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Perioperative Occupational Exposure to *Coxiella burnetii*-Infected Thoracic Endovascular Aneurysm Stent Graft

Adebisi Idowu Obafemi¹, Jade Le²

Abstract

We conducted this study to determine the risk of transmission of Q fever to health care workers (HCWs) during perioperative exposure to *Coxiella burnetii*-infected thoracic endovascular aneurysm stent graft. Pre-operative and 6-week post-operative phase I and II IgG Q fever antibody titers were determined in 14 staff members of an operation room. The room had a negative pressure and all the members of the surgical team wore either a fitted N-95 mask or a powered purified air respirator. Phase I and II IgG antibody titers were <1:16 for 11 of the 14 studied HCWs; 2 HCWs did not follow up at 6 weeks and 1 had a pre-exposure phase II IgG titer of 1:128 with no change 6 weeks later. We concluded that risk of transmission of *C. burnetii* in the operating room from infected patient to HCWs who wore appropriate personal protective equipment is low.

Keywords: *Coxiella burnetii*; Q fever; Infection control; Health personnel; Personal protective equipment; Operating rooms; Stents

Introduction

C*oxiella burnetii* is the causative agent of Q fever, a zoonotic infection distributed worldwide that may be found among abattoir workers and people who work with animals such as sheep and goat, either on the farm or in the laboratory.¹ *C. burnetii* is currently classified as a Proteobacteria with different antigenic phase variations. It expresses a phase I and phase II antigens which form the basis for the differentiation of acute from chronic Q fever infection. Acute or recent infection can be diagnosed with a four-fold rise in anti-phase II IgG by immunofluorescence assay (IFA) between serum samples from the acute and convalescent phases taken

3–6 weeks apart. A single anti-phase II IgG IFA titer >200 (or >50 for IgM) can also be used to diagnose recent infection. PCR can also be used in suspected cases, if the initial serology reveals no or low levels of antibodies.²

Most human infections are from occupational exposures, through inhalation of *C. burnetii* from dried feces, urine or birth products of infected animals.³ Infection can also occur through transdermal inoculation, blood transfusion,^{4,5} and consumption of infected raw milk⁶ or through a contaminated air conditioning system⁷. Human to human transmission is rare. A case report described transmission of *C. burnetii* during sexual intercourse in a woman whose husband acquired Q fever

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occupationally.⁸ Nosocomial human to human transmission has been described, but there are unconfirmed case reports.⁹⁻¹¹

There are still lingering questions about Q fever² including the possibility of nosocomial transmission¹⁵. Recently, a group of surgeons and anesthesiologists in our center expressed concern about the possibility of Q fever transmission during a planned explantation of a *C. burnetii*-infected thoracic endovascular aneurysm stent graft from a patient with history of thoracic abdominal aortic aneurysm, who subsequently developed aorto-enteric fistula and mycotic thoracic aneurysm. The explantation was done under left heart bypass and replacement of the ascending aorta and transection of the esophagus at the level of the aortic arch.

This case series was designed to explore the risk of transmission of *C. burnetii* from a patient with chronic Q fever to a group of surgeons and support crew in an operating room, while taking all the necessary precautions including using appropriate personal protective equipment (PPE).

Materials and Methods

We had group and individual meetings about occupational transmission of Q fever with members of the surgical team, as well as the pitfall of prophylactic treatment with doxycycline prior to exposure or onset of symptoms. Pre- and post-surgery blood samples were drawn for phase I and phase II IgG Q fever antibody titer six weeks apart to investigate the risk of acute Q fever transmission intra-operatively. None of the 14 staff members studied had valvular heart disease, or prosthetic device or graft in their body.

A negative pressured operating room and a fit tested N-95 respirator as well as an eye protector were used by the staff members. Powered purified air respirator was used by those who were unable to

wear N-95 mask. Disposable boot covers were provided as part of the PPE. Proper containment and disposal of contaminated waste to avoid aerosolization were observed. Specimens were collected in a closed container with wet gauze and labeled as "Caution." The operating room was cleansed using Xenex ultraviolet light-C machine and the equipment was sterilized and also autoclaved for 30 minutes post-procedure.

Results

There were 14 staff members of the surgical team, including cardiothoracic surgeons, anesthesiologists, and the nursing team. Sixth week post-exposure serology results were not available for two staffs who failed to show up for blood draw at the specified time, despite all the efforts made to contact them. However, their pre-operative phase I and phase II IgG antibody titers were <1:16. One of the staff had a baseline phase II IgG titer of 1:128, but there was no rise in the titer with the subsequent serology test.

The remaining 11 staff members of the surgical team had pre- and post-exposure phase I and phase II IgG serology that were <1:16. None of the staff members, including the one with the titer of 1:128, complained of any symptoms suggestive of Q fever or any febrile illness during the course of the investigation.

The patient had a Q fever IgG phase I antibody titer of 1:16 384, IgG phase II antibody titer of 1:2048, and phase I and phase II IgM titers of <1:16—consistent with chronic Q fever infection. The patient aortic wall showed positive molecular and immunohistochemical evidence of *C. burnetii*.

Discussion

To the best of our knowledge, this is the

first study to describe the pre- and post-exposure Q fever serology titers of staff members of a surgical team in order to determine whether transmission of Q fever infection occurs in an operating room after taking all the necessary infection prevention precautions.

The patient had a mycotic aneurysm, which eventually leaked necessitating repair of the vessel. Everyone agreed with the necessity of surgery and that the use of PPE as described were indicated. Doing the serologic test was important to demonstrate that the risks were controlled during the surgical procedure. Serology results of the patient revealed that she had chronic Q fever infection.

Our study showed that there was no perioperative transmission of *C. burnetii* in the operating room from an infected graft of a patient with chronic Q fever to surgical staffs who wore appropriate personal protective equipment (PPE). Eleven of the 14 staff members of the surgical team did not have a rise in their IgG titers, suggesting they were not infected with the organism during the surgical procedure. Two of the staffs did not return for the sixth week blood draw so we cannot be sure if there was a rise in the titer from the baseline. However, none of these two has reported any symptoms to either infectious control or occupational health department; though we recognized that acute Q fever infection may not present with any symptoms.² One of the staff has a baseline

Q fever IgG phase II serology of 1:128 suggesting the infection at some time prior to the date of the serum collection. Also, there was no rise in his IgG phase I and phase II antibodies six weeks post-exposure. He was referred to an infectious disease specialist for counseling.

In a study by Fenga, *et al*, on the seroprevalence and occupational risk survey of *C. burnetii*, in exposed workers and controls, they found antibodies to *C. burnetii* in 62.9% of the exposed and in 14.3% of the control group.¹ The exposed group in that study was veterinarians, livestock handlers, and laboratory/technical personnel. There was no mention of whether PPE were used that would have prevented inhalation of *C. burnetii*. In our study, PPE such a powered purified air respirator and fitted N-95 respirators were worn during the procedure. This might have been protective in preventing infection in our studied staff.

There have been reports in the literature of nosocomial transmission of Q fever including cases of transmission of *C. burnetii* to pathologists, mortuary attendants, doctors and medical student during post-mortem examination.¹²⁻¹⁴ Some of these case reports, however, have not been verified and phenotypic studies were not done. Many patients with valvular heart disease who developed chronic Q fever infection with endocarditis following acute infection underwent surgery on the diseased valve without subsequent report of a member of the surgical team developing acute Q fever.^{2,21}

The limitation of this case series is the limited number of sample examined. This may affect the generalizability of the results. However, the findings of the study were consistent with other studies in the literature that suggest human to human transmission of Q fever is rare.^{15,18-20}

In conclusion, this study suggested that the risk of transmission of *C. burnetii* in

TAKE-HOME MESSAGE

- Most human Q fever infections are from occupational exposures, through inhalation of *Coxiella burnetii* from dried feces, urine or birth products of infected animals.
- Risk of transmission of *C. burnetii* in the operating room from an infected patient to health care workers who wore appropriate personal protective equipment is low.

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the operating room from an infected patient to health care workers who wore appropriate PPE is low.^{16,17,19} Although some studies suggest that as many as half of humans infected with *C. burnetii* do not show symptoms, none of the 14 exposed operating room health care workers had any symptoms, including the one who has prior evidence of Coxiella infection evidence from his serology result. Also the before and after serology test results for IgG phase I and phase II antibodies suggested that there was no perioperative transmission of Q fever.

Conflicts of Interest: None declared.

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