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A CONTRIBUTION TO THE QUESTION OF
THE ORIGIN OF ANTERIOR PARADISCAL
DEFECTS AND SO-CALLED PERSISTING
APOPHYSES IN THE VERTEBRAL
BODIES

BY

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In *Acta Orthopaedica Scandinavica*, 1947, I reported a series of cases of dorsal insufficiency in whom radiography showed a special characteristic lesion of the vertebral body, viz, a defect in the anterior part, adjacent to the intervertebral disc. This defect communicated with the disc and was surrounded by a sclerotic zone. I called it an anterior paradiscal defect.

I also discussed the cause of these lesions, and came to the conclusion that they probably originated in an impaction of disc tissue into the vertebral body, as had been suggested earlier by W. Müller; and that both local failure of enchondral ossification (R. Hanson's view) and Scheuermann's disease (which Overgaard and Brocher accept) could be excluded. The mode of origin would seem to resemble that of Schmorl's Knorpelknötchen, though, while these, which are situated in the central and posterior part of the vertebral body, are caused by impaction of the nucleus pulposus into the body, in the case of the anterior paradiscal defects it is supposed to be the tissue lying anterior to the nucleus pulposus which is impacted. In only a small proportion of the cases could the anterior paradiscal defect be attributed to a definite single trauma, in most it had resulted from gradual impaction of

the disc tissue into the cancellous bone of the vertebral body, owing presumably to the daily stress of ordinary activity.

I also described two cases in which there was a so-called persisting apophysis adjoining the anterior paradiscal defect, which communicated with the narrow cleft separating the apophysis from the rest of the vertebral body, suggesting that both must be due to the same causal factor.

Since publishing that paper I have seen several additional cases of anterior paradiscal defect. A remarkably high proportion were associated with special anatomical conditions, which particularly favoured the impaction of disc tissue just at the site of the defect.

Normally the anterior angles of the vertebral bodies lie in identical positions exactly opposite one another, above and below the disc. But this is remarkably often not so in cases where there is an anterior paradiscal defect. Frequently the vertebral body in which the defect is situated is wider antero-posteriorly than the adjacent vertebra and thus its anterior angle lies in front of the angle on the other side of the disc. In all those cases in which the vertebrae differed in size the defect was found in the upper part of the vertebral body, whose upper anterior angle lay in front of the lower anterior angle of the vertebra above. (Fig. 1). This difference in the sizes of the vertebral bodies is of course of congenital origin.

In these cases, if the subject bends forward, the lower frontal angle of the upper vertebra will with considerable force press down the intervertebral disc slightly behind the upper anterior angle of the lower vertebra, that is to say at just the place where the defect is found.

Thus the intervertebral disc is impacted into the cancellous part of the vertebral body, inside its comparatively firm margin. The more curved is the anterior margin of the upper vertebra, the bigger will be the part of it which is behind the anterior margin of the lower vertebra and the greater will be the force with which the disc will be pressed down into the cancellous bone. The depression is filled with elastic disc tissue. Every time the spine is flexed the pressure is trans-

mitted through the elastic disc to the bone round the depression and sclerosis develops in response to the repeated variations in pressure. Thus the defect is eventually enclosed in sclerotic bone, which prevents further impaction of the disc tissue into the vertebral body.

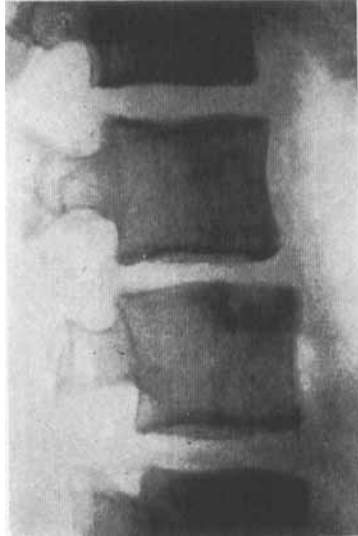


Fig. 1.

This area of sclerosis is also often of importance for the radiographic demonstration of the defect. If the cortex is intact, only a fairly big defect will be visible radiographically. Böhmig and Prévôt using a curette produced cherry-sized cavities in vertebral bodies and were unable to detect them in the radiographs taken subsequently. Therefore an impaction produced by trauma may not be detected in radiographs taken immediately after the accident and may only become visible later, i.e. when sclerosis has developed round the depression.

There are, in addition, some cases in which there is no difference in the sizes of the vertebral bodies, but, instead, the vertebra next to that with a defect fails to show a normal anterior angle (see Fig. 2). The angle is, as it were, cut off.

In these cases, as in those where the vertebral bodies are of different sizes, flexion of the spine causes the intervertebral disc to be pressed with considerable force down onto the lower vertebra just behind the anterior angle, that is, onto just that area in which the paradiscal defect is found. The absence of a normal anterior angle is due to a developmental failure, either the anterior apophysis was never formed, or it developed away from the normal position, either near the centre of the vertebral body or more posteriorly.



Fig. 2.

Thus special anatomical conditions, such as different sizes of two adjacent vertebral bodies, or absence of a normal anterior angle, which are mechanically favourable to the impaction of disc tissue at the site of the paradiscal defect, are remarkably often present in these cases.

My series comprised 16 patients with anterior paradiscal defects. One patient had defects in two vertebral bodies, while in the remainder only one vertebra was affected. Thus the total number of paradiscal defects was 17. The Table shows

that the anatomical conditions which predispose to their formation, as has just been described, were present in 12 out of the 17 instances, that is to say in the majority. In 9 cases the vertebrae differed in size and in 3 the anterior angle was absent. 12 of the defects were situated in the upper part of the vertebral body and 5 in the lower. In all the cases where the vertebrae were of different sizes the defect was found in the upper part of the affected vertebra. The upper vertebra was the one which was comparatively small compared with the lower which showed the paradiscal defect. Of the 3 instances where there was no normal anterior angle, 2 had the defect in the upper, and 1 in the lower part of the vertebra.

In 16 cases the defect was found in a lumbar vertebra and in 1 in the twelfth thoracic. Thus, it is mainly the lumbar spine which is affected. It may be supposed that this particular susceptibility of the lumbar spine is largely due to the relatively free movement between the vertebra, and the greater thickness of the intervertebral disc in this region.

The age at which spinal symptoms developed varied from 9 to 52 years, but a strikingly high proportion of the patients were between 9 and 16 years; this age-group comprised 8 out of the 16 patients, i.e. one half of the series.

In 6 out of the 16 cases the back symptoms developed immediately after an accident: all these 6 cases belonged in the age group 9-19 years. In the age-group 9-16 years, in which there were 8 patients, 5 had developed their symptoms in close relation to trauma. Thus the defects were relatively more common during childhood, and at this age they more frequently followed trauma. This observation can perhaps be partly explained by the morphology of the intervertebral discs during childhood, when they are relatively broad and elastic, and partly by the relative deficiency of lime salts of the cancellous bone in the vertebral body, which therefore yields more easily to pressure. In the cases where the symptoms did not appear before the patient was 20-years-old no relation between their onset and a particular trauma could be established and in these cases therefore the defects would appear to have devel-

Cases of Anterior Paradiscal Defect.

Case no.	Sex	Age in years at which symptoms developed	Site of lesion	Special anatomical conditions	Trauma	
1	♂	16	Lower part of 1st lumbar	None	Yes	
2	♂	22	Upper part of 3rd lumbar	Defective anterior angle	No	
3	♀	31	Upper part of 12th lumbar	Vertebrae differing in size	No	
4	♂	13	Lower part of 1st lumbar	Defective anterior angle	No	
5	♂	9	Upper part of 2nd lumbar	Vertebrae differing in size	Yes	
6	♀	10	Lower part of 1st lumbar; lower part of 2nd lumbar	None	Yes	
7	♂	52	Upper part of 4th lumbar	Vertebrae differing in size	No	So-called persisting apophysis
8	♂	40	Upper part of 3rd lumbar	Vertebrae differing in size	No	So-called persisting apophysis
9	♂	19	Upper part of 3rd lumbar	Vertebrae differing in size	Yes	
10	♂	32	Upper part of 5th lumbar	Vertebrae differing in size	No	
11	♀	36	Upper part of 3rd lumbar	Defective anterior angle	No	
12	♂	10	Upper part of 3rd lumbar	None	No	
13	♀	10	Upper part of 4th lumbar	Vertebrae differing in size	Yes	
14	♂	15	Upper part of 4th lumbar	Vertebrae differing in size	No	
15	♂	12	Upper part of 1st lumbar	None	Yes	
16	♀	31	Upper part of 4th lumbar	Vertebrae differing in size	No	So-called persisting apophysis

oped gradually with the stress of everyday activity. An essential preliminary is the degeneration of the cartilaginous lamina which covers the surface of the vertebral body, this is followed by the formation of fissures in it and by subsequent re-absorption. In this way the elastic pressure of the intervertebral disc is now transmitted directly to the bone of the body, which is absorbed, and a paradiscal defect appears. It is also conceivable that the defect may be due to a trauma which occurred during childhood and has been forgotten when the dorsal symptoms arise many years later and a radiographic examination is made.

Amongst these cases 3 showed so-called persisting apophyses situated immediately in front of the paradiscal defect. The defect communicated with the narrow cleft separating the apophysis from the rest of the vertebral body. In every instance the defect and the apophysis were situated in the upper part of the vertebral body, and the body of the vertebra above was relatively small, so that its anterior angle was exactly opposite the paradiscal defect (see fig. 3). Thus with flexion of the spine the lower anterior angle of the upper vertebra pressed down the intervertebral disc towards just that place where the defect was situated.

The origin of these so-called persisting apophyses may be imagined in the following way. First, the anterior paradiscal defect is produced by the impaction of disc tissue behind the anterior angle of the vertebral body, and with each flexion of the spine pressure is transmitted from the upper vertebral body through the intervertebral disc to the bony margin of the paradiscal defect. Thus the pressure acts on the intact angle of the vertebral body in front of the defect. Either this angle fractures suddenly as a result of strong pressure, or a so-called insidious fracture occurs through the angle as a result of the repeated variations in pressure transmitted to it by the intervertebral disc. These variations in pressure in the disc tissue which is protruding into the defect are transmitted to the fractured anterior angle and prevent it from re-uniting with the body, so that a pseudarthrosis develops.

This view of the mode of origin of so-called persisting apophyses is also in agreement with Schmorl's conception, which was based on a pathological-anatomical study. In Schmorl's opinion, the so-called persisting apophyses are not of development origin (as Hellmer et al. maintained), but result either from fractures which have not united or from gradual im-



Fig. 3.

paction of disc tissue into the vertebral body, separating off the anterior angle.

Many cases of so-called persisting apophysis show no radiographic evidence of anterior paradiscal defect, and it may be that not all originate in the manner sketched here. It is not entirely improbable that the developmental processes may sometimes miscarry so that the apophyseal nucleus fails to unite with the vertebra. True persisting apophyses might arise in this way.

Similar conditions to those which appear with vertebrae of different sizes are also present in spondylolisthesis. Here the anterior angle of the lower vertebra is behind the anterior

border of the vertebra which has slipped forward. It might therefore be assumed that spondylolisthesis would predispose to anterior paradiscal defects, but there have been no cases of this condition in my series, and though I have examined a number of radiographs of spondylolisthesis, as well as reproductions in papers on the subject, for anterior paradiscal defects, I have not found any. However, in most cases of spondylolisthesis there is degeneration of the intervertebral disc, which therefore lacks the elasticity necessary for the production of a paradiscal defect.

SUMMARY

In most of the cases of anterior paradiscal defect there were special anatomical conditions, which were conducive to their formation. Usually the vertebral body affected was comparatively big, so that on flexion of the spine the lower anterior angle of the vertebra above pressed down the disc with considerable force just where the paradiscal defect was situated. A similar condition is found when the anterior angle of a vertebral body is defective. Half of the patients were between 9 and 16 years old, and in a notably large proportion of them the symptoms had appeared in close relation to trauma. Some so-called persisting apophyses are due to paradiscal defects. There may also be true persisting apophyses, i. e. those due to failure of development.

RESUME

Dans la plupart des cas de déféctuosités paradiscals antérieures, il existe des conditions anatomiques spéciales devant forcément conduire à leur formation. En général, le corps vertébral atteint est relativement grand, de sorte qu'en cas de flexion de la colonne vertébrale, l'angle antérieur inférieur de la vertèbre située au-dessus appuie sur le disque avec une force considérable, juste à l'endroit où est située la déféctuosité paradiscals. Il en est de même lorsque l'angle antérieur du corps vertébral est déféctueux. La moitié des malades étaient

âgés de 9 à 16 ans, et pour beaucoup de ceux-ci les symptômes se sont manifestés en relation étroite avec un trauma. Les apophyses dites persistantes sont dues à des déféctuosités paradiskales. Il peut exister aussi de véritables apophyses persistantes, c'est-à-dire celles qui sont dues à des défauts de développement.

ZUSAMMENFASSUNG

In den meisten Fällen von vorderen paradiskalen Defekten waren besondere anatomische Verhältnisse vorhanden, die ihre Entstehung gefördert haben. Gewöhnlich war der angegriffene Wirbelkörper verhältnismässig gross, so dass die untere vordere Ecke des darüberliegenden Wirbels bei einer Flexion der Wirbelsäule den Diskus mit beträchtlicher Kraft gerade an der Stelle herunterdrückte, wo der paradiskale Defekt lokalisiert war. Eine ähnliche Sachlage wurde festgestellt, wenn die vordere Ecke eines Wirbelkörpers defekt ist. Die Hälfte der Patienten war zwischen 9 und 16 Jahren alt, und bei einem bemerkenswert grossen Teil von ihnen sind die Symptome in enger Verbindung mit einem Trauma aufgetreten. Einige sog. persistierende Apophysen sind auf paradiskale Defekte zurückzuführen. Es mag auch echte persistierende Apophysen geben, d. h. solche, die auf einer Fehlentwicklung beruhen.

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