A Consideration of the Differences between a Janeway's Lesion and an Osler's Node in Infectious Endocarditis

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Janeway's lesions and Osler's nodes are regarded as excellent clues to the diagnosis of infectious endocarditis; however, very few physicians have actually witnessed these findings, and there is some confusion in distinguishing between the two. This article concerns a patient with infectious endocarditis due to Diplococcus pneumoniae, who had tender vesicular lesions thought to be Osler's nodes and a nontender erythematous nodule on the foot.

Janeway's lesions and Osler's nodes are highly prized clues to the diagnosis of infectious endocarditis, and the differences between an Osler's node and a Janeway's lesion are poorly understood. For this reason, we believed that a review of the original description and subsequent modifications might be of interest.

CASE REPORT

A 16-year-old boy was admitted to Henrietta Egleston Hospital with meningitis and bacterial endocarditis. Two years prior to admission, he had undergone corrective surgery for tetralogy of Fallot and, in addition, had had a prosthetic aortic valve inserted. One year prior to admission, the patient was successfully treated for bacteremia and Schönlein-Henoch purpura. He did well until two weeks prior to admission, when he developed acute otitis media in the right ear. The patient improved with three days of therapy with cephalixin monohydrate. Two days after the therapy with cephalixin monohydrate was discontinued, he developed fever spiking to 38.3°C (100.5°F) associated with increasing headaches, irritability, nausea, and vomiting.

On admission, the patient was acutely ill, with a temperature of 38.3°C (101°F), a pulse of 116 beats per minute, a respiratory rate of 30/min, and blood pressure of 124/90 mm Hg. No petechiae or other cutaneous lesions were initially noted. Both tympanic membranes were red and retracted; however, no pus was seen. There was moderate nuchal rigidity. The lungs were clear to auscultation and percussion. Cardiac examination revealed a regular rhythm and sharp opening and closing clicks of the aortic prosthetic valve. No murmurs or gallops were heard. Laboratory results included a hematocrit reading of 34 percent and a white blood cell count of 8,850/cu mm, with 81 percent neutrophils. A chest x-ray film showed clear lung fields, a normal cardiac silhouette, and the aortic prosthetic valve. A spinal tap produced purulent fluid, revealing Diplococcus pneumoniae on gram staining and culture.

Hospital Course

The patient was initially treated with 2 gm of ampicillin and then was given 17 million units of penicillin intravenously per day. He continued to have daily fever spikes in excess of 39.4°C (103°F). His mental status rapidly cleared. On the fifth day of hospitalization, a grade 2/6 crescendo-decrescendo systolic murmur was noted in the second right intercostal space at the sternal edge, and a grade 1/6 diastolic murmur was noted in the third left intercostal space of the sternal edge. No change was noted in the prosthetic valvular sounds. On the tenth day of hospitalization, three exquisite tender, 2 to 3-mm vesicles were noted in the pad of the right index finger. Cultures and gram stained preparations of the serous fluid from these lesions were negative. On the following day, the patient developed a raised 3-mm erythematous nodule of only four hours' duration in the sole of the right foot (Fig 1). This lesion was initially mildly tender but was not tender when reexamined after one hour. The lesion was associated with a fever spike to 40.5°C. No other cutaneous lesions were noted during the rest of the patient's hospital course. He continued to do poorly, with daily fever spiking to 38.9°C (102°F). Attempts to obtain repeated cultures while the patient was not receiving antibiotics were negative. On the 24th day of hospitalization, he was taken to surgery for replacement of the prosthetic valve.
FIGURE 1. Raised erythematous nontender area occurring on sole of right foot of our patient.

This was accomplished; however, the patient died shortly after surgery.

Examination of the heart at autopsy revealed multiple areas of abscess formation and necrotic debris in the suture sites. Gram-positive diplococci compatible with *Diplococcus pneumoniae* were seen in the necrotic tissue. In addition, a rupture of the membranous septum was present, allowing communication between the left ventricle and both right cardiac chambers.

**DISCUSSION**

**Janeway's Lesions**

Edward Janeway's original description was in the context of separating "malignant" (acute) endocarditis from other infectious disorders. In an 1899 article published in *Medical News,* he stated:

In trying to determine whether a given case is more probably due to endocarditis or to another malignant process I have found that attention to the position of the hemorrhages is at times very helpful. Several times I have noted numerous small hemorrhages with slight nodular character in the palms of the hand and soles of the feet, when possibly the arms and legs had but a scanty crop in malignant endocarditis, whereas this has not been my experience with processes likely to be mistaken for it.

Emanuel Libman, a student of Janeway, applied the eponym, "Janeway’s lesion.” Libman originally considered these lesions to be pathognomonic for acute bacterial endocarditis but later remarked that he had also seen them in three patients with subacute bacterial endocarditis. Libman pointed out that this lesion was not tender, in contrast to the exquisitely painful Osler's node. A Janeway's lesion must have been a relatively uncommon finding, for it was not mentioned by other authors; however, in 1912, Osler did note lesions that may have been Janeway's lesions. He described the lesions as "peculiar areas of persistent erythema" on the palms and soles in a patient with subacute bacterial endocarditis. These were different from the lesions he associated with infectious endocarditis that were later called Osler's nodes. Libman and Friedberg's ex-

**Figure 2.** Janeway's lesion. This artist's illustration of Janeway's lesion is reproduced from Libman and Friedberg's monograph on subacute bacterial endocarditis published in 1949 (reproduced by permission of Oxford University Press).

**Figure 3.** Osler's node. This exquisitely tender erythematous nodule appeared on fingertip of drug addict with tricuspid regurgitation due to staphylococcal endocarditis (from Silverman and Hurst; reproduced by permission of McGraw-Hill Co., from color plate 4B, chapter 13 [General Inspection] in Hurst JW, Logue RB, Schlant RC, Wenger NK: The Heart, Arteries and Veins, 3rd ed, 1974).
tensive monograph on endocarditis in The Oxford Medicine of 1949 uses an artist’s illustration of a Janeway’s lesion (Fig 2), rather than an actual photograph. A black-and-white photograph of only fair quality can be found in Kerr’s10 description of Janeway’s lesion. In 1966, Cross and Ellis11 published color photographs of painless irregular hemorrhages resulting from a cutdown site infected with Pseudomonas organisms; the hemorrhages were reported as Janeway’s lesions. Libman and Friedberg6 in 1949 and Friedberg12 in 1966, emphasized the fact that Janeway’s lesions were infrequent, were more characteristic of acute bacterial endocarditis than subacute bacterial endocarditis, and were never painful. These investigators also noted that Janeway’s lesions may appear erythematous, rather than hemorrhagic, in subacute bacterial endocarditis.

A pathologic description of a tender Janeway’s lesion found among numerous nontender lesions is in Kerr’s10 text on Subacute Bacterial Endocarditis. Kerr10 describes the histologic findings of the tender lesion as a microabscess of the dermis with marked necrosis and inflammatory infiltrate not involving the epidermis. Cultures of a nearby area were positive for Staphylococcus aureus, although no organisms were seen on tissue examination of the actual lesion.

Osler’s Nodes

In contrast, the description of Osler’s nodes (Fig 3) by William Osler13 was associated with chronic (subacute) bacterial endocarditis.5,8 In his Güstonian Lecture Series of 1885, Osler14 described the cutaneous manifestations of severe infectious endocarditis as “haemorrhages . . . upon the skin, serous and mucous surfaces,”14,417 in which “a minute necrotic or suppurrative centre can sometimes be seen.”14,417 In a 1909 issue of the Quarterly Journal of Medicine, Osler13 reported the findings subsequently associated with his name. Osler13 gave credit to Dr. J. A. Mullin of Hamilton, Ontario, who brought these lesions to his attention in 1893.10 Other authors had also noted this lesion prior to 1908.2,16,11,13 In Osler’s13 report, he presents ten patients with chronic infectious endocarditis, including seven with cutaneous lesions described as:

Ephemeral spots of a painful nodular erythema chiefly in the skin of the hands and feet. . . . The spots came out at intervals as swollen areas, some the size of a pea, others a centimetre and a half in diameter, raised, red, with a whitish point in the center. I have known them to pass away in a few hours but more commonly they last for a day or even longer. The commonest situation is near the tip of the finger which may be slightly swollen. . . .

F. Parkes Weber in 191315 named these findings “Osler’s sign, Osler’s spots or Osler’s symptom,” pointing out that Osler distinguished them from other lesions and made their significance known to general medicine.

Since Osler’s original description, others have added additional clinical manifestations, such as: “painful nodule;”9 “preceded by painful sensation . . . reddish cyanotic induration occurring within one hour of the pain;”16 “not hemorrhagic but erythematos and occasionally with vivid pink hue and opaque center;”10,13 “deeper lesions painless or if painful may display few other objective signs;”8,10 “occasionally break down in the center and result in small ulcers;”8 “skin over them may desquamate but only rarely does it ulcerate;”6 and “erythematous area without the formation of nodules . . . always tender when recent . . . even if the patient is not sensitive to pain . . . tender anemic area.”8

Histologically, Osler’s nodes are reported to be a necrotizing vasculitis of the dermal glomus in the lower keratinous mantle, leading to congestion, obstruction, and inflammatory infiltration of the vascular channels.10,15,16 Cultures of Osler’s nodes have generally been negative. Although Osler’s nodes are strongly suggestive of bacterial endocarditis, they have been noted to occur in lupus erythematosus.9

Current textbook descriptions of Janeway’s lesions and Osler’s nodes are given in Table 1. In a written communication (April 23, 1975), Louis Weinstein stated:

While there be no difference histologically since both may be evidence of a vasculitis, there is a striking difference in the clinical presentation of these lesions. Clinically the Osler’s node is a painful, tender blush-purple nodular lesion situated in the terminal phalanges of the finger or toes. The Janeway’s lesion on the other hand is a painless nontender lesion that is pink in color, irregular in shape and a macular lesion present more commonly on the thenar and hypothenar eminences of the hands and feet.

From these various descriptions and the few pictures available, it may be difficult to decide if a lesion is an Osler’s node or a Janeway’s lesion. It must be remembered that both Janeway and Osler were describing clinical findings which were used as diagnostic clues to distinguish acute from subacute bacterial endocarditis, as well as from other infectious disorders such as typhoid and malaria. The only distinguishing features between the two lesions seem to be the pain and suppuration that occur with an Osler’s node and not with a Janeway’s lesion. Other criteria, such as color, nodularity, size, location, and duration, are variable. Pathologically, both lesions are described as a vasculitis with inflammation and central necrosis.15,17,18 A similar histologic
Our patient displayed a cluster of exquisitely painful intracutaneous vesicles which were probably Osler's nodes in the suppurative phase. In addition, he had an essentially nontender erythematous nodule similar to the lesion described by Janeway. These lesions were distinctly different clinically and led us to the final conclusion that the patient had both Osler's nodes and a Janeway's lesion.

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Ethology

It is also both scientifically legitimate and operationally necessary to ascribe mind to highest animals. This is obvious as regards the anthropoid apes: they not only possess very similar bodies and sense-organs to ours, but also manifest similar behavior, with quite a similar range of emotional expression; a range of curiosity, anger, alertness, affection, jealousy, fear, pain and pleasure. This is obvious as regards the anthropoid apes. It is equally legitimate and necessary for other mammals, although the similarities are not so close. We cannot begin to understand or interpret the behavior of elephants or dogs or cats or porpoises unless we do so to some extent in mental terms. This is not anthropomorphism: it is merely an extension of the principles of comparative study that have been so fruitful in comparative anatomy, comparative physiology, comparative cytology and other biological fields. It is equally fruitful when you extend it to the study of animal behavior, this rapidly developing modern branch of science which is called ethology.