CRYSTAL SYNOVITIS

Every now and again the practice of medicine or surgery is enlivened by a new concept which makes sense, and the clinician begins to observe and understand some phenomenon which hitherto he had not noticed, or had ignored simply because it did not fit in with preconceived ideas. How often in the past, for example, have we laid down radiographs of a knee joint showing calcified menisci without particular comment? And how often has the diagnosis of septic arthritis been made despite sterility of joint fluid, or the diagnosis of gout maintained in the absence of hyperuricaemia?

It took over a century, from 1848 to 1961, for the fact to be demonstrated that acute gouty arthritis is due to precipitation of urate crystals in the joint fluid. In 1961 Faires and McCarty were able to produce acute arthritis in both man and dog by intra-articular injection of sodium urate crystals, and this was confirmed the next year by Seegmiller and his colleagues (Seegmiller, Howell and Malawista 1962). Finally there was the observation first made by McCarty and his colleagues (McCarty and Gatter 1962, 1963; Kohn, Hughes, McCarty and Faires 1962) that in some cases of acute arthritis with crystals demonstrable in the joint fluid, the crystals were not of urate. These microcrystals, whether or not of urate, can often be seen to lie within the leucocytes, which appear to behave in much the same way as they do with bacteria, the whole appearance thus resembling septic arthritis. This led to the concept that there was a condition of crystal synovitis, of which "gout" is only one example. Microscopy of joint fluids by polarised light may thus distinguish two types of crystal: those not birefringent and digested by uricase—urate crystals; and those weakly birefringent and not digested by uricase—usually identified as consisting of calcium pyrophosphate. It is to the latter group that the diagnosis of "pseudogout" has been applied. This nomenclature, which has historical roots, is in other respects unsatisfactory because it may well conceal a number of different types of crystal synovitis. Indeed gout is probably an unsatisfactory diagnosis itself, because it is the hyperuricaemia which is the essential abnormality. This may have multiple causes and multiple effects, the joint manifestations representing only one of
these, often occasional and indeed sometimes absent. Thus "podagra" is a useful descriptive
term whilst "gout" is no longer a strictly useful diagnosis. The term chondrocalcinosis
articularis may be preferred for "pseudogout," for it seems clear that a characteristic feature
of calcium pyrophosphate crystal synovitis is calcification of both hyaline and fibrocartilage.
This term was coined by Žišnán and Šit'aj (1963) who described twenty-seven cases in which
the diagnosis was based on the radiographic appearance—an appearance typical of calcium
pyrophosphate synovitis (McCarty and Haskin 1963)—but did not, in fact, examine the joint
fluid for crystals. In practice the radiological finding of calcified cartilage, often first seen in
the menisci, provides the commonest clue to the diagnosis and should never be overlooked.
It should be added that crystals of calcium pyrophosphate have been identified in affected
cartilage (McCarty and Gatter 1962, 1963), but further work is clearly needed, including the
examination of control material. It would perhaps be best if "chondrocalcinosis articularis"
was restricted to describing the radiological appearances alone.

Our understanding of this syndrome is carried further by two contributions which are
published in this issue. The resemblance to septic arthritis is emphasised by Hamblen, Currey
and Key from the London Hospital, who describe two patients who were thought initially
to be suffering from septic arthritis of the ankle in one, and of the knee in the other, but in
whom crystals of calcium pyrophosphate were demonstrated in the joint fluid and the
characteristic radiological appearance was seen. Mann describes five further patients in whom
the diagnosis was supported by the clinical and radiological findings but without the
demonstration of crystals. He argues that the estimation of synovial calcium levels is valuable:
but if the concept of crystal synovitis is correct it seems preferable to look for these rather
than to talk of "calcium irritation." As both Hamblen and his colleagues and Mann point out,
however, we must not allow ourselves to be carried away by a too ready assumption that all is
going to remain quite simple. In many cases no evidence of a metabolic disorder can be found.
On the other hand, as Bywaters and his colleagues have pointed out (Bywaters, Dixon and
Scott 1963; Scott, Dixon and Bywaters 1964), in hyperparathyroidism the radiological picture of
chondrocalcinosis articularis may develop and this is presumably a result of the hypercalcaemia.
Furthermore, hyperuricaemia may occur secondarily in hyperparathyroidism. It has been
accepted that the acute attacks of arthropathy which occur in this condition may be due
to secondary gout. Unfortunately the synovial fluid in these cases has not been examined for
microcrystals and it is not known whether these are of urate or of calcium pyrophosphate.
Recently Jackson and Harris (1965) described such a case—a patient with gout and
hyperparathyroidism with acute arthropathy and chondrocalcinosis articularis. They claimed
to have found crystals of both calcium pyrophosphate and of urate in the joint fluid from
the knee. But their evidence was rejected by Currey and Swettenham (1965) on good technical
grounds; so the question still remains open. Moreover, as Currey and Swettenham point out,
in the London Hospital experience, which is now of at least thirty patients whose joint fluids
have been examined, the findings are not quite so clear-cut as was hoped and there is the
possibility that other crystals may occasionally be implicated—possibly, for example, calcium
oxalate. It may be that both "gout" and "pseudogout" can co-exist in the same
patient, because they report one case of classical "gout" in which the joint fluid showed
calcium pyrophosphate crystals. We have another patient in this series with hyperuricaemic
gout but with the typical radiological appearance of chondrocalcinosis articularis; the joint
fluid contained only urate crystals.

The next few years should show a rapid advance in our understanding of crystal synovitis,
especially if we do not, once more, close our minds by accepting the diagnosis of "pseudogout"
as being sufficient and definitive. It would be better in our present state of knowledge to accept
"crystal synovitis" as a syndrome and to endeavour to identify the crystals and the possible
metabolic anomalies causing their precipitation.

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REFERENCES


