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Review article

Small, but smelly: the importance of *Solobacterium moorei* in halitosis and other human infections

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ARTICLE INFO

Keywords
Microbiology
Epidemiology
Infectious disease
Dentistry
Clinical research
Solobacterium moorei
Halitosis
Malodor
Bacteremia
Anaerobe
MALDI-TOF MS

ABSTRACT

Solobacterium moorei (*S. moorei*) has been described as Gram-positive, non spore forming, obligate anaerobic bacillus from human feces. The traditional culture and identification of these strains is very difficult (as the strains are often not cultivable or they grow only relatively slowly, in addition to producing only a very few positive biochemical reactions in commercially available identification kits); thus, reliable identification may only be carried out using methods, such as matrix-assisted laser desorption/ionization time-of-flight mass spectrometry and DNA sequencing. Regarding its pathogenic role, the relevance of *S. moorei* in halitosis (oral malodor) has a good standing, as it has been suggested by multiple studies, while the isolation of these bacteria from invasive infections is very rare; there are only a few reports available in the literature, regarding infections outside the oral cavity. Based on these reports, affected patients are predominantly characterized compromised immunity and are frequently associated with a dental focus of infection. The aim of our present review is to summarize the currently available knowledge on the pathogenic role of *S. moorei* in halitosis and other infections and to emphasize the relevance of this neglected anaerobic pathogen.

1. Introduction, halitosis as a clinical problem

Halitosis (foetor oris, foetor ex ore, colloquially: foul breath, oral malodour) is the medical term used to describe all of the unpleasant smells and odors from the expired air, mouth or from outside the oral cavity, without any information about the correct source [1]. Approximately 10–30% of the adults in US and in the European Union and 30–60% of adults in developing countries suffer from this type of oral malodor [2]. Halitosis may occur under physiological conditions, such as nocturnal hyposalivation, however, this may be amended by proper oral hygiene [3]. Other causes (especially for transient malodor) without any pathological conditions include smoking, mastication of different parts of vegetables and consumption of various spices (e.g., onion or garlic) and alcohol [4, 5, 6, 7]. Hyposalivation/xerostomia (due to Sjögren's syndrome, adverse events associated with anticholinergic drugs, centrally-acting psychoactive agents, chemotherapy and radiation therapy) is also a very common cause of halitosis [8, 9, 10]. Some patients may suffer from halitophobia, a condition during which the affected person constantly fears that the smell of their oral region is regarded as repulsive by other people [8]. When assessing the origins of halitosis, most cases are found to originate from the oral cavity (80–90%; intra-oral halitosis: IOH, including infections or abscesses in the oral cavity, deep carious lesions, periodontal diseases, pericoronitis), while remaining cases are corresponding to non-oral causes (extra-oral halitosis: EOH) [11]. Based on the classification of Aydin and Harvey-Woodworth, the revised classification of halitosis includes physiological (Type 0) and pathological Type 1–5 (1: oral, 2: airway, 3: gastro-esophageal, 4: blood-borne, 5: objective) halitosis [12]. Ingestion of some chemical compounds may also cause halitosis – if the metabolic end-products of these agents are stable in blood, or they are capable of increasing the pH locally – facilitating the growth of halitosis-causing bacteria in the oral cavity [13, 14, 15, 16, 17]. Gastro-esophageal reflux disease (GERD) is also an important cause of malodor, with a similar underlying mechanism (increasing the pH in the oral cavity) [18]. The presence and overgrowth of many microorganisms has been implicated in causing oral malodor; with the advent of novel microbiological and diagnostic technologies, novel microbial species have also been associated with halitosis [19]. Although the literature regarding the topic

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https://doi.org/10.1016/j.heliyon.2020.e05371
Received 6 July 2020; Received in revised form 25 August 2020; Accepted 26 October 2020
Available online xxx

is scarce, one of the novel species suggested to contribute to halitosis is *Solobacterium moorei* (*S. moorei*) [20]. In the present review, we aimed to surmise the the currently available knowledge on this neglected anaerobic pathogen – regarding its role in halitosis, in addition to the relevance of these bacteria in other, invasive human infections – to increase its notoriety among clinicians, dentists and clinical microbiologists.

2. Microbiology of *Solobacterium moorei*

S. moorei has been described as Gram-positive, non-spore-forming, obligate anaerobic bacillus from human feces by Kageyama and Benno in 2000, currently being the only member of the *Solobacterium* genus [21]. The bacterium was considered first as a native member of the human intestinal microbiome, but later, their pathogenic role has been identified in halitosis [22]. At the time of their discovery, phylogenetic analysis disclosed that the isolated strains were members of the *Clostridium* subphylum of Gram-positive bacteria [21]. The name of this species was originally known as *Bulleida extructa* or *B. moorei*; the genus *Bulleida* is a member of the *Erysipelotrichidae* family, which shows pronounced differences from the other genera of non-spore-forming Gram-positive bacilli, e.g., *Bifidobacterium*, *Cutibacterium* and *Lactobacillus* [23]. According to the original investigation, the tested *S. moorei* strains showed a 93% sequence similarity only to *B. extructa*, in addition to other phenotypic differences, therefore, a brand new genus was created [24]. Based on 16S rRNA gene sequencing data, *S. moorei* only has around 86% sequence homogeneity to *Erysipelothrix rhusiopathiae* and 87% to *Holdemania filiformis*, other representative members of the *Erysipelotrichidae* family [25]. The first deposited type strain of *Solobacterium moorei* is JCM 10645T [6]. Phenotypically, these rods are 0.2 μm by 0.4–0.7 μm in size and they do not possess flagella [1]. The traditional culture and identification of these strains is very difficult, as the strains are often not cultivable or they grow only relatively slowly, in addition to producing only a very few positive biochemical reactions in commercially available identification kits [21, 26]. The traditional phenotypic biochemical tests (as shown in the literature) are not adequate for the identification of these bacteria, due to the phenomenon of wide variety of phenotypic presentation, i.e. the isolates are not able to produce an uniform amount of positive and negative reactions to be reliably identified by biochemical methods [21, 22, 26]. Most of the published clinical *S. moorei* isolates characterized ferment glucose, galactose, fructose, maltose and ribose and hydrolyse esculine [21, 22, 26, 27]. The main product from glucose fermentation is acetic acid, while the other non-spore-forming anaerobic bacteria (e.g., *Bifidobacterium*, *Cutibacterium*, *Eubacterium* and *Lactobacillus*) may produce propionic acid, lactic acid, acetic acid, butyric acid and formic acid [28]. Zheng et al. reported that by using Rapid ID 32A test (bioMérieux, Marcy-l'Étoile, France), the investigated *S. moorei* clinical strains were show to produce α- and β-galactosidase (which play important roles in halitosis), α-glucosidase, arginine dihydrolase, arginine arylamidase, leucine arylamidase, proline arylamidase, alkaline phosphatase enzymes [24]. In a similar experiment, nitrate reduction, esterase and valine arylamidase production was also shown by the type strain JCM 10645T by the API ZYM system (bioMérieux, Marcy-l'Étoile, France) [24]. After 48–72 h of strict anaerobic cultivation, *S. moorei* forms small, grey colonies with a 0.5–1 mm diameter, some strains were described as α-haemolytic on anaerobic blood agar [24], although the isolate published by Lau et al. was non-haemolytic [27]. *S. moorei* strains have the ability to adhere to the lipophilic surface molecules of the oral epithelial cells through adhesins of hydrophobic character [29]. In addition, they may produce biofilms in the oral cavity; this biofilm-formation may be the first key step in the ecological succession in the mouth, leading to the development of halitosis [30].

Due to these hindrances, i.e. obscurity of the pathogen, difficulties in the isolation and correct species-level identification of *S. moorei* strains (especially in mixed cultures), there is a considerable underestimation of the real clinical significance of these bacteria in clinical situations [31]. The amount of publications concerning *S. moorei* are still scarce, nevertheless, the reported cases involving *S. moorei* infections increased significantly in recent years [21, 24, 27, 32, 33, 34, 35, 36, 37]. Nowadays, there has been considerable development and technological advancements in the methods used by routine clinical microbiology laboratories. Molecular diagnostic methods, such as matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF) and DNA sequencing methods are essential for finding and appropriately identifying these bacteria from human clinical specimens [21, 22, 24, 38, 39, 40]. A summarizing table on the relevance of various microbiological techniques in the identification and diagnosis of *S. moorei* is presented in Table 1.

3. Role of *S. moorei* halitosis

The presence of anaerobic bacteria in clinical specimens can easily be distinguished owing to their putrescent smell [41]. This smell is due to the anaerobic respiration of these bacteria and the production of indole, skatole, polyamines, volatile sulfur compounds (VSCs; such as hydrogen sulfide, dimethyl sulfide, methyl mercaptan and allyl methyl sulfide), organic acids (e.g. amines such as cadaverine and putrescin; acetic acid and propionic acid) and short-chain fatty acids (SCFAs; including isovaleric and isobutyric acid) [42, 43]. The dorsum of the tongue provides a suitable environment for the accumulation of bacteria associated with halitosis, as the oxygen levels in the deep crypts of the tongue tend to be very low [44]. Species of the genera *Bacteroides*, *Centipeda*, *Eikenella, Fusobacterium*, *Porphyromonas*, *Prevotella, Tannerella* and *Treponema* have been highlighted as potent producers of the main VSCs and related compounds implicated in halitosis [45]. In addition, most of the abovementioned bacteria has been described as important periodonto-pathogenic species [46]. Out of the VSCs, *S. moorei* is capable to converting cysteine into hydrogen sulfide; in contrast –based on the results of Tanabe et al. and de Lima et al. – these bacteria are unable to produce methyl mercaptan from methionine or to produce other VSCs from complex proteins [47, 48]. The enzyme responsible (and the gene encoding for this enzyme) has been identified as the cysteine desulfhydrase, catalyzing the breakdown of cysteine into hydrogen sulfide, ammonia and pyruvate [47]. In addition, they have noted that *S. moorei* can only produce VSCs from mucin in the presence of an exogenous protease (e.g., Arg-gingipain produced by *P. gingivalis*) [48]. Nevertheless, as the dorsal region of the tongue is a rich source of proteases, VSC-production may in fact be important for *S. moorei in vivo* [49]. This might explain why there were higher levels of *S. moorei* found in the tongue coatings and saliva of patients with halitosis [50]. Due to its β-galactosidase-activity, *S. moorei* can also produce VSCs from salivary glycoproteins; this is relevant, considering that increased β-galactosidase-activity in the saliva has been associated with oral malodor [51, 52].

There is evidence to suggest that even very low concentrations of VSCs may be toxic [53, 54]. The toxicity may be related to the mechanisms of action of the agents that comprise VSCs; e.g., hydrogen sulfide can split protein disulfide bonds to form persulfide groups, bind essential metal ions and may inhibits enzymes such as myeloperoxidase, catalase, carbonic anhydrase and Na^+/K^+ ATP-ase, thus potentiating the mutagenicity of hydrogen peroxide [53, 54]. Another source of VSCs for example methyl mercaptan, increases the permeability of intact oral mucosa and stimulates cytokine production, leading to inflammation; for example, VSCs may induce the secretion of IL-8, which will subsequently induce osteoclast differentiation and contribute to the development of periodontitis [55, 56, 57]. Increased VSCs levels even may play a potential role in the link between oral infection and systemic diseases, such as preterm low birth weight and cardiovascular

Table 1
Relevance of various microbiological techniques in the identification and diagnosis of *S. moorei*.

Method	Description	Characteristics	Producer	Advantage	Disadvantage
Conventional methods	Gram stain, wet mount characteristics, and susceptibility to metronidazole, vancomycin, kanamycin, colistin, and bile	Obligately anaerobic, Gram-positive, non-motile, nonsporing rod, Cell size: 0.2 μm x 0.4–0.7 μm	-	Essential as a basic test	The strains grow slowly and phenotypic variations appear to be commonly exhibited by different strains.
Gas-liquid chromatography (GLC)	Differentiation is based on fermentation products from glucose	Moderate amounts of acetic, lactic, and butyric acids and some strains also produced a small amount of pyruvic acid from PYFG broth.	-	None, outdated method.	-
Fermentation of carbohydrates and enzyme tests		Acid production from ribose, glucose, galactose, fructose, and maltose No acid production from arabinose, xylose, rhamnose, mannose, sucrose, cellobiose, lactose, trehalose, raffinose, melezitose, starch, glycogen, mannitol, sorbitol, inositol, erythritol, esculin, salicin, or amygdalin. Hydrolysis of starch: negative. esculin positive. Negative for: gas formation, indole production, nitrate reduction, gelatin liquefaction.	Diagnostic tablets/ disks Statens Serum Institut Diagnostica, Copenhagen, Denmark, Rosco Diagnostica, Taastrup, Denmark	None.	*S. moorei* strains produce relatively few positive biochemical reactions. The phenotypic identification of the isolates were not able to produce uniform amount of positive and negative reactions, the isolates also differed from the ones previously described.
Commercially available tests	Fermentation of carbohydrates and enzyme tests		Vitek 2 ANC, BBL Crystal Anaerobe, RapID ANA II, API ID32A	None.	*S. moorei* cannot be identified using any commercially available identification kits, and report results as "no identification" There are differences in results between different commercial kits may be due to different amounts of the substrate or different reagents for detection.
MALDI-TOF MS	Ribosomal protein analysis		Vitek (bioMérieux) or Bruker Matrix-Assisted Laser Desorption/Ionization Time-of Flight (MALDI-TOF)	Suitable, fast, cheap, but requires expensive instruments and software. Requires continuous software updates.	The strains grow slowly and the organism cannot be identified reliably by MALDI-TOF. The organism's spectrum is unavailable in the older databases.
16S rRNA gene sequencing				DNA sequencing is necessary in the process of identifying this bacterium from clinical samples.	Only this method is considered truly reliable.

diseases [58]. Ierardi et al. reported a possible relationship between halitosis and *Helicobacter pylori* carriage in the stomach [15].

Years after its discovery, infections caused by *S. moorei* have been seldom reported, and in these cases, the bacteria were mainly implicated in different types of oral diseases: periodontal diseases, periimplantitis [33], dentoalveolar abscesses [36], subgingival plaques from patients with refractory periodontitis [59], localized aggressive periodontitis [60], halitosis [22, 64, 65], endodontic infections [14, 63], peri-radicular lesions [35], infected root canals [34]. As of now, there is insufficient evidence to suggest a role to *S. moorei* in the development of dental caries [20]. Schirrmeister et al. [35] found that *S. moorei* and *F. nucleatum* were the most common isolates in their study of n = 10 periradicular infections; these organisms were each found in six of the patients, and they were found in the same infections in five of these cases. Some of these abovementioned studies showed that *S. moorei* mostly resides in the oral region of patients experiencing halitosis, whereas the detection frequency in healthy volunteers was much lower. *S. moorei* adheres to oral epithelial cells through the use of their adhesins and the subsequent formation of bacterial biofilm is thought to be a key step in the development of halitosis [4]. In their study Haraszthy et al. investigated the role of „non-culturable" bacteria in halitosis: they have used direct amplification of 16S rDNA, together with traditional microbial culture methods to identify the oral microbiome in the patients enrolled in the study. They found a greater bacterial species-diversity in samples taken from the dorsal surface of the tongue of subjects with halitosis: 32 species (13 "uncultured" or "unidentified" species) in the halitosis group, while 17 species only in the control group; *S. moorei* was considered a key, or „signal" bacterial species among the species demonstrative of the halitosis group. In this study, *S. moorei* was found in all halitosis subjects (100%), however, it was not detected by either culture or direct amplification of bacterial nucleid acid in the control subjects [61]. It is interesting to note that in their study, only 14% of tested individuals without oral malodor were positive for *S. moorei*, but all of these patients were experiencing peri-

odontitis [61]. In a previous study in 2003, Kazor et al. associated the presence of halitosis with *S. moorei*, identifying it in only 1 out of 5 subjects without halitosis and in 3 out of 6 subjects with halitosis [62]. According to Gonzales et al. who carried out a EuroPerio9 study in 2019, *S. moorei* has the ability to generate VSCs in monospecies biofilms, although at lower concentrations than *F. nucleatum* or *P. gingivalis* [64]. The participation of *S. moorei* in a biofilm fosters the presence of pigmented *Prevotella* and *Porphyromonas* species in the biofilm. This could be explained by the β-galactosidase activity of *S. moorei*, which would provide greater access to nutrients for proteolytic bacteria, such as *P. gingivalis* or *P. intermedia* [24, 65]. Thus, *S. moorei* would facilitate integration of VSC-producing bacteria in the biofilm, but on the other hand, it is necessary to study the odor power of the VSCs that appeared in biofilms with *S. moorei* to establish how closely associated they are to bad breath [66]. In a study by Bernardi et al., culture-dependent and independent methods were carried out to characterize the composition of the tongue biofilm of halitosis patients: in their report; their study also highlighted the role of *S. moorei* (together with *F. periodontium*, *P. melaninogenica* and *T. forsythia*) in halitosis [67].

4. Role of *S. moorei* in human infections, other than halitosis

Although *S. moorei* was mostly isolated from human feces and was described as a member of the oral microbiota, this microorganism should also be considered as an opportunistic pathogen, responsible for severe infections. In addition to oral diseases, *S. moorei* has been reported to cause various infections, although the reports concerning extra-oral infections are relatively rare [68]. Based on the currently published reports, cases of infections caused by *S. moorei* mainly include bloodstream and surgical wound infections. These infections were associated with different types of cancers, an immunosuppressed state and some other predisposing factors, such are intravenous drug abuse, thrombosis and other bacterial and viral infections [20]. There are only eight different reports on bloodstream infection caused by *S. moorei* in the literature (presented on a patient-by-patient basis in Table 2.). Interestingly, in many affected patients, the source of the infection was presumed to be an abscess in the oral region. It has been suggested that the VSC-production of halitosis-causing bacteria (high concentrations of VSCs lead to increased permeability of the oral mucosa) The first case of bloodstream infection was described by Detry et al. in 2006 from a University Hospital in Belgium: the patient was a 67-year-old male with multiple myeloma, when the source of infection was presumably related to multiple dento-alveolar abscesses [69]. In a study concerning anaerobic blood culture positivity between 2013 and 2017 (which coincided with the introduction of MALDI-TOF MS in the routine diagnostic workflow of the laboratory) in a Southern Hungarian tertiary-care hospital by Gajdács et al., out of the n = 423 strict anaerobic isolates, four novel species were first reported in this geographical region [74]. All of the abovementioned novel species were among non-spore forming Gram-positive anaerobes and in one case of a 58-year-old male patient, *S. moorei* was the isolated pathogen. The improvements in the identification of these non-spore forming Gram-positive bacteria may be attributable to the developments in the MALDI databases, containing bacterial spectra [75]. In another report from Hungary, Sárvári et al. described five additional cases from the same region: all patients presented with underlying diseases (malignancy in 4 out of the 5 cases), and in most cases, *S. moorei* was isolated in mixed culture with other anaerobes of facultative anaerobes [31].

Zheng et al. described nine cases (out of 400) of surgical wound infections, when the complex aerobic-anaerobic bacterial culture involving the numerically dominant *S. moorei* flora played a principal role in the development of the infections [24]. They found *S. moorei* and *F. nucleatum* together in n = 2 of their cases, while *S. moorei* was found with two other *Fusobacterium* species (namely *F. equinum* and *F. gonidiaformans*) in one other case. They have hypothesized that *S. moorei* and *Fusobacterium* strains may act synergistically (i.e. pathogenic synergy), and/or each may supply growth factors for one another in *in vivo* conditions [24, 76]. Although the underlying mechanisms are not yet fully understood, there are several examples for this kind of advantageous interaction, e.g., between *A. viscous* and *P. gingivalis* (enhancing eachothers' growth and colonization levels in the oral cavity [77, 78]) and between *F. nucleatum* and *T. forsythia* (acting synergistically in protein degradation [79, 80]). A possible mechanism for *in vivo* synergism between *S. moorei* and fusobacteria may be found in the initial steps of VSC-production, where *S. moorei* is responsible for the initial deglycosylation step, which is required for *F. nucleatum* subsequently degrade proteins more effectively [81, 82].

5. Therapy of *S. moorei* infections, concluding remarks

In regards to the treatment of halitosis, the first step is the identification of the extent and the origin of the malodor [83]. If the halitosis is not pathological, adequate oral hygiene (including mechanical removal of tongue biofilm with tongue scraping or with a toothbrush) and abstaining from the consumption of odorous compounds will resolve the complaints [84]. Oral care may be supplemented by the use of products containing cetylpyridinium chloride, chlorhexidine, chloride dioxide, zinc salts or natural (plant-based) compounds capable of inhibiting bacterial growth or neutralizing VSCs [85]. If halitosis is diagnosed to be pathological, the management of the complaints will largely depend on the origin (IOH or EOH), the organ systems affected and/or existing underlying diseases of the patient [11].

S. moorei infections should be treated with antibiotic-therapy, taking into account the anaerobic respiration of the pathogen [41]. Thus, only agents with anti-anaerobic activity should be considered (penicillin, piperacillin-tazobactam, clindamycin, metronidazole, meropenem, moxifloxacin, tigecycline and vancomycin), in addition to the characteristics of the patient (e.g., kidney function, hypersensitivity) and the clinical presentation of the infection [86]. Based on the abovementioned reports available, clinical *S. moorei* isolates exhibit uniform susceptibility to common antibiotics used for the treatment anaerobic infections. In dentistry, amoxicillin/clavulanic acid, metronidazole and clindamycin are most frequently used: the former for its good tolerability, metronidazole for its potent anti-anaerobic activity (except for some Gram-positive anaerobes, such as (*Actinomyces* spp., *Bifidobacterium* spp., *Cutibacterium* spp. and *Lactobacillus* spp.)), while the latter is characterized by good penetration into abscesses [87]. For dental infections and wound infections, surgical debridement and drainage of pus is commonly required to supplement therapy and to oxygenize the affected tissues.

Solobacterium moorei is a recently described Gram-positive, non-spore-forming, obligate anaerobic bacillus; this pathogen is less well-known to clinicians as other members of Gram-positive rods. In addition, the literature concerning these bacteria is extremely scarce, which was demonstrated in this review. Regarding its pathogenic role, the relevance of *S. moorei* in halitosis has a good standing, as it has been suggested by multiple studies, while the isolation of these bacteria from invasive infections is very rare; there were only a few reports of infections outside the oral cavity. Nevertheless, all of abovementioned articles point to the fact that *S. moorei* might be more prevalent in bacteremia than believed so far, and the usefulness of MALDI-TOF MS and 16S RNA gene sequencing for the discovery and identification of this non-spore-forming strictly anaerobic Gram-positive bacillus (if these technologies are available) cannot be emphasized enough times.

The clinical data available from the published reports indicate that the patients to whom have compromised immunity (e.g., hematological malignancies or solid tumors, diabetes, intravenous drug use and a significant medical history previous surgical intervention) are more prone to *S. moorei* bacteremia across all genders and age groups. Sepsis and/

Table 2
Published cases of *S. moorei* bacteremia presented in a patient-by-patient basis.

Case number	Age/Sex	Infection	Underlying Diseases	Output	Treatment	Author/Country	Year
1.	67/M	Sepsis Dentoalveoral abscess	Multiple myeloma, autologous bone marrow graft	Improved and discharged	Cefepime No surgical	Detry et al., [69] Belgium	2006
2.	43/F	Acute proctitis	Carcinoma of the cervix stage III-B	Recovered	Piperacillin-tazobactam No surgical	Lau et al., [27] Hong-Kong	2006
3.	37/M	Septic pulmonary embolism femoral vein	Intravenous drug abuse	Recovered	penicillin and metronidazole No surgical	Martin et al. [70] UK	2007
4.	43/M	Fever, anemia, diarrhea, Tooth abscess	Lymphoma, kidney transplantation	Recovered	penicillin and metronidazole No surgical	Pedersen et al. [26] Denmark	2011
5.	66/F	Pulmonary abscess, Sepsis	Non-small-cell lung carcinoma with meningeal carcinomatosis	Not known	1. cefuroxime + gentamicin 2: meropenem + metronidazole + ciprofloxacin 3.metronidazole No surgical		
6.	64/M	Sepsis, fever	Complicated abdominal surgery, Colon cancer	Improved and discharged	Cefuroxime + metronidazole No surgical		
7.	33/F	Femoral vein thrombosis and abscess, fever, headache	Intravenous drug abuse, Chronic HBV infection	Recovered	1.Cefuroxime 2.Penicillin + metronidazole No surgical		
8.	77/M	Pneumonia, fever, dry cough, toothache	Prostate cancer Previous chronic heart disease	Recovered	Penicillin No surgical		
9.	56/M	General malaise	Fournier gangreana	Not known	Not known	Hernandez Blaco et al. [71] Spain	2017
10.	58/M	Sepsis	alcoholic polyneuropathy, encephalopathy, COPD, broken left femoral neck and neuromuscular dysfunction of the urinary bladder	Died	Did not accept any antibiotics, intensive or surgical therapy	Gajdács et al. [74] Hungary	2017
11.	61/M	Persistent coma, intermittent convulsions, halitosis, intermittent fever	TTC, hypertension, hyperlipidemia, type 2 diabetes, rectal cancer, brucellosis	Died	Vancomycin + meropenem No surgical	Liu et al. [72] China	2019
12.	70/M	Pneumonia	HSV-1 oesophagitis, HIV infection Diabetes	Not known	Amoxicillin/clavulanic acid	Genderini et al. [73] Belgium	2019

M: male; F: female; TTC: thrombotic thrombocytopenic purpura; HBV: hepatitis B virus; HIV: human immunodeficiency virus; HSV-1: herpes simplex virus-1.

or bacteremia caused by *S. moorei* may originate from different oral infections, lung abscess, abdominal infections, and from the lesser-known habit of licking needles by intravenous drug users. During clinical assessment of patients with blood cultures positive for *S. moorei*, it is recommended to first search for dental focus of infection. Based on the currently available reports, a case of possible pathogenic synergy between *S. moorei* and *Fusobacterium* spp. has been suggested (as they have been commonly co-isolated), however, the exact mechanism is poorly understood. Overall, it can be concluded that our present knowledge about *S. moorei* is scarce, and intense research associated to this anaerobe is warranted to further characterize its role in the pathogenesis of human infections, including halitosis and invasive processes.

Declarations

Author contribution statement

All authors listed have significantly contributed to the development and the writing of this article.

Funding statement

This work was supported by the University of Szeged Open AccessFund (Grant number: 4837). M.G. was supported by the János Bolyai Research Scholarship (BO/00144/20/5) of the Hungarian Academy of Sciences and the New National Excellence Programme (ÚNKP-20-5-SZTE-330) of the Ministry of Human Resources. Support from Ministry of Human Capacities, Hungary grant 20391-3/2018/FEKUSTRAT is acknowledged. M.G. would also like to acknowledge the support of ESCMID's "30 under 30" Award.

Competing interest statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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