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An autopsy report of rare purulent pericarditis caused by *Streptococcus anginosus* infection that spread from the liver abscess

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ABSTRACT

Streptococcus anginosus group (SAG) is Gram-positive streptococci and a commensal in the upper airway and gastrointestinal and conceptive swathe. It is a commensal microorganism and not well recognized as a pathogen that causes the disease; moreover, death due to the infection is also rare. However, it can cause invasive infections and induce purulent infection with abscess formation under certain conditions and risk factors. We report a rare case of purulent pericarditis in a male deceased who presented with sudden death. The underlying purulent pericarditis that spread from the liver abscess was identified through autopsy examination. Local spread from adjacent organs is rare. The culprit organism was a commensal that became a pathogen under a certain environment. SAG pyogenic pericarditis is an infrequent but severe and fatal condition. A holistic approach to the case must emphasize autopsy findings in a case where the history is limited, especially in forensic practice. Two crucial processes are required to determine the cause of death: through a thorough autopsy and by understanding causation. To conclude that a death was caused by a commensal pathogen is the most difficult task in forensic pathology, and careful examination of postmortem investigations is essential.

INTRODUCTION

Streptococcus anginosus group (SAG) is classified under the category of Gram-positive streptococci, common commensals in the upper airway, gastrointestinal, and progenitive pathways consisting of three well-defined species: *S. intermedius, S. constellatus*, and *S. anginosus* (Fazili *et al*., 2017). As a commensal/microbiota, this microorganism cannot cause any pathogenic infection in an immunocompetent individual. Nevertheless, when certain risk factors exist, the colonized SAG can induce noninvasive infections independently. On the other hand, it can cause quick and aggressive infections in a sterile environment by entering through the bloodstream and serosal cavities, ultimately causing infection in the tissues and organs of different systems (Jiang *et al*., 2020). Additionally, it also tends to cause pyogenic infections with abscess formation. SAG was formerly thought to cause oral cavity infections, but subsequent case reports have revealed that it can also cause infections of other body parts, including the skin, soft tissue, lungs, and liver. The fact that these opportunistic pathogens could cause invasive infections is not well known. This case report highlights the challenge of interpreting microbiological analysis in forensic practice, mainly when commensal organisms were grown in the culture.

CASE REPORT

In August 2021, a 41-year-old male was brought in dead to the forensic department by the police. A legal order to perform an autopsy examination was issued (Pol 61) by the police to ascertain the cause of death. As a routine practice, history was obtained from the next of kin, and a complete

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autopsy was performed by the forensic pathologist at 12 hours postmortem interval (PMI) to determine the cause of death of the deceased. The PMI was established from rectal temperature and was consistent with the time of death pronounced by a paramedic at the scene.

Autopsy findings

At autopsy, the body was that of a customarily built male with a Body Mass Index (BMI) of 20.4. Pallor was seen on the lower palpebrae conjunctiva and finger nailbeds. Sclera showed a tinge of jaundice. The external autopsy examination revealed that no fatal injuries were found on the body. The internal autopsy examination showed no pathology changes in the head and neck regions. In the thoracic region, no rib fracture was seen. Both pleura cavities were intact and clear. There was dense adhesion between the posterior surface of the upper portion of the right-sided pulmonary tree to the right parietal pleura. Both lungs were congested and edematous upon cut section. No consolidation was felt. The pericardium contained 250 ml of purulent fluid with strands of stringy pale fibrin between the visceral and parietal pericardium (Fig. 1). The heart weighed 230 g and appeared dusky and flabby. The coronaries were patent and typically arose from the aorta. All the valves showed no calcifications or vegetation. The myocardium was healthy, and no area of fibrosis or hemorrhage.

The peritoneum was filled with 300 ml of straw-colored fluid. There was hepatomegaly (1,872 g) with multiple variable sizes of liver abscesses. The largest was 7 × 6 × 3 cm, and the smallest was 3 × 2 × 2 cm (Fig. 2). A stent was found in the pancreatic duct (Fig. 3). Pancreatic tissue showed no evidence of hemorrhage or necrosis grossly. Both kidneys were unremarkable, and the remaining abdominal organs appeared normal.

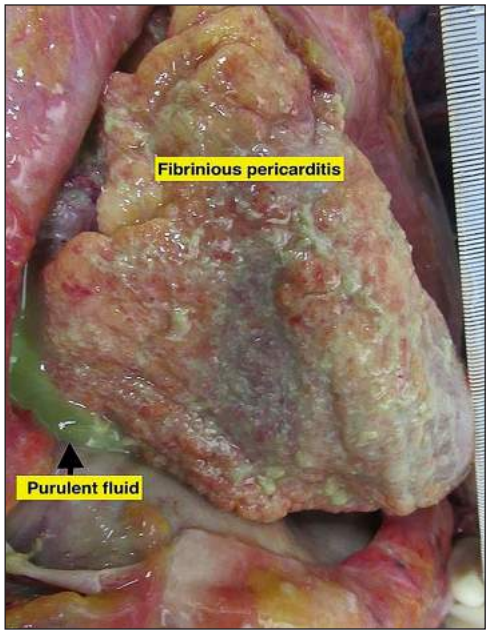

Figure 1. The pericardium contained 250 ml of purulent fluid with strands of stringy pale fibrin between the visceral and parietal pericardium.

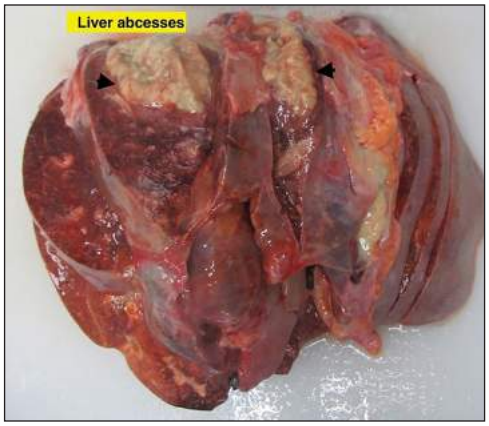

Figure 2. The liver showed hepatomegaly (1,872 g) with multiple variable-sized liver abscesses. The largest was 7 × 6 × 3 cm, and the smallest was 3 × 2 × 2 cm.

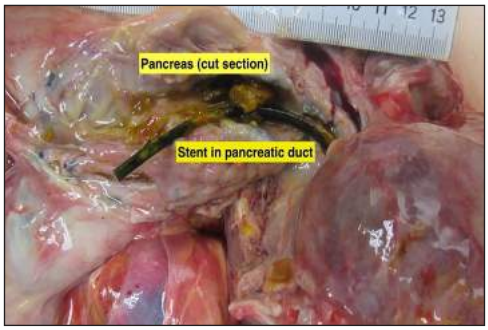

Figure 3. The pancreatic duct is the presence of a stent in the lumen.

Laboratory investigations

The microbiology specimens were taken by an aseptic technique to minimize contamination. The heart and liver tissue cultures revealed *S. anginosus*, whilst pericardial and peritoneal fluids showed mixed growth of *S. anginosus* and other microorganisms. On the other hand, the blood culture and sensitivity grew *Bacillus* species (aerobic) and *Staphylococcus hominis* (anaerobic). The biochemistry and toxicology analyses were all within the normal range.

Histology findings

Pathologic tissues and representative organs, including skin, were taken for microscopic examination. The liver tissue showed an area of necrotic hepatocytes with abundant inflammatory infiltrates, predominantly neutrophils, lymphocytes, plasma cells, and Kupffer cells. The portal triad showed increased lymphocytic infiltrates and bile duct hyperplasia. The adjacent intact liver tissue shows marked sinusoidal congestion and no evidence of parasitic infiltration or malignancy. The pericardium and heart tissues showed generalized infiltrations, predominantly neutrophils at the superficial epicardial layers, with proof of mesothelial cell metaplasia. The myocardium and endocardium showed no ischemia or myocarditis changes. The lungs section showed multifocal infiltration by neutrophils, lymphocytes, and plasma cells within the edematous alveoli and no evidence of

microabscess or granulomatous lesion. Pancreatic tissue with a stent shows foci of lymphocytic infiltrates in the background of fibrosis, and patchy lymphocytic infiltrates are seen at the peripancreatic fat area. No evidence of fat necrosis and neutrophilic infiltrates in the area adjacent to the stent. The skin, kidneys, spleen, adrenal, and brain were congested.

DISCUSSION

It is a routine forensics practice that history was taken from the next of kin. The history obtained was often incomplete since the next of kin and the deceased did not live together. Due to this limitation, the history obtained is occasionally unreliable. This could lead to the wrong approach to managing the autopsy and incorrectly handling the necessary ancillary tests. However, this problem can be tackled by performing a complete autopsy. The burden of responsibilities lies on a forensic pathologist to conscientiously analyze the case before reaching a reasonable conclusion on the cause of death.

The clinical significance of these SAG strains in causing pulmonary infection and empyema has only recently been identified. According to previous literature, SAG strains can enter the thoracic cavity through aspiration of oral discharge, direct impregnation following injury or surgery, and spread from adjacent bordering focus points of infection such as subphrenic abscesses and hematogenic propagation (Hocken and Dussek, 1985; Okada *et al*., 2013).

The case reports of *S. anginosus* pericarditis are limited; moreover, this is an autopsy-based diagnosis describing a documented infection with purulent pericarditis furnishing manifold liver abscesses. In this case, pericarditis was caused by local spread from the nearby liver abscess. It has been postulated that SAG commensal gains access to the liver via the earlier stenting procedure in the pancreas. This was supported by the microscopic examination of the adjacent pancreatic tissue that showed chronic inflammatory changes, indicating that the tissue response to infection occurred for quite some time. The age of the tissue response is further confirmed by retrospective inquiry to the next of kin regarding the date of previous surgery. Since the liver abscess was located adjacent to the heart, this commensal may have reached the pericardial sac by direct transmission from the liver abscess. Nowadays, purulent pericarditis has grown scarce.

After everything, it is a crucial impediment to lung infections such as pneumonia. In this case, the abdomen and chest were principally taken in, and most infections originated from *S. anginosus*, which accords with formerly published research reports (Claridge *et al*., 2001; Junckerstorff *et al*., 2014). It is interesting to understand the immune response of the deceased. In this case report, although the autopsy findings revealed significant pathologic changes in the affected organs, the history elicited from the next of kin failed to address any illness symptoms on the deceased. Age, cytokines, and their antagonists play substantial factors in host immune responses to infection and inflammatory stimuli. Catania *et al*. (1997) described that in elderly patients, the plasma concentration of the interleukin-1 (IL-1) receptor antagonist and soluble tumor necrosis factor-alpha receptor were high; therefore, febrile reactions toward underlying infection are inhibited (Cartmell *et al*., 2001; Catania *et al*., 1997; Stefferl *et al*., 1996). Older patients with pneumonia present with considerably fewer symptoms than younger patients. El-Solh *et al*. (2001) reported that 44% of very old patients with severe pneumonia presented with fever (Li *et al*., 2015).

The analysis of microbiology tissue culture in forensic practices is challenging. The presence of the microorganism in the autopsy tissue culture does not necessarily indicate genuine infections. To conclude that death is due to infections, it must incorporate the gross pathological changes of the organs, supported by vital reactions in the microscopic examination and the presence of the pathogen in the tissue culture. The presence of the microorganism after death can be regarded as normal postmortem changes. The earlier studies stated that two salient theories explain bacterial magnification in postmortem blood and tissue cultures either via (i) agonal spread or (ii) postmortem bacterial transmigration. In the former, it is best understood that the bacteria can invade the bloodstream after death during the agonal period because the systemic circulation is de-escalating or artificially sustained during resuscitation. In 1916, this concept was first introduced and was later supported by other researchers (Fredette, 1916; Koneman and Davis, 1974; Riedel, 2014). On the contrary, this concept was argued by other researchers in 1975. They disagreed and published their findings on bacteremia and postmortem microbiology in children suffering from burn wounds (Smith, 1975).

On the other hand, in the latter, the action by which the microorganism migrates from mucosal surfaces and tissues into the blood flow after circulation has halted is used to describe the term by multiple studies "postmortem bacterial transmigration" (Morris *et al*., 2007; Riedel 2014; Saegeman *et al*., 2009). The process has first described this process in 1904 (Gradwohl, 1904) and was subsequently supported by findings in other studies (Carpenter and Wilkins, 1964; Epstein and Kugel, 1929; Rose and Hockett, 1971). A few studies revealed bacterial transmigration in animal studies and *in vitro* experiments combined with the verification provided by observing the autopsy series (Burn, 1934; Kellerman *et al*., 1976). Although only a few historical data exist in support of the concept of agonal spread is merely theoretical, the theory of bacterial transmigration has far more evidence in the most scientific and medical literature (Tuomisto *et al*., 2013). Kellerman and his colleagues reported that bacteria could migrate through the intact human intestinal wall within 12–15 hours after death based on *in vitro* experiments (Kellerman *et al*., 1976).

Even though death has been categorized as natural death due to infection, conscientious interpretation of microbiological analysis in autopsy is vital. This case report highlights the infection caused by a commensal and this autopsy-based diagnosis may benefit the clinician. Ignoring the potential of a commensal may lead to deteriorating illness and even death in a patient.

CONCLUSION

It has been reported that SAG possesses the potential to generate pyogenic infections irrespective of age because of its opportunistic pathogenic quality. The capability of distinguishing between true-positive culture findings and postmortem transmigration and/or contaminant exists in considerable defiance to microbiologists and pathologists. This case exemplifies the importance of a careful approach in dealing with the commensal pathogen. An understanding of postmortem microbiology interpretation is crucial to analyze a case comprehensively. Finding a commensal organism in a postmortem sample is not unusual,

and the correlation with gross and histology findings is critical for determining the cause of death with certainty. Though history in forensic practice is just "hearsay," it should not be excluded and must be illicit retrospectively.

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AUTHOR CONTRIBUTIONS

NAR is the first author that prepared this article and assisted in the autopsy. SAMS, a forensic pathologist, led the autopsy. The corresponding author MH and author RA supervised this preparation of articles.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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INFORMED CONSENT

The anonymity of the subject and confidentiality were well preserved. Written informed consent was obtained from the next of kin of the deceased for the publication of this case report and accompanying images. The next of kin were informed regarding the findings after the postmortem and the usage of the tissues for this case report remains anonymous.

DATA AVAILABILITY

All data generated and analyzed are included in this research article.

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