Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.3077-19
Intern Med 59: 149, 2020
http://internmed.jp

【 LETTERS TO THE EDITOR 】

Rapid Progression of *Cardiobacterium* Endocarditis

Key words: infective endocarditis, *Cardiobacterium hominis*, *Cardiobacterium valvarum*, 16S-rDNA sequencing

(Intern Med 59: 149, 2020)
(DOI: 10.2169/internalmedicine.3077-19)

To the Editor We read with interest the article entitled: "Intracranial Hemorrhaging following *Cardiobacterium Hominis* Endocarditis" by Okumura et al. in Internal Medicine (1). The authors described the case of a patient who suffered intracranial hemorrhage, followed by *Cardiobacterium hominis* (*C. hominis*) prosthetic valve endocarditis. The medical teams among different hospitals adequately communicated with each other and transferred this patient for prompt treatment, including a neurosurgical operation and the immediate administration of antibiotics. We strongly appreciate the dedicated assessment and treatment by these medical teams.

However, one important clinical issue related to his clinical course should be noted. This patient initially suffered intracranial hemorrhage without any febrile episodes. Additionally, the period before clinically significant symptoms occurred was only seven days. Whether this "slow-growing" and insidious bacterium could cause such a rapid clinical course in this short period is questionable (1).

One author among us reported a case series at a Japanese university hospital (2). The report described 82 cases of definite infective endocarditis, one of which was caused by *C. hominis*. This case took two months to diagnose because the patient presented vague and less significant symptoms, including general malaise, weight loss and leg edema. Additionally, Walkty (3) and Malani et al. (4) reviewed the literature on *C. hominis* endocarditis and reported that the mean duration of symptoms before the diagnosis was 145 days (<1 week to >11 months) in 67 cases and 138±128 days (2-540 days) in 61 cases, respectively.

There are two possible explanations for this controversy. First, host factors (e.g., an immunocompromised patient or a previous diagnosis of cerebral aneurysm), neither of which were mentioned in this article, might have been involved. Second, pathogen factors might have been involved. Han et al. described the case of a 37-year-old man who suffered bicuspid-aortic-valve infective endocarditis caused by *Cardiobacterium valvarum* (*C. valvarum*) with a sudden rupture of a cerebral aneurysm without any febrile episode (5). This newly identified pathogen has many phenotypic similarities to *C. hominis*. These authors suggest that some previous cases of afebrile endocarditis that was thought to have been caused by "*C. hominis*" might have been caused by *C. valvarum*. Thus, we hypothesize that this patient was infected with a similar but significantly different strain, *Cardiobacterium valvarum*. Due to the great phenotypic similarities, Han et al. detected the difference between these two pathogens using both 16S-rDNA sequencing and phenotypic characterization (5). We would like to ask the authors whether this pathogen was analyzed by 16S-rDNA sequencing?

The authors state that they have no Conflict of Interest (COI).

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Received: March 25, 2019; Accepted: June 20, 2019; Advance Publication by J-STAGE: September 3, 2019
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