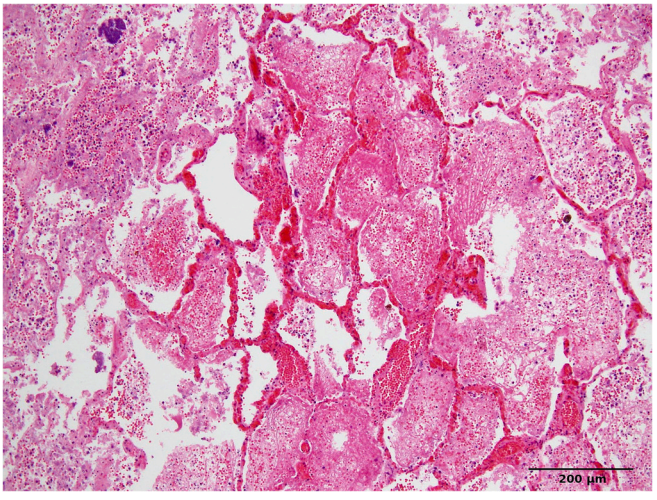

Fig. 1. Necrotizing hemorrhagic bronchopneumonia with extensive intra-alveolar fibrin deposition and hemorrhage (H&E, 10×; scale bar = 200 µm).

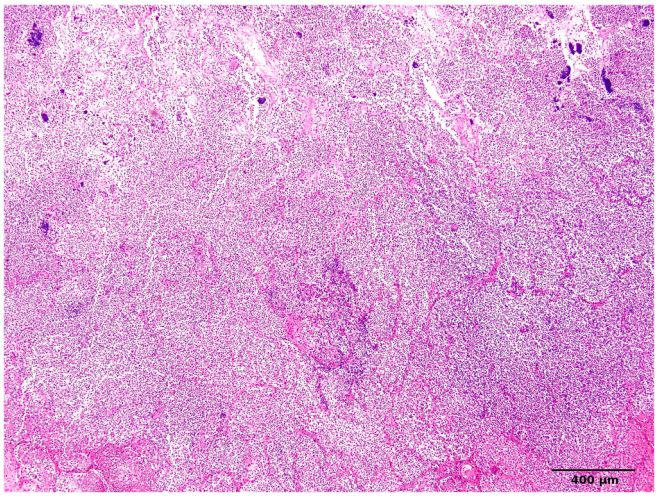

Fig. 2. Diffuse pneumonia characterized by dense neutrophilic infiltrate with basophilic aggregates suggestive of bacterial colonies (H&E, 4×; scale bar = 400 µm).

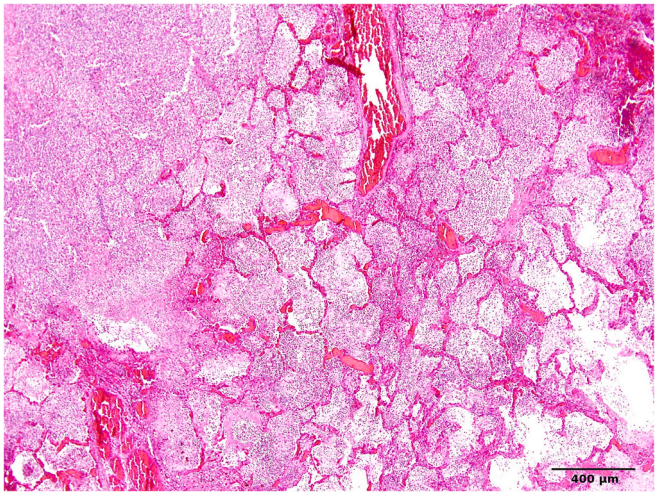

Fig. 3. Diffuse pneumonia with extensive neutrophilic infiltrate filling the alveolar spaces (H&E, 4×; scale bar = 400 µm).

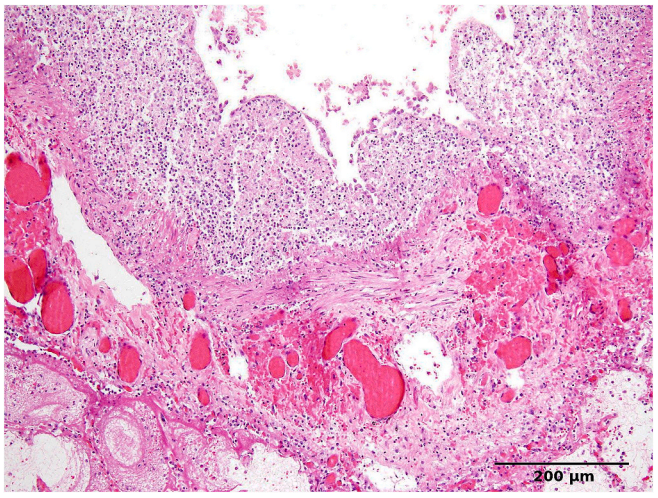

Fig. 4. Necrotizing bronchitis with epithelial destruction, hemorrhage, and prominent neutrophilic inflammatory infiltrate (H&E, 10×; scale bar = 200 µm).

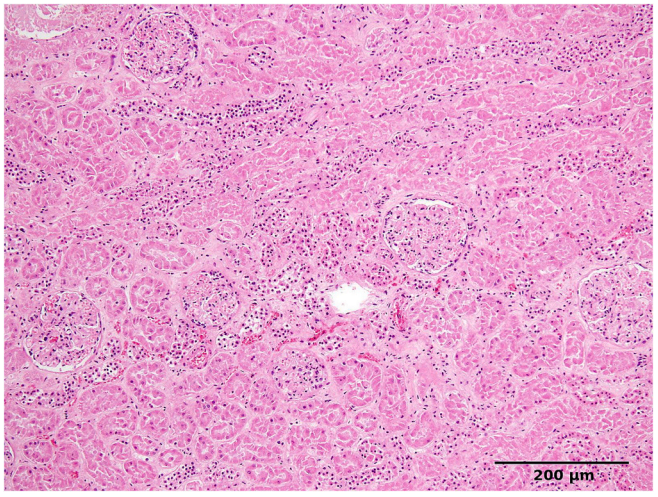

Fig. 5. Renal tubular epithelial injury with loss of normal tubular architecture, compatible with shock-related renal damage in the setting of severe septic shock (H&E, 10×; scale bar = 200 µm).

included non-fatal cases, PVL-negative *S. aureus* infections, review articles, editorials, conference abstracts without full case data, and duplicate publications.

A systematic search of PubMed/MEDLINE and Scopus was performed from database inception to 12 March 2026 using predefined search strategies combining the terms "Panton–Valentine leukocidin", "*Staphylococcus aureus*", "pneumonia", "necrotizing pneumonia", "autopsy", "postmortem", "fatal", "death", "mortality", and "septic shock". Database-specific search strategies are provided in **Supplementary Appendix S1**.

After removal of duplicates, records were screened independently based on titles and abstracts, followed by full-text assessment of potentially eligible studies. Disagreements regarding study eligibility were resolved by discussion and consensus.

Data extraction was performed using a standardized form developed prior to study selection. The following variables were collected: first author, year of publication, country, age, sex, bacterial strain, viral co-infection, presence of hemoptysis or pulmonary hemorrhage, occurrence of septic shock, and availability of postmortem confirmation. When autopsy data were available, additional pathological features were recorded, including pulmonary pattern, hemorrhagic findings, airway necrosis, abscess formation, bacterial presence in tissue, and extra-pulmonary findings.

Given the descriptive nature of the included studies and the marked heterogeneity of reported variables, a quantitative meta-analysis was not feasible. Data were therefore synthesized narratively and summarized in tabular form.

4. Results – literature review

The literature search identified a total of 818 records, including 605 from Scopus and 213 from PubMed/MEDLINE. After removal of 333 duplicate records and one retracted article, 484 records remained for screening. Of these, 445 were excluded based on title and abstract. Thirty-nine reports were sought for full-text retrieval, of which three could not be retrieved. Thirty-six articles were assessed for eligibility, and five were excluded (two non-fatal cases and three PVL-negative infections). Ultimately, 31 studies were included in the qualitative synthesis [10–40]. The study selection process is shown in the PRISMA flow diagram (Fig. 6).

The clinical and microbiological characteristics of the included cases

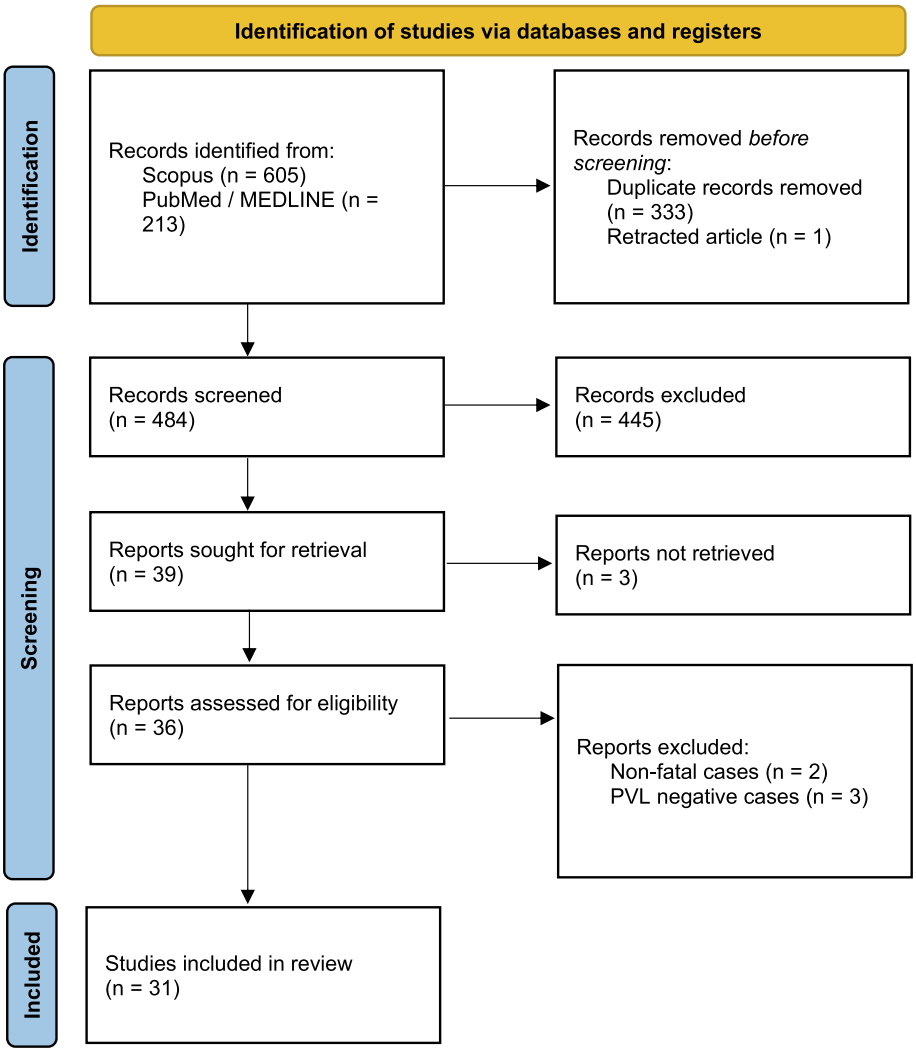

Fig. 6. PRISMA Flow diagram of the systematic review.

are summarized in Table 1. A total of 31 fatal cases of PVL-positive *Staphylococcus aureus* pneumonia were identified across Europe, North America, and Asia. Patients ranged widely in age, from infancy to older adulthood, with several cases occurring in previously healthy individuals. Both sexes were represented.

Most infections were caused by methicillin-susceptible *S. aureus* (MSSA) (20/31 cases), whereas methicillin-resistant strains (MRSA and CA-MRSA) accounted for 11/31 cases. Viral coinfection was documented in 5/31 cases, most commonly due to influenza B, with one case associated with parainfluenza infection.

Hemoptysis or pulmonary hemorrhage was reported in 14/31 cases, often described as massive or associated with hemorrhagic respiratory secretions. Septic shock was documented in 26/31 cases when reported, highlighting its strong association with fatal outcomes. Postmortem confirmation was available in 13/31 cases, including full autopsies and postmortem lung cultures.

Autopsy findings are summarized in Table 2. In cases where postmortem examination was performed, a consistent pattern of severe necrotizing pulmonary infection was observed. The most frequently reported patterns included necrotizing pneumonia, necrotizing bronchopneumonia, and hemorrhagic bronchopneumonia. Pulmonary hemorrhage was a common feature, ranging from intra-alveolar hemorrhage to massive pulmonary bleeding.

Airway involvement, including bronchial or tracheobronchial necrosis, was reported in several cases, and abscess formation or microabscesses were occasionally observed. Histological examination frequently demonstrated clusters of Gram-positive cocci within lung tissue. Extra-pulmonary findings, although less consistently reported, included pulmonary infarcts, necrotizing vasculitis, adrenal hemorrhage, fibrinopurulent pleurisy, and acute tubular necrosis.

5. Discussion

PVL-positive *Staphylococcus aureus* pneumonia represents a distinctive cause of rapidly fatal infectious disease, particularly relevant in the forensic setting. In the present study, combining an autopsy-confirmed case with a systematic review of 31 fatal cases, a consistent clinicopathological pattern emerged, characterized by rapid clinical deterioration, frequent septic shock, and necrotizing hemorrhagic pulmonary infection.

A key finding of our review is the extremely high frequency of septic shock, documented in 26 of 31 cases, underscoring the fulminant systemic progression of PVL-associated infection. In addition, hemoptysis or pulmonary hemorrhage was reported in 14 of 31 cases, often described as massive, supporting its role as a characteristic clinical feature of this condition. These findings are consistent with previous studies and reviews, including a recent analysis of pediatric PVL-producing *S. aureus* infections reporting pulmonary involvement in approximately 47% of cases and bacteremia in nearly half of patients, highlighting the invasive potential of these strains [41]. Similar

Table 1
Clinical and microbiological characteristics of reported fatal cases of Panton–Valentine leukocidin (PVL)–positive *Staphylococcus aureus* pneumonia included in the review. For each case, demographic data, bacterial strain (MSSA, MRSA or CA-MRSA), presence of viral coinfection, occurrence of hemoptysis or pulmonary hemorrhage, septic shock, and availability of postmortem confirmation are reported. NR: not reported.

First author	Year	Country	Age	Sex	Strain	Viral coinfection	Hemoptysis / pulmonary hemorrhage	Septic shock	Postmortem confirmation
Al-Talib [10]	2011	Malaysia	29	M	MSSA	NR	NR	Yes	NR
Ambrozova [11]	2013	Czech Republic	10 months	M	MSSA	NR	NR	NR	NR
Bai (case 1) [12]	2020	China	64	F	MSSA	Influenza B	NR	Yes	NR
Bai (case 2) [12]	2020	China	39	F	MSSA	Influenza B	NR	Yes	NR
Balis [13]	2007	Greece	37	F	MRSA	NR	Yes	Yes	Lung necropsy culture
Banthia [14]	2005	USA	28	M	CA-MRSA	NR	NR	Yes	Autopsy
Beilouny [15]	2008	France	47	F	MSSA	NR	NR	Yes	Autopsy
Benes [16]	2010	Czech Republic	29	M	MRSA	NR	NR	Yes	NR
Boussaud (case 1) [17]	2003	France	67	M	MRSA	NR	Yes (massive)	Yes	NR
Boussaud (case 2) [17]	2003	France	38	M	MSSA	NR	Yes (massive)	Yes	Autopsy
Campion [18]	2000	France	14	F	MSSA	NR	NR	Yes	NR
Chalumeau [19]	2005	France	4 months	M	MSSA	NR	NR	NR	Autopsy
Chetchotisakd [20]	2007	Thailand	38	M	MSSA	NR	Yes (massive)	Yes	Lung necropsy culture
Costa [21]	2025	Portugal	65	M	MSSA	NR	NR	Yes	NR
Daneshvar [22]	2011	UK	45	F	MSSA	NR	NR	Yes	NR
Escudier [23]	2015	France	16	M	MSSA	NR	Yes (massive)	Yes	Autopsy
Frazee [24]	2005	USA	31	M	CA-MRSA	NR	Yes	Yes	NR
Haider [25]	2013	UK	12	M	MSSA	NR	NR	NR	Lung necropsy culture
Honarpour [26]	2007	USA	48	F	MSSA	NR	Yes (massive pulmonary hemorrhage)	Yes	Autopsy
Hörnig-Franz [27]	2007	Germany	12	F	MSSA	NR	Yes	Yes	Autopsy
Joguet [28]	2003	France	51	M	MSSA	NR	Yes	Yes	NR
Laporte-Turpin [29]	2006	France	10	M	MSSA	NR	NR	Yes	NR
Laverdure [30]	2014	France	35	F	MSSA	NR	Yes (hemorrhagic secretions)	Yes	NR
Li [31]	2011	UK	14	M	MSSA	NR	NR	Yes	NR
Magira [32]	2007	Greece	61	F	CA-MRSA	NR	NR	Yes	NR
Mosquera [33]	2017	Spain	12	F	MRSA	NR	Yes	Yes	Autopsy
Mushtaq [34]	2008	UK	14	M	MSSA	NR	NR	Yes	NR
Obed [35]	2006	Germany	60	F	MRSA	NR	NR	Yes	NR
Rein [36]	2014	USA	38	F	MRSA	Influenza B	NR	Yes	NR
Roberts [37]	2008	USA	30	F	MSSA	Influenza B	NR	Yes	Autopsy
Trieu [38]	2009	France	Child	F	MSSA	NR	NR	NR	Autopsy
Tseng [39]	2005	Taiwan	2 years	M	CA-MRSA	NR	NR	Yes	NR
Wenzel [40]	2009	Germany	12	M	MSSA	Parainfluenza	NR	Yes	Autopsy

observations have identified PVL-producing *S. aureus* as a major determinant of highly aggressive necrotizing pneumonia associated with elevated mortality rates [5,7,8,42].

The pathophysiological mechanisms underlying this severe presentation are closely related to the cytotoxic activity of PVL, a pore-forming toxin that targets leukocytes, leading to intense inflammatory responses, tissue necrosis, and vascular damage within the lung parenchyma [3,7]. This process results in extensive parenchymal destruction and diffuse intra-alveolar hemorrhage, which are consistently reflected in the autopsy findings observed both in our case and in the reviewed literature [14,17,23,26,37,40].

Autopsy examination plays a crucial role in the identification of this entity. In cases where postmortem investigation was performed, a remarkably consistent pattern of necrotizing and hemorrhagic pulmonary infection was observed, including necrotizing pneumonia, necrotizing bronchopneumonia, and hemorrhagic bronchopneumonia. Additional features such as airway necrosis, abscess formation, and the presence of Gram-positive cocci within lung tissue further support the diagnosis [14,17,23,26,37,40]. These findings highlight the importance of integrating macroscopic, histological, and microbiological data in the investigation of rapidly fatal infectious deaths.

An important observation from our review is that most fatal cases were caused by methicillin-susceptible *S. aureus* (20/31 cases), whereas methicillin-resistant strains accounted for a minority. This suggests that PVL-associated virulence may play a more critical role in determining disease severity than antimicrobial resistance itself. In addition, viral coinfections—particularly with influenza viruses—were identified in 5 of 31 cases, supporting the hypothesis that preceding viral infections may predispose to severe PVL-associated pneumonia.

From a clinical perspective, the rapid progression of PVL-associated pneumonia poses a significant diagnostic challenge. Patients may deteriorate within hours of hospital admission, as observed in the present case, or even before microbiological confirmation is available. In such scenarios, early recognition of suggestive features—such as hemoptysis, rapidly progressive respiratory failure, and severe systemic involvement—may be crucial for prompt management.

From a forensic perspective, this entity represents an important cause of sudden or rapidly fatal infectious death. In cases characterized by a short clinical course and limited diagnostic information, postmortem examination becomes essential. Autopsy findings of necrotizing and hemorrhagic pneumonia, particularly when associated with evidence of septic shock, should raise suspicion for PVL-positive *S. aureus* infection. Targeted postmortem microbiological and molecular investigations, including testing for PVL genes, are therefore critical to establish the etiological diagnosis [43–47].

The present case further illustrates the importance of postmortem

TABLE 2
Autopsy findings reported in fatal cases of PVL-positive *Staphylococcus aureus* pneumonia. The table summarizes the main pulmonary pathological patterns, including necrotizing pneumonia, hemorrhage, airway necrosis, abscess formation, presence of bacterial clusters within lung tissue, and associated extrapulmonary findings when available. NR: not reported.

First author	Pulmonary pattern	Hemorrhage	Airway necrosis	Abscesses	Bacteria in tissue	Extra-pulmonary findings
Banthia [14]	Bilateral necrotizing pneumonia	Yes	Ulcerative tracheobronchitis	NR	NR	Pulmonary infarcts, necrotizing vasculitis
Beilouny [15]	Necrotizing bronchopneumonia	NR	NR	Multiple microabscesses	NR	NR
Boussaud [17]	Necrotizing pneumonia	Intra-alveolar	Bronchial epithelial necrosis	NR	Gram+ cocci	NR
Chalumeau [19]	Necrotizing hemorrhagic pneumonia	Massive	NR	NR	NR	NR
Escudier [23]	Necrotizing pneumonia	Massive intra-alveolar	Bronchial necrosis	NR	Gram+ cocci	NR
Honarpour [26]	Necrotizing pneumonia	Massive pulmonary hemorrhage	NR	NR	Gram+ cocci	NR
Hörnig-Franz [27]	Abscessing necrotizing bronchopneumonia	Hemorrhagic foci	NR	Yes	Cocci clusters	Multiorgan failure changes
Mosquera [33]	Bilateral abscessed pneumonia	NR	NR	Yes	MRSA isolated	Adrenal hemorrhage, fibrinopurulent pleurisy
Roberts [37]	Necrotizing bronchopneumonia	Intra-alveolar hemorrhage	Bronchial epithelial necrosis	Yes	Cocci clusters	Acute tubular necrosis
Trieu [38]	Necrotizing pneumonia	NR	NR	NR	NR	NR
Wenzel [40]	Confluent hemorrhagic bronchopneumonia	Extensive intra-alveolar	Tracheobronchial necrosis	NR	Cocci clusters	NR

investigation in rapidly fatal infections. In our patient, the autopsy findings of necrotizing hemorrhagic bronchopneumonia prompted targeted microbiological and molecular analyses, ultimately leading to the identification of PVL-positive *S. aureus*. Previous studies have shown that PVL-positive *S. aureus* bacteremia is frequently associated with pulmonary infection, supporting the link between bloodstream infection and necrotizing pneumonia [48]. This integrated diagnostic approach is essential in cases where ante-mortem data are limited or inconclusive.

5.1. Limitations

This study has several limitations. First, the number of available cases is limited, reflecting the rarity of reported fatal PVL-positive *S. aureus* pneumonia. Second, the included studies are predominantly case reports and case series, which are subject to publication bias and incomplete reporting of clinical and pathological data. Third, heterogeneity in data reporting limited the possibility of performing a quantitative meta-analysis. A further limitation is that fibrinopurulent pleuritis was documented only at gross autopsy examination and therefore should be interpreted as a macroscopic finding rather than as a histopathological diagnosis. Finally, postmortem examination was not available in all cases, potentially limiting the assessment of pathological patterns.

6. Conclusions

PVL-positive *Staphylococcus aureus* pneumonia represents a rare but highly aggressive infection that may rapidly progress to respiratory failure, septic shock, and death. The present case, together with the systematic review of 31 fatal cases, highlights a distinctive clinicopathological pattern characterized by rapid clinical deterioration, frequent septic shock, and necrotizing hemorrhagic pneumonia.

Given the fulminant course of the disease, autopsy combined with postmortem microbiological and molecular analyses plays a crucial role in establishing the diagnosis. Recognition of this entity and its characteristic features may assist both clinicians and forensic pathologists in identifying PVL-associated pneumonia as a cause of sudden or rapidly fatal infectious death.

Author contribution

J.C.: Conceptualization, Writing – original draft, Investigation, Visualization (microphotography). A.L.S.: Investigation, Visualization (microphotography), Writing – review & editing. R.C.: Writing – review & editing. E.R.: Writing – review & editing. E.C.: Writing – review & editing. M.P.B.: Conceptualization, Supervision, Visualization (microphotography), Writing – review & editing. All authors approved the final version of the manuscript.

Human Ethics and Consent to Participate declarations

not applicable.

Funding

The authors did not receive support from any organization for the submitted work.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

none.

Appendix B. Supplementary appendix 1

Database search strategy. Supplementary data to this article can be found online at [https://doi.org/10.1016/j.legalmed.2026.102935].

Data availability

All relevant data are within the paper.

References

[1] C. De la Calle, L. Morata, N. Cobos-Trigueros, J.A. Martinez, C. Cardozo, J. Mensa, et al., *Staphylococcus aureus* bacteremic pneumonia, Eur. J. Clin. Microbiol. Infect. Dis. 35 (2016) 497–502, https://doi.org/10.1007/s10096-015-2563-5.

[2] Y. Gillet, T. Henry, F. Vandenesch, Fulminant staphylococcal infections, Microbiol. Spectrum 6 (2018), https://doi.org/10.1128/microbiolspec.GPP3-0045-2018.

[3] W.T. Lo, C.C. Wang, Panton-valentine leukocidin in the pathogenesis of community-associated methicillin-resistant Staphylococcus aureus infection, Pediatr. Neonatol. 52 (2011) 59–65, https://doi.org/10.1016/j.pedneo.2011.02.004.

[4] M.L. Castellazzi, S. Bosis, I. Borzani, C. Tagliabue, R. Pinzani, P. Marchisio, et al., Panton-valentine leukocidin *Staphylococcus aureus* severe infection in an infant: a case report and a review of the literature, Ital. J. Pediatr. 47 (2021) 158, https://doi.org/10.1186/s13052-021-01101-8.

[5] Y. Gillet, B. Issartel, P. Vanhems, J.C. Fournet, G. Lina, M. Bes, et al., Association between Staphylococcus aureus strains carrying gene for Panton-valentine leukocidin and highly lethal necrotising pneumonia in young immunocompetent patients, Lancet 359 (2002) 753–759, https://doi.org/10.1016/S0140-6736(02)07877-7.

[6] J. Cuddihy, S. Patel, N. Mughal, C. Lockie, R. Trimlett, S. Ledot, et al., Near-fatal Panton-valentine leukocidin-positive *Staphylococcus aureus* pneumonia, shock and complicated extracorporeal membrane oxygenation cannulation: a case report, World J. Crit. Care Med. 10 (2021) 301–309, https://doi.org/10.5492/wjccm.v10.i5.301.

[7] Y. Gillet, P. Vanhems, G. Lina, M. Bes, F. Vandenesch, D. Floret, et al., Factors predicting mortality in necrotizing community-acquired pneumonia caused by Staphylococcus aureus containing Panton-valentine leukocidin, Clin. Infect. Dis. 45 (2007) 315–321, https://doi.org/10.1086/519263.

[8] Y. Gillet, A. Tristan, J.P. Rasigade, M. Saadatian-Elahi, C. Bouchiat, M. Bes, et al., Prognostic factors of severe community-acquired staphylococcal pneumonia in France, Eur. Respir. J. 58 (2021), https://doi.org/10.1183/13993003.04443-2020.

[9] M.J. Page, J.E. McKenzie, P.M. Bossuyt, I. Boutron, T.C. Hoffmann, C.D. Mulrow, et al., The PRISMA 2020 statement: an updated guideline for reporting systematic reviews, BMJ 372 (2021) n71, https://doi.org/10.1136/bmj.n71.

[10] H. Al-Talib, H. Hasan, C.Y. Yean, S.M. Al-Ashwal, M. Ravichandran, Fatal necrotizing pneumonia caused by Panton-valentine leukocidin-producing hospital-acquired Staphylococcus aureus: a case report, Jpn. J. Infect. Dis. 64 (2011) 58–60.

[11] H. Ambrozova, V. Maresova, M. Fajt, P. Pavlicek, H. Rohacova, I. Machova, et al., The first case of fatal pneumonia caused by Panton-valentine leukocidin-producing *Staphylococcus aureus* in an infant in the Czech Republic, Folia Microbiol. (Praha) 58 (2013) 225–228, https://doi.org/10.1007/s12223-012-0198-5.

[12] B. Bai, H. Wang, M. Li, X. Ma, J. Zheng, Q. Deng, et al., Two cases of influenza B virus-related fatal fulminant pneumonia complicated with *Staphylococcus aureus* infection in China diagnosed using next-generation sequencing, Front. Public Health 8 (2020) 121, https://doi.org/10.3389/fpubh.2020.00121.

[13] E. Balis, C. Diacaki, P. Tselioti, D. Perimeni, S. Vourli, A. Vatopoulos, et al., Community-acquired pneumonia and bacteremia due to methicillin-resistant Staphylococcus aureus carrying Panton-valentine leukocidin gene in Greece: two case reports and literature review, J. Chemother. 19 (2007) 703–708, https://doi.org/10.1179/joc.2007.19.6.703.

[14] S. Banthia, V.G. Meka, S.K. Pillai, K. Wener, R. Freedman, S.S. Pandya, et al., A fatal case of necrotizing pneumonia caused by community-associated methicillin-resistant *Staphylococcus aureus*, Infect. Dis. Clin. Pract. 13 (2005) 132–138, https://doi.org/10.1097/01.idc.0000163498.80152.28.

[15] B. Beilouny, A. Ciupea, C. Eloy, G. Simon, Fatal community-acquired pneumonia due to Staphylococcus aureus carrying Panton-valentine leukocidin genes after a stay in Africa, Intensive Care Med. 34 (2008) 388–389, https://doi.org/10.1007/s00134-007-0909-2.

[16] J. Beneš, O. Myslivec, J. Laštíková, A. Gabrielová, P. Petráš, R. Pantůček, Septic shock in fatal pneumonia caused by Staphylococcus aureus: the significance of producing Panton-valentine leukocidin, Anesteziol. Intenziv. Med. 21 (2010) 337–341.

[17] V. Boussaud, A. Parrot, C. Mayaud, M. Wislez, M. Antoine, C. Picard, et al., Life-threatening hemoptysis in adults with community-acquired pneumonia due to Panton-valentine leukocidin-secreting *Staphylococcus aureus*, Intensive Care Med. 29 (2003) 1840–1843, https://doi.org/10.1007/s00134-003-1906-z.

[18] A. Campion, R. Cremer, S. Leteurtre, C. Fourier, F. Leclerc, Fatal community-acquired pneumonia due to Staphylococcus aureus infection, Réanim. Urgences. 9 (2000) 301–304.

[19] M. Chalumeau, P. Bidet, G. Lina, M. Mokhtari, M.C. André, D. Gendrel, et al., Transmission of Panton-valentine leukocidin-producing *Staphylococcus aureus* to a physician during resuscitation of a child, Clin. Infect. Dis. 41 (2005) e29–e30, https://doi.org/10.1086/431591.

[20] P. Chetchotisakd, S. Anunnatsiri, S. Puapermpoonsiri, C. Prariyachatgul, J. Chumpol, A rapidly fatal case of Panton-valentine leukocidin-positive Staphylococcus aureus necrotizing pneumonia in an HIV-infected patient, southeast Asian, J. Trop. Med. Public Health. 38 (2007) 690–694.

[21] C.A. Costa, H. Inácio, S. Trevas, Â. Simas, Panton-Valentine leukocidin in necrotizing pneumonia: a case report, Cureus 17 (2025) e78774, https://doi.org/10.7759/cureus.78774.

[22] C. Daneshvar, N.C. Smith, D.J. Waghorn, G.A. Luzzi, C.G. Wathen, Community-acquired pneumonia due to Panton-valentine leukocidin-producing Staphylococcus aureus in an HIV-2-infected patient, Int. J. STD AIDS 22 (2011) 610–612, https://doi.org/10.1258/ijsa.2011.011058.

[23] E. Escudier, L. Pagani, J. Gaillat, M. Sirodot, A. Levrat, Discussion of a fatal case of necrotizing pneumonia due to Panton-valentine leukocidin-producing Staphylococcus aureus, Réanimation 25 (2016) 361–365, https://doi.org/10.1007/s13546-015-1143-4.

[24] B.W. Frazee, T.O. Salz, L. Lambert, F. Perdreau-Remington, Fatal community-associated methicillin-resistant Staphylococcus aureus pneumonia in an immunocompetent young adult, Ann. Emerg. Med. 46 (2005) 401–404, https://doi.org/10.1016/j.annemergmed.2005.06.003.

[25] S. Haider, D. Wright, Panton-valentine leukocidin *Staphylococcus* causing fatal necrotising pneumonia in a young boy, BMJ Case Rep. (2013), https://doi.org/10.1136/bcr-2013-200458.

[26] N. Honarpour, J.T. Mao, A case of fatal community-acquired necrotizing pneumonia caused by Panton-valentine leukocidin-positive methicillin-sensitive Staphylococcus aureus, Curr. Respir. Med. Rev. 3 (2007) 49–51.

[27] I. Hörnig-Franz, B.C. Kahl, W. Tebbe, C. Kersting, H. Bürger, K. Nolte, et al., Necrotising pneumonia caused by *Staphylococcus aureus* (PVL gene positive), Monatsschr. Kinderheilkd. 155 (2007) S10–S15, https://doi.org/10.1007/s00112-007-1573-0.

[28] G. Joguet, J.M. Perez, C. Herrmann, M. Strobel, Lethal necrotising pneumonia by Panton-valentine leukocidin-producing Staphylococcus aureus, Med. Mal. Infect. 33 (2003) 272–273, https://doi.org/10.1016/S0399-077X(03)00056-3.

[29] E. Laporte-Turpin, M.O. Marcoux, I. Claudet, E. Grouteau, P. Micheau, S. Fédérici, et al., Necrotizing pneumonia and arthritis due to *Staphylococcus aureus* producing Panton-valentine leukocidin in a 10-year-old boy, Arch. Pediatr. 13 (2006) 449–452, https://doi.org/10.1016/j.arcped.2006.01.014.

[30] F. Laverdure, C. Neulier, J. Sudant, S. Legriel, F. Bruneel, Fatal Panton-valentine leukocidin-associated Staphylococcus aureus necrotizing pneumonia, Ann. Fr. Anesth. Reanim. 33 (2014) 596–599, https://doi.org/10.1016/j.annfar.2014.07.748.

[31] Z. Li, D.L. Stevens, S.M. Hamilton, T. Parimon, Y. Ma, A.M. Kearns, et al., Fatal Staphylococcus aureus hemorrhagic pneumonia: genetic analysis of a unique clinical isolate producing both PVL and TSST-1, PLoS One 6 (2011) e27246, https://doi.org/10.1371/journal.pone.0027246.

[32] E.E. Magira, D. Zervakis, C. Routsi, M. Kontogiorgi, C. Roussos, S. Nanas, et al., Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-valentine leukocidin genes: a lethal cause of pneumonia in an adult immunocompetent patient, Scand. J. Infect. Dis. 39 (2007) 466–469, https://doi.org/10.1080/00365540601173965.

[33] M. Mosquera, L. Montero, F.J. Pérez-Lescure, A. Aragón, Pediatric case of fatal necrotizing pneumonia due to Panton-valentine leukocidin-positive methicillin-resistant Staphylococcus aureus in Spain, Enferm. Infecc. Microbiol. Clin. 37 (2019) 63, https://doi.org/10.1016/j.eimc.2017.12.011.

[34] F. Mushtaq, S. Hildrew, G. Okugbeni, R.W. Ellis, Necrotizing haemorrhagic pneumonia proves fatal in an immunocompetent child due to Panton-valentine leucocidin, toxic shock syndrome toxins 1 and 2 and enterotoxin C-producing Staphylococcus aureus, Acta Paediatr. 97 (2008) 985–987, https://doi.org/10.1111/j.1651-2227.2008.00812.x.

[35] A. Obed, A.A. Schnitzbauer, T. Bein, N. Lehn, H.J. Linde, H.J. Schlitt, Fatal pneumonia caused by Panton-valentine leukocidin-positive methicillin-resistant Staphylococcus aureus transmitted from a healthy donor in living-donor liver transplantation, Transplantation 81 (2006) 121–124, https://doi.org/10.1097/01.tp.0000188144.99614.a5.

[36] J.L. Rein, A.M. Etra, J.J. Patel, J.L. Stein, A.L. Rivers, H.B. Gershengorn, et al., Death of woman with peripartum influenza B virus infection and necrotizing pneumonia, Emerg. Infect. Dis. 20 (2014) 1259–1260, https://doi.org/10.3201/eid2007.131791.

[37] J.C. Roberts, S.P. Gulino, K.K. Peak, V.A. Luna, R. Sanderson, Fatal necrotizing pneumonia due to a Panton-valentine leukocidin-positive community-associated methicillin-sensitive Staphylococcus aureus and influenza co-infection: a case report, Ann. Clin. Microbiol. Antimicrob. 7 (2008) 5, https://doi.org/10.1186/1476-0711-7-5.

[38] T.V. Trieu, J. Gaudelus, S. Lefevre, A.M. Teychene, I. Poilane, A. Colignon, et al., Sudden death caused by *Staphylococcus aureus* carrying Panton-valentine leukocidin gene in a young girl, BMJ Case Rep. (2009), https://doi.org/10.1136/bcr.01.2009.1430.

[39] M.H. Tseng, B.H. Wei, W.J. Lin, J.J. Lu, S.Y. Lee, S.R. Wang, et al., Fatal sepsis and necrotizing pneumonia in a child due to community-acquired methicillin-resistant *Staphylococcus aureus*: case report and literature review, Scand. J. Infect. Dis. 37 (2005) 504–507, https://doi.org/10.1080/00365540510035368.

[40] J.J. Wenzel, J. Hentschel, W. Silvis, W. Permanetter, J. Mattes, B. Kochanowski, et al., Rapidly fatal necrotizing pneumonia in a 12-year-old boy caused by co-infection with parainfluenza virus type 1 and Panton-valentine leukocidin-positive methicillin-sensitive *Staphylococcus aureus*, Infection 37 (2009) 75–77, https://doi.org/10.1007/s15010-008-7324-0.

[41] V. Garbo, L. Venuti, G. Boncori, C. Albano, A. Condemi, G. Natoli, et al., Severe Panton-valentine-leukocidin-positive *Staphylococcus aureus* infections in pediatric age: a case report and a literature review, Antibiotics 13 (2024) 1192, https://doi.org/10.3390/antibiotics13121192.

[42] K. Hussain, A. Bandyopadhyay, N. Roberts, N. Mughal, L.S.P. Moore, L.C. Fuller, Panton-valentine leucocidin-producing Staphylococcus aureus: a clinical review, Clin. Exp. Dermatol. 47 (2022) 2150–2158, https://doi.org/10.1111/ced.15355.

[43] A.L. Santunione, J. Camatti, R. Cecchi, First forensic case of fatal *Salmonella typhimurium* var. Copenhagen gastroenteritis diagnosed by postmortem microbiology, Am J Forensic Med Pathol (2025), https://doi.org/10.1097/PAF.0000000000000994.

[44] J. Camatti, M.P. Bonasoni, A.L. Santunione, R. Cecchi, E. Radheshi, E. Carretto, Postmortem microbiology in forensic diagnostics: interpretation of infectious causes of death and emerging applications, Diagnostics (Basel). 16 (2026) 2, https://doi.org/10.3390/diagnostics16020325.

[45] J. Camatti, M. Tudini, M.P. Bonasoni, A.L. Santunione, R. Cecchi, E. Radheshi, et al., *Candida albicans* meningoencephalitis after vestibular schwannoma surgery: an autopsy-confirmed case report, Diagnostics (Basel). 16 (2026) 2, https://doi.org/10.3390/diagnostics16020228.

[46] C. Ferronato, J. Camatti, F. Zucchi, P. Ius, R. Cecchi, A.L. Santunione, Fatal Epstein-Barr virus myopericarditis in a young adult male, Am J Forensic Med Pathol 46 (2025), https://doi.org/10.1097/PAF.0000000000001053.

[47] A.L. Santunione, J. Camatti, F. Zucchi, C. Ferronato, F. Ferrari, S. Caramaschi, et al., Fatal Waterhouse-Friderichsen syndrome caused by *Streptococcus pneumoniae* in a vaccinated adult with traumatic splenectomy: a case report, Leg. Med. (Tokyo) 72 (2025), https://doi.org/10.1016/j.legalmed.2025.102569.

[48] M.D. Qu, H. Kausar, S. Smith, P.G. Lazar, A.R. Kroll-Desrosiers, C. Hollins 3rd, et al., Epidemiological and clinical features of Panton-valentine leukocidin-positive *Staphylococcus aureus* bacteremia: a case-control study, PLoS One 17 (2022) e0265476, https://doi.org/10.1371/journal.pone.0265476.