Pyogenic Osteomyelitis: The Normal Bone Spaces As A Bacterial Reservoir

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PYOGENIC OSTEOMYELITIS: THE NORMAL BONE SPACES AS A BACTERIAL RESERVOIR
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It is well known that pyogenic osteomyelitis is a different clinical infectious process from pyogenic soft tissue infections even though the organisms causing the two may be the same. Empirically clinicians have learned that, depending on the causative organism, pyogenic osteomyelitis has a significant tendency to recur after cessation of therapy and that larger and longer dosage of antibiotic is necessary in pyogenic bone infections than in pyogenic soft tissue infections.

The reasons for these differences are not generally known or published although the basic factual material needed to explain them is available. In this paper, explanation is begun by outlining the ability of bone's normal spaces to harbor bacteria.

TYPES OF SPACES IN BONE

The gross appearance of a bone is so convincingly that of a homogeneous solid that this concept unconsciously colors our thinking when we deal with skeletal anatomy and physiology in clinical practice. Yet we know that various spaces exist in bone.

The normal spaces in bone are the lacunae which contain the bone cell, the osteocyte; the canaliculae, which connect the lacunae to the nearest source of blood supply, either directly or by a relay system through anastomoses with other lacunae; Haversian canals, which are longitudinal channels containing blood vessels of varying size along with some loose connective tissue; Volkmann's canals, which run perpendicularly through the cortex bringing in blood vessels from the periosteal and endosteal surfaces and connecting adjacent Haversian systems; and the large medullary or marrow canal. With the exception of the canaliculae all of these spaces normally contain cells. The canaliculae are only partly filled by short cytoplasmic processes extended from the osteocytes.

NUMBER OF SPACES IN BONE

Measurements made on material from a group of human cases of various ages in the Henry Ford Hospital Orthopaedic Research Laboratory are presented here. They were made on sections prepared by the writer's methods so no significant error occurs from shrinkage.

There are from 4 to 25 vascular channels/mm\(^2\) in human lamellar bone, there being characteristic variation with age, bone and part of a bone examined.

There are about 26,000 osteocyte lacunae/mm\(^2\) in human lamellar bone, there being about a 25% variation in this figure in different cases and ages. There are from two to three times this many lacunae in fibrous bone.

There are about 40 canaliculae per osteocyte lacuna, baking a total of over 1,000,000 canalicular spaces/mm\(^2\) in human lamellar bone and far more than this in fibrous bone.
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This is a truly impressive number of spaces in a cubic millimeter of bone. These spaces are so small that they normally comprise only about 7% of the total volume of the cortex, about 5% of the cortical volume being vascular canals and about 2% being the lacunar and canalicular lumens.3

BACTERIAL INVASION OF THE BONE SPACES

Now let us consider a portion of a bone which has been killed as the result of a pyogenic osteomyelitic process.4 The dead bone portion may be cortical or cancellous and initially it is in physical continuity with adjacent, living bone. Only when repair processes intervene is a dead portion of bone separated from living. When separated it is called a sequestrum.

The vascular spaces in the dead portion of osteomyelitic bone contain multiplying pathogenic bacteria. Since the bone is dead, then by definition its cells are dead and constitute a good culture medium. The bacteria will multiply and grow into unininvaded dead tissue until all of this culture medium has been exhausted. The culture medium occupies every vascular channel and most of the lacunae. The result is that there may be bacteria present in about 1,000,000 spaces per mm^3 of the infected, dead bone. These bacteria may actually be photographed in situ in the bone spaces3 in specimens from fresh cases of pyogenic osteomyelitis. We may accordingly consider the spaces in a dead, infected portion of a bone and the bacteria in them to be the bacterial reservoir of pyogenic osteomyelitis.

PERIOD OF VIABILITY OF THE BACTERIAL RESERVOIR

It is natural to inquire next about the length of time required for the bacteria in the bacterial reservoir described above to die as the result of exhaustion of their culture material. Here clinical experience is drawn on to provide an answer.

An attribute of osteomyelitis caused by beta hemolytic streptococcae and pneumococcae is the lack of recurrence once the immediate disease has run its course. This was true even in the preantibiotic era in patients who survived their initial infection. It is notable that pyogenic osteomyelitis due to these organisms is commonest in the first six months of life.

An attribute of osteomyelitis caused by staphylococcus aureus is its tendency to recur, often at some time remote from the initial osteomyelitic episode. Numbers of cases are known in which exacerbation occurred fifty years after the onset of the disease and the writer knows first hand of a case of Dr. Ward Plummer's of Buffalo5 in which the recurrence was 70 years after the initial osteomyelitis.

If recurrences were due to reinfection of dead bone with new organisms by the hematogenous route, then the streptococcal and pneumococcal infections would also recur and there would be recurrences in which the organism was different from the

(a) It is understood that various factors in varying degree may be responsible for this local bone death. These factors would include venous thrombosis, increased hydrostatic pressure within the marrow cavity, direct bacterial invasion, action of bacterial toxins and accumulation of a large volume of exudate.

(b) Deceased 1952.

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organism causing the initial ostemyelitic episode. Neither is the case. In fact, even strain types and antibiotic sensitivities of each recurrence are regularly identical to the types and sensitivities of the original organism, where this data is available. We may conclude that staphylococci in some manner can remain viable and virulent for many years in the bacterial reservoir. Similar behavior is distinctly unusual for streptococcae and pneumococcae.

DISCUSSION

The invasion of millions of tiny bone spaces by multiplying bacteria explains the source of the bacteria responsible for a recurrence of a pyogenic osteomyelitis. Questions are automatically raised as to the physical and chemical means of survival of staphylococci over long periods in a portion of dead bone, the reason for the failure of other organisms to survive under apparently similar conditions, and the behavior of the large group of organisms which only occasionally cause pyogenic osteomyelitis but which have not been directly referred to in this paper. These are matters for additional work.

Another question occurs which is equally pertinent at this point, and that is: why do some cases of Staphyogenic osteomyelitis recur and some not? In the hypotheti-

Figure 1

Cross section fibula 11 yr. girl with chronic osteomyelitis; surgically resected specimen. Fresh, undecalcified, fuchsin stain. The identifying black marks designate canaliculae within the depth of the section which contains minute, spherical particles that are probably staphylococci. The border of a resorption space is seen at the left of the figure. Such bacteria existing in the minute bone spaces over variable lengths of time constitute the "bacterial reservoir" of osteomyelitis.

(c) And perhaps some other organisms.
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cal situation outlined, the factors stated were assumed to be constant from one case to another, leaving no room for the clinical variability which occurs. Additional factors such as the virulence of the organism, effectiveness of the host defense mechanisms, size of the dead portion of the bone, growth, remodelling activity and behavior of antibiotics in bone are known to exist and would be expected to influence the clinical course and outcome. Some of these factors will be considered in additional publications in preparation.

SUMMARY

Bone normally contains over 1,000,000 spaces/mm³. These spaces are heavily contaminated with bacteria in portions of bone killed during an osteomyelitic process. Clinical evidence suggests that the bacteria in these spaces constitute a bacterial reservoir which supplies the organisms responsible for recurrences of the osteomyelitis. The period of time during which the bacteria remain viable differs with different organisms, (and probably differs with additional factors not considered here), being longest for staphylococci and shortest for streptococci and pneumococci.

REFERENCES