Heart Science and Heart Surgery Journal

Chembio Publishers
Right Choice for Researchers

Case Report *Volume 2; Issue 1*

A Rare Case of Delayed Presentation of Sternal Wound Infection 5 Years Post Midline Sternotomy for Aortic Valve Replacement

Kasthuri Shamugam*, Mat Saad AZ and Halim AS

Department of Reconstructive Sciences, Universiti Sains, Malaysia

***Corresponding author:** Dr. Kasthuri Shamugam, Department Of Reconstructive Sciences, Universiti Sains Malaysia, School of Medical Sciences, Kubang Kerian, Kelantan 16150, Malaysia, Tel no: 60124720027; Email: kas_kasthuri@hotmail.com

Received Date: December 20, 2018; **Published Date:** January 11, 2019

Abstract

Delayed sternal wound infection is the most feared complication of a midline sternotomy. The incidence of sternal wound infection reported to be 1.5% and the longest delayed sternal wound infection was reported to be 417 days postoperatively [1,2]. This is a case of a patient with a comorbid of hypertension and chronic rheumatic heart disease, who presented with delayed sternal wound infection 5 years' post midline sternotomy for aortic valve replacement. He is also a chain smoker, whom continued to smoke perioperatively till the current admission. There are many factors contributing to delayed sternal wound infection. However, in this patient there are few causes attributing to it such as smoking, Staphylococcus infection and biofilm. Although smoking and Staphylococcus infection has been extensively studied, the presence of biofilm as a cause for delayed sternal wound infection is still in progress.

Keywords: Delayed sternal infection; Biofilm; Staphylococcus; Smoking

Abbreviations: CT: Chest Computed Tomography; TB: Tuberculosis; HIV: Human Immunodeficiency Virus.

Introduction

Sternal wound infection and the subsequently sternal wound dehiscence is one of the most dreaded complications of midline sternotomy. The incidence of sternal wound infection is reported to be 1.5% and the longest delayed sternal wound infection was reported to be 417 days post operatively [1,2]. There are many factors involved in the delayed presentation of sternal wound infection which could be attributed to the patient factor as well as the surgical procedure itself. Patient factor can be divided into conditions such as obesity, history of radiation, and other comorbidities which inhibit wound healing such as diabetes, smoking, steroid use, and preoperative and postoperative malnutrition all tend to increase the risk factors for infection. Long hour surgery

Citation: Kasthuri Shamugam, et al. A Rare Case of Delayed Presentation of Sternal Wound Infection 5 Years Post Midline Sternotomy for Aortic Valve Replacement. Heart Sci Heart Surg J 2019, 2(1): 180006.

as well as breach in sterility can also be associated with sternal wound infection [3]. Cardiac surgeries using bilateral internal mammary graft increases the risk of sternal wound infection in 30% of cases and the reexploration of the chest due to postoperative complication nearly doubles the occurance of sternal infection to 68%. Surgical procedure which includes the use of internal thoracic artery for CABG, long hour of surgery, breach in sterility can all contribute to sternal wound infection [4,5]. With the presence of all the risk factors mentioned as above, it raises the risk of infection. Most common microorganism cultured in the delayed sternal wound infections are Staphylococcus [6]. The theory of low dose inoculum of Staphylococcus is mentioned to be a reason of delayed sternal infection [2]. Sometimes sternal wire as a foreign body can be covered with biofilm which can also be a source of infection [7]. Here I present a rare case of delayed sternal wound infection which occurred 5 year's post midline sternotomy for aortic valve regurgitation secondary to chronic rheumatic heart disease.

Case Presentation

A 53 years old man was referred to the plastic team for sternal wound dehiscence post wound debridement and primary closure for deep sternal wound infection post aortic valve replacement which was done 5 years earlier. He has underlying hypertension as well as aortic valve regurgitation secondary to chronic rheumatic heart disease where he underwent aortic valve replacement via midline sternotomy in 2010 at Hospital Universiti Sains Malaysia. His surgery was uneventful and was discharged home well with regular follow up till he presented to the Emergency Department 5 years later complaining of fever, sternal scar redness associated with pain and sinus discharge over the inferior sternal scar. He denied chronic cough, trauma or recently being ill.

He is an active smoker. He was febrile only upon the first presentation with no significant raised infective parameters and was treated symptomatically with Amoxicillin Clavulanate and dressing over the wound. In view of delayed wound healing progression, he underwent wound debridement and 2 sternal wire removals with primary closure of the wound which complicated again with sinus discharge. His infective screening for TB, HIV, and Hepatitis were all negative and all his cultures taken from the wound was negative for microorganisms. CT Thorax which was done showed no signs of osteomyelitis (Figure 1).

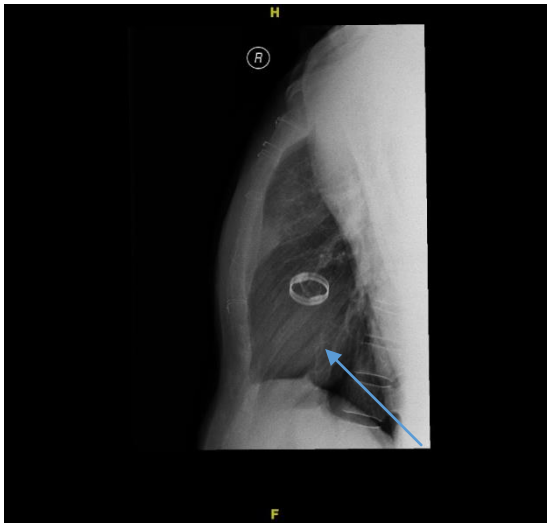


Figure 1: X-ray post-sternal wire removal and arrow indicating the remaining sternal wire beneath the wound dehiscence.

He was subjected for another wound debridement with primary closure which was complicated by complete wound dehiscence after 14 days during the removal of the sutures. His intraoperative Tissue C & S grew Staphylococcus aureus and antibacterial treatment was initiated as per sensitivity. He was then subsequently referred to the plastic surgery team for wound management and reconstruction. Upon assessment of the wound, the wound extended from the sternoclavicular junction up till xiphoid process exposing the sternum with intact perichondrium, the sternum was stable and well healed (Figure 2).

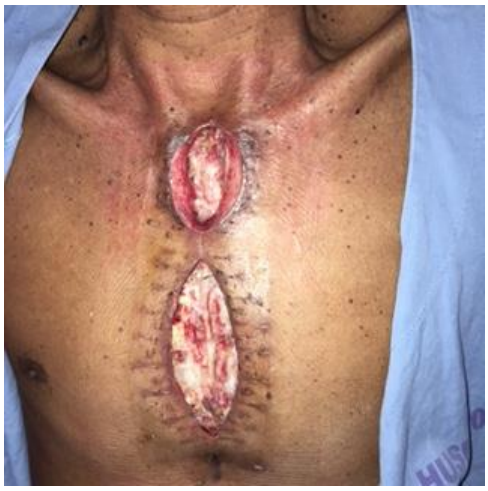


Figure 2: Pre-operative image during wound assessment.

He underwent bilateral pectoralis major advancement flap 11 months from the initial presentation. Pre-operatively the surface marking of the bilateral thoracoacromial pedicle (a line connecting the acromian process and the xiphoid, the midpoint of the clavicle marks the pedicle) is marked. Intraoperatively, via the dehisced sternotomy wound, the muscle is elevated from medial to lateral sparing the pectoralis minor muscle. Both muscle's insertions on the rectus abdominis and external oblique inferiorly are divided. The left pectoralis major's additional mobility was achieved by releasing the muscle insertion to the humerus. Then, bilateral pectoralis major muscle was approximated and the wound closed in layers without tension. Patient was then nursed in the cardiac unit and was discharged well 5 days post operation. Post operatively upon regular follow-up, he is doing well with no significant complain of pain, difficulties in breathing or mobility of his shoulders. He will be under our follow- up till he is deemed fit for discharge (Figure 3&4).

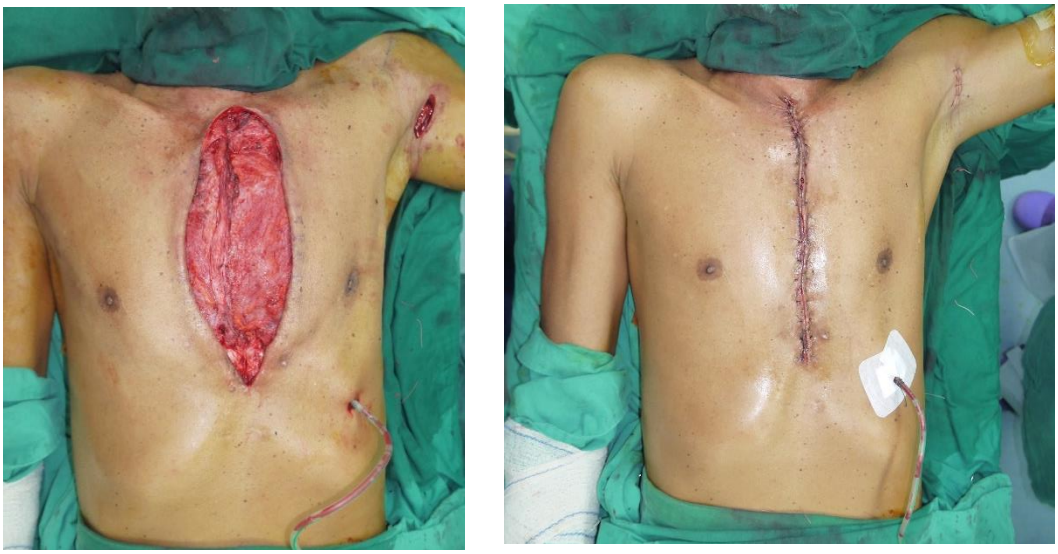
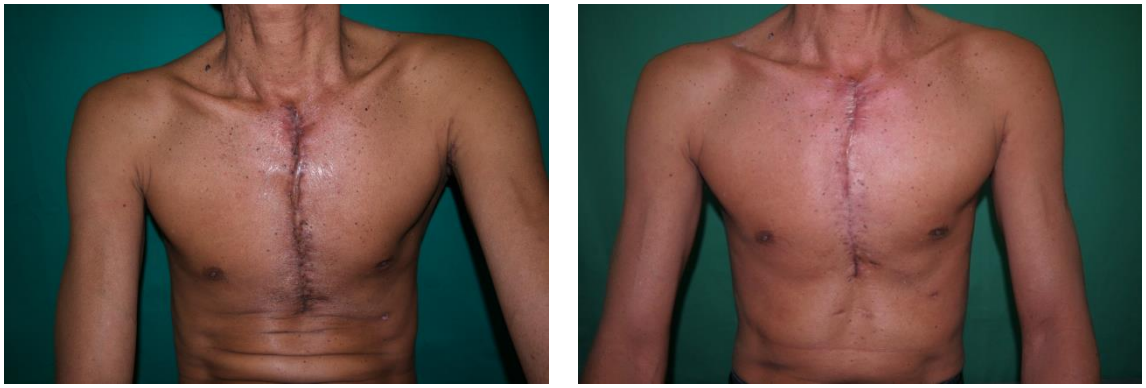


Figure 3: Intra-operative image of post-bilateral pectoralis major advancement flap.



A B

Figure 4: (A) 1Month post-surgery (B) 3Months post-surgery.

Discussion

Midline sternotomy was initially described by Milton in 1887 and was re- introduced by Julian in 1957, and it is now a gold standard approach for cardiac surgeries [7]. Since the introduction of this method, many refinements have been made to reduce the complication arising from the midline sternotomy. Sternal wound dehiscence is one

of a rare complication of midline sternotomy which is associated with high morbidity and mortality. It can be due to infection or sterile wound dehiscence [5]. Most sternal wound dehiscence is secondary to infection. In a systemic review by Balachandran et al, female gender, obesity, smoking, diabetes mellitus, bilateral internal mammary artery graft, reoperation for postoperative complication and blood product requirement are the risk factors which contributes to sternal infection. Female is found to have higher incidence compared to men in up to 30% especially women with larger breasts whom are subjected to increased inferolateral tension across their median sternotomy.

This also explains obesity as a risk factor due to the increased tension on the midline sternotomy wound which subsequently causes the sternum instability and predisposes for infection [4]. Hyperglycemia associated with diabetes mellitus has a deleterious effect on the wound healing as well as impaired immune system. On the other hand, the use of bilateral IMA grafts significantly increases the risk of sternal infection to 35%. Given the IMA is a major blood supply to the sternum, some studies report that it may lead to sternal hypoperfusion and increase the risk of sternal infection. However, the use of skeletonized grafts preserves collateral blood supply and optimizes sternal healing [8]. Exploration due to unavoidable post cardiac surgeries predisposes the mediastinum to airborne, environmental pathogens hence increasing sternal infection [4].

These factors could interfere in any of the 4 stages of wound healing which is hemostasis, inflammation, proliferation and remodeling which leads to impairment of the healing process. One of the leading factors which can be amended by patient and reduce the morbidity of wound healing is smoking. This patient was a chain smoker with difficulties in giving up his habit. Smoking has a detrimental influence on wound healing. The bad effects of smoking and wound healing were first reported in 1977 by Mosley and Finest, who observed impaired healing of a wound in a smoker with arteriosclerosis [9]. There are over 4000 substance in cigarette, however particularly nicotine, carbon monoxide and hydrogen cyanide suggest potential mechanism where is undermines the expected wound repair.

The presence of nicotine in the cigarette can causes vasoconstriction which leads to local ischemia, increase platelet adhesiveness which can leads to thrombosis and further enhances the ischemia. Presence of carbon monoxide with the high affinity towards oxygen which causes significant nonfunctional hemoglobin leads to reduced oxygenation to the wound which causes reduction is oxygen radical particle which subsequently makes the wound susceptible for wound infection [10]. Tippava Nagachinta et al. noted that current cigarette smoking is one of the risk factors for surgical wound infection following cardiac surgery [3]. Sorensen et al in his paper stated that subjects who continued to smoke during wound healing, are associated with increased wound infection. Tobacco is associated with weaker scar which is associated with recurrent injury [11,12]. In our patient, failure of smoking cessation may have led to increased susceptibility for wound infection. Common microorganisms cultured from an infected sternal wound include the common skin flora streptococcus, staphylococcus and diptheroids [6]. Sometimes gram negative organisms such as pseudomonas aeruginosa are cultures in at risk patients such as diabetics, those with prolonged hospitalization or with extensive devitalized tissue (burn) [13].

However, as a clinician it's important to be able to distinguish whether the cultured bacteria are contaminant, colonizer or infection and a negative culture doesn't rule out infection. This patient's tissue culture and sensitivity intraoperatively grew Staphylococcus Aureus. It could the patients endogenous skin flora as it's the skin resident flora or a contaminant from contaminated surgical instrument or surgical material. However, it was reported that theory of low inoculum is associated with the development of late mediastinitis. The theory suggests that the low inoculum of Staphylococcus is present during surgery, which may remain dormant for many years [2]. Elgharably et al. in his study on presence of biofilm in patients with deep sternal wound infection found three-dimensional staphylococci aggregates attached to the sternal wires and the infection was localized to the sternotomy wound and with negative blood cultures.

He also mentioned that the failure of broad spectrum antibiotics in curbing the biofilm related infection [14]. This could be related to progression of wound infection from the initial delayed wound presentation despite on broad spectrum antibiotics. Biofilm can't be routinely picked up during standard culture method. A more specific method such as confocal microscopy and PCR (polymerase chain reaction) test can be used to determine the three-dimensional aggregates of biofilm. Staphylococcus is known to form robust biofilms and is frequently responsible for biofilm- associated medical devices infection. Biofilm infected – wounds are difficult to be treated medically and usually require surgical interventions. To our knowledge this is the first case to describe a possible biofilm on the sternal wire. Over the years there are many classifications which were created to classify sternal wound infection to ease on

management as well as exchange of data among surgeons. Pairelero & Arnold was the 1st to classify based on the time of onset of sternal wound infection post midline sternotomy in 1984 and Oakley in 1996 subsequently added the risk factors and the attempts of treatment of the initial infection [15].

Jones et al in 1997 was the first to specify sternal infection according the anatomical site with infective parameters and proposed a treatment algorithm based on the 3 groups [15]. Type 1 infections can be treated by incision, drainage and healing by secondary intention. Type 2A infections can be treated by soft tissue debridement and closure in the absence of residual infection. In type 2B wounds, the sternum can be rewired if the wound is sterile; however, if there is any concern that the sternum cannot withstand rewiring, sternal debridement and flap closure are indicated. Type 3 wounds require urgent debridement and either primary or delayed closure. Whatever the classification the patients fall in, individualized treatment must be advocated based on findings and the principles of chest wall reconstruction (Table 1).

Classification	Depth	Description
Type 1A	Superficial	Skin and subcutaneous
Type 1B	Superficial	Exposure of sutured deep fascia
Type 2A	Deep	Bone exposed, sternum with stable steel suture
Type 2B	Deep	Bone exposed, sternum with unstable steel suture
Type 3A	Deep	Necrotic bone exposure or fractured, unstable sternum, exposed heart
Type 3B	Deep	Type 2 or 3 with septicemia

Table 1: Classification proposed by Jones in 1997 based on anatomical site and including sepsis [15].

Chest wall reconstruction principles are of eradication of infection, local wound care, extensive debridement of devitalized soft tissue, cartilage, and bone; obliteration of all residual spaces with well vascularized flaps, re-establishment of skeletal stability if needed and early definitive flap coverage of defects [15]. Based on the evaluation of the wound in regards to form and function, the choice of bilateral pectoralis advancement flap was made. Pectoralis advancement flap is consistent with Mathes and Nahai type V muscle flap. The arc of rotation of pectoralis major based on the thorocoacromial axis allows the flap to be used in coverage of central chest wounds, supraclavicular defects, and axillary and lateral chest wall defects. The advantage of this technique allows for repeat procedures to be performed through the midline later, for instance repeat cardiothoracic procedures without sacrificing the viability of the reconstruction. The disadvantage of this technique is that, most often than not, the vertical central chest wall defect extends inferior to xiphoid process and into epigastrium and it may be out of reach for pectoralis flap [16].

Conclusion

Delayed sternal wound infection is rare which is related to high mortality rate. A presence of low inoculum of Staphylococcus during surgery coupled with concurrent smoking could have led to delayed sternal wound infection. Sternal wire biofilm as a cause of delayed sternal wound infection needs to be further studied upon as this could change the future management of the patient.

References

1. Bor DH, Rose RM, Modlin JF, Weintraub R, Friedland GH (1983) Mediastinitis after cardiovascular surgery. Reviews of Infectious Diseases 5(5): 885-897.

2. Shagufta Ahsan (2015) Clinical Microbiology & Case Reports A Delayed Case of Sternal Wound Infection after Coronary Artery Bypass Graft Surgery. Clin Microbial Case Rep 1(3): 1-7.

3. Nagachinta T, Stephens M, Reitz B, Polk BF (1987) Risk Factors for Surgical-Wound Infection following Cardiac Surgery. J Infect Dis 156(6): 967-973.

4. Balachandran S, Lee A, Denehy L, Lin KY, Royse A, et al. (2016) Risk Factors for Sternal Complications After Cardiac Operations: A Systematic Review. Ann Thorac Surg 102(6): 2109-2117.

5. Fu RH, Weinstein AL, Chang MM, Argenziano M, Ascherman JA, et al. (2016) Risk factors of infected sternal wounds versus sterile wound dehiscence. J Surg Res 200(1): 400-407.

6. El Oakley RM, Wright JE (1996) Postoperative mediastinitis: Classification and management. Ann Thorac Surg 61(3): 1030-1036.

7. Rupprecht L, Schmid C (2013) Deep sternal wound complications: an overview of old and new therapeutic options. Open J Cardiovasc Surg 6: 9-19.

8. Cheng K, Rehman SM, Taggart DP (2015) A review of differing techniques of mammary artery harvesting on sternal perfusion: Time for a randomized study? Ann Thorac Surg 100(5): 1942-1953.

9. Mosely LH, Finseth F, Goody M (1978) Nicotine and its effect on wound healing. Plast Reconstr Surg 61(4): 570-575.

10. Broughton G, Janis JE, Attinger CE (2006) The basic science of wound healing. Plast Reconstr Surg 117(7suppl): 12S-34S

11. Anderson K, Hamm RL (2012) Factors that impair wound healing. J Am Coll Clin Wound Spec 4(4): 84-91.

12. HUNT A H (1960) Wound Healing. Proc R Soc Med 53: 41-48.

13. Okonta KE, Mohanraj A, Agarwal V, Jamesraj J, Mathew VK, et al. (2011) Sternal wound infection following open heart surgery: appraisal of incidence, risk factors, changing bacteriologic pattern and treatment outcome. Indian J Thorac Cardiovasc Surg 27: 28-32.

14. Elgharably H, Mann E, Awad H, Ganesh K, Ghatak PD, et al. (2013) First Evidence of Sternal Wound Biofilm following Cardiac Surgery. PLoS One 8(8): e70360.

15. Anger J, Dantas DC, Arnoni RT, Farsky PS (2016) A new classification of post-sternotomy dehiscence. Rev Bras Cir Cardiovasc 30(1): 114-118.

16. Bakri K, Mardini S, Evans K, Carlsen B, Arnold P (2011) Workhorse Flaps in Chest Wall Reconstruction: The Pectoralis Major, Latissimus Dorsi, and Rectus Abdominis Flaps. Semin Plast Surg 25(1): 43-54.