Pediatr Radiol (1994) 24: 418–424

Pediatric Radiology

Punctate epiphyses: a radiological sign not a disease

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Abstract. Punctate epiphyses are caused by a diverse group of conditions. They may be an inherited part of certain bone dysplasias or an incidental finding occurring occasionally in various disorders. The pattern of the puncta together with other radiologic findings aid in making the correct diagnosis.

Punctate epiphyses have small calcifications in the cartilage in the ends of bone and around the spine, which are present at birth in a variety of conditions. Originally, they were considered to be a sign of a single bone dysplasia – chondrodysplasia punctata. Subsequently, two different forms were identified, one inherited as autosomal dominant, the other as autosomal recessive. It is now known that there are many more forms of chondrodysplasia punctata. Also, there are many other causes of punctate calcifications in disorders other than bone dysplasias. Some of the causes of punctate epiphyses are listed in Table 1. Localization and extent of the puncta as well as the anatomical changes associated with them are of value in the diagnosis of these various conditions. Some helpful diagnostic signs of various forms of chondrodysplasia punctata include short humeri, seen in the recessive form of chondrodysplasia punctata, polydactyly, seen in the X-linked dominant form, and calcification as well as puncta, primarily in the patellae, seen in the Zellweger syndrome.

Frequently the puncta are associated with retarded growth of the involved bone [1]. In the foot there may be puncta instead of tarsal ossification centers (Fig. 1). Puncta usually disappear by 3–5 years of age (Fig. 1). Subsequently, the diagnosis of these conditions becomes much more difficult as it can be based only on secondary signs. The effects of the puncta on growth of the bones include a delay in ossification of a bone or ossification center, decreased growth of bones, angulation of bone ends, deformity of bones, and cartilaginous rests within the bones (Fig. 2). They also have an effect on other cartilaginous, non-bony structures resulting in a small nose and lack of tracheal growth. A small nasal septum has been seen in rats treated with warfarin and a vitamin K deficiency [2] and this parallels some of the nasal changes in children of mothers receiving warfarin during gestation. The lack of tracheal growth may be a significant finding in the follow-up of these children. As they get older the calcified trachea does not grow in proportion to the lung, resulting in an insufficient supply of air to the lungs [3].

Table 1. Causes of punctate epiphyses

Bone dysplasias	Conradi-Hünermann CDP (XAD)
	Rhizomelic CDP (AR) (peroxisomal disorder)
	Brachytelephalangic type (X-R/X_p-)
	Mesomelic metacarpal type
	Pacman dysplasia
	Sheffield type
	Atypical forms
Other genetic disorders	Zellweger syndrome (peroxisomal disorder)
	Child syndrome
	GM-1 gangliosidosis
	Trisomy 21
	Trisomy 18
	De Lange syndrome
	Smith-Lemli-Opitz syndrome
Vitamin K disorders	Warfarin embryopathy
	Vitamin K reductase deficiency
Other conditions acquired in utero	Fetal alcohol syndrome
	Hydantoin exposure
	Febrile illness
	Phenacetin intoxication
Conditions that can be confused with puncta	Amelia and other absence deficiencies
	Cerebro-costo-mandibular syndrome
	Metachondromatosis
	Calcifying arthritis

AR, Autosomal recessive; CDP, chondrodysplasia; XAD, X-linked autosomal dominant

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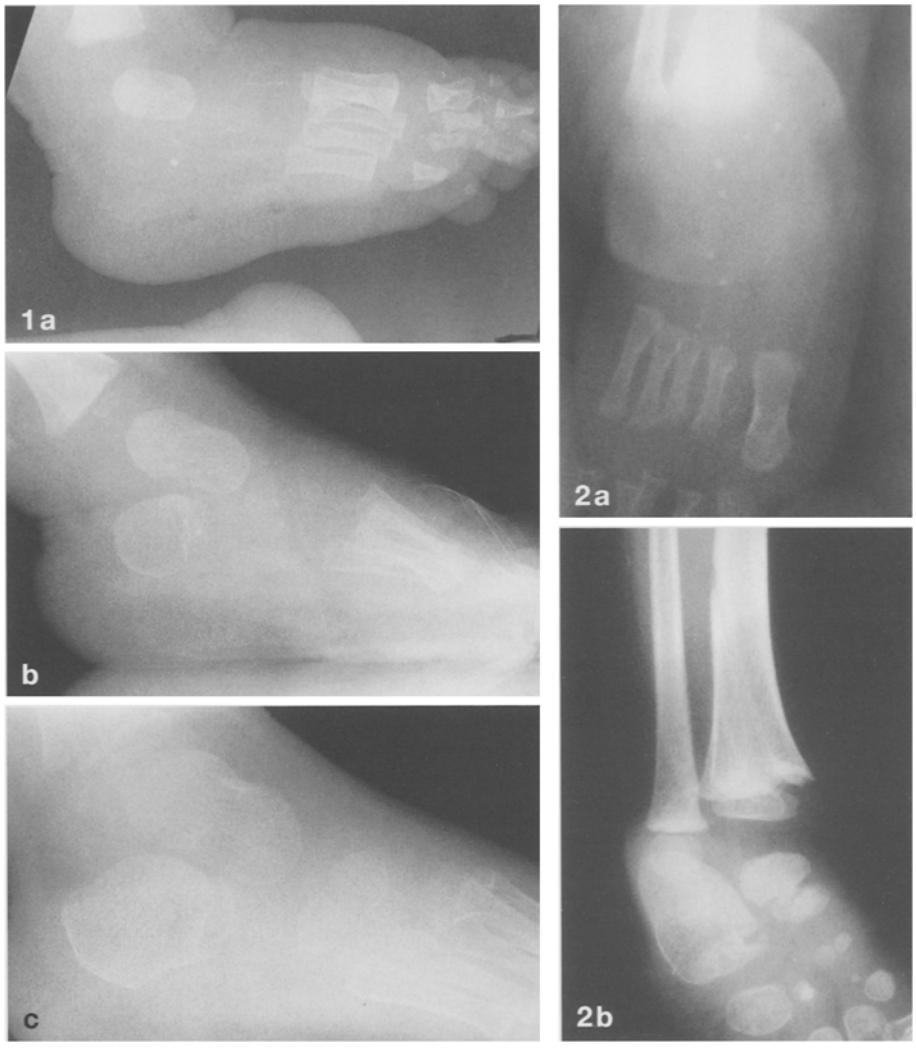

Fig. 1a–c. Chondrodysplasia – metacarpal type.
a At birth there is no calcaneal ossification center and only puncta are present.
b, c As the infant gets older a deformed, shortened calcaneus forms and the puncta disappear

Fig. 2a, b. Metaphyseal cartilage rests resulting from punctate epiphyses.
a At birth multiple puncta are present instead of ossified calcaneus and talus centers.
b On follow-up there is a lucent streak in the metaphysis and the tarsals are now ossified but markedly deformed

Conradi-Hünermann-type chondrodysplasia punctata (CHCDP)

Although this entity is well defined radiologically, it is somewhat heterogeneous in that there are both autosomal dominant and X-linked dominant forms. There may be asymmetric shortening of the limbs, although in the neonate the limbs may be symmetric. The facial appearance is characteristic with a very flattened face due to malar hypoplasia with a very small nose and a flat nasal bridge. The forehead is prominent with wide-set eyes and a mongoloid slant of the palpebral fissures. Cataracts are seen in 18% of patients [4]. The infants often have skin lesions which may include ichthyosiform erythroderma or systematized atrophoderma mainly involving hair follicles and circumscribed alopecia [5]. Contractures are common and a variety of foot deformities may be seen. The infants are short at birth and remain so during childhood. Contractures of the hip and knees may be seen. Some patients with CHCDP die in infancy, though most survive. This may be related to the fact that this is a heterogeneous group with some forms having an inherited as autosomal dominant mode of inheritance while in others the inheritance is as X-linked dominant [5, 6]. The latter forms occur only in girls, being fatal in boys. Patients with the X-linked dominant form appear to have a better survival rate than those with the autosomal dominant form.

Radiologically multiple areas of puncta are seen in the region of both the epiphyses and of unossified bones, such as the carpals and tarsals (Fig. 3). Some of the calcifications appear to be unrelated to the cartilaginous centers and may be extra-osseous. Puncta can involve the spine, including the sacrum, and sometimes the cartilage of the trachea. They often result in asymmetrical shortening of the long bones. The small tubular bones may also be affected. Scoliosis may occur due to asymmetry in leg length, or it may be due to vertebral anomalies. Talipes equinovarus is commonly present. Polydactyly has been seen only in the X-linked dominant form (Fig. 3b) and may be a helpful sign to separate it from the autosomal dominant form [5, 6].

Rhizomelic chondrodysplasia punctata

This is an autosomal recessively inherited condition. Infants usually have symmetrically shortened limbs with

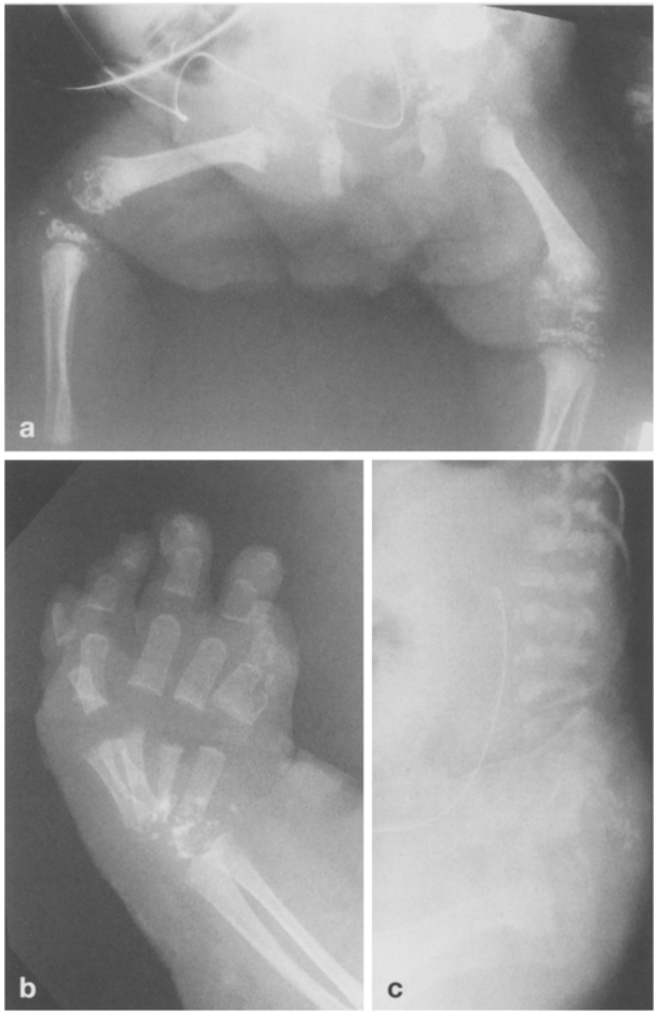

Fig. 3a–c. Chondrodysplasia punctata – Conradi-Hünermann form (X-linked dominant). **a** Multiple puncta are seen in the knee and hip epiphyses as well as around the pelvis. **b** There are multiple puncta in the wrist. There is also post-axial polydactyly. **c** Multiple puncta are present around the spine

the most marked shortening in the upper arms. They usually have cataracts and they have neurological degeneration [7, 8]. They have a high forehead and low nasal bridge. Cataracts are seen in 75% of patients [7]. They have full cheeks giving them a chipmunk appearance. Skin changes are much less common in rhizomelic chondrodysplasia punct (RCP) than in the Conradi-Hünermann form. The disorder is associated with abnormalities of peroxisomes [9–11]. RCP is often fatal in infancy, but survival into childhood has been reported.

A useful radiological finding is the presence of coronal clefts in the lumbar and thoracic vertebrae as seen in the lateral projection (Fig. 4a). These clefts are most clearly seen in the young infant and usually disappear with age, although sometimes there may be a small trace of cleft remaining. The shortening of the long bones is primarily rhizomelic and is most noticeable in the humeri (Fig. 4b). The femora may be shortened but usually not as much as the humeri (Fig. 4c). The metaphyses are usually flared. Puncta are less common in RPC than in the Conradi-Hünermann form. They may involve the proximal ends of the humeri and may occur in patellae and other bone ends as well. In childhood there may be demineralization, which is probably secondary to the neurological changes. Cervical kyphosis has been reported with compression of the spinal cord (Fig. 4d). This may be secondary due to laxity caused by the neurological manifestations. On MRI, migrational disturbances in the brain may be seen. As the infants get older the puncta disappear.

Chondrodysplasia punctata – metacarpal type

This also is a heterogeneous group of disorders with short metacarpals and shortening of some of the long bones. It includes the tibial metacarpal type reported by Rittler et al. [12], the humeral-metacarpal type described by Borochowitz [13], and the mesomelic dysplasia with punctate epiphyses described by Burck [14]. Some of the cases described by Sheffield et al. [15] also have similar manifestations. All these are named for a specific long bone but there is overlap in the long bone involvement, and in all cases there are short metacarpals. The children affected have a hypoplastic midface, a depressed nasal bridge, small mouth, micrognathia, short neck, and short limbs. In the series of Rittler et al. [12] the children were 2–4 standard deviations below normal in height. The psychomotor development in these children is normal and no skin changes or cataracts are seen.

Radiologically, patients are characterized by short metacarpals, particularly the third and fourth, but other metacarpals may also be affected. The proximal phalanges of the second digits may be shortened as may the limbs. The tibias were shortened in the series reported by Rittler et al. [12] with associated relatively long fibulas. One case also had forearm shortening. The ulnas were shortened in the series described by Burck [14] although one of her cases showed short tibiae. Some humeral shortening was seen in the Borochowitz series [13]. There is much overlap of findings in the long bones involved in the cases reported in these three papers; the main radiographic finding they have in common is short metacarpals (Fig. 5a). Coronal cleft vertebrae are seen, which close with age (Fig. 5b). Very few puncta are seen in this condition, and they are primarily in the sacrum and the carpals and tarsals. This suggests that this may not be an actual bone dysplasia but in syndrome with puncta.

Brachytelephalangic type of chondrodysplasia punctata

This form was originally described by Maroteaux [16]. It represents a definite clinical entity with X-linked recessive inheritance and is thus seen only in males. The infants affected have a very small nose with nares that are anteverted and grooved. The appearance is similar to that seen in Binder maxillofacial dysostosis. The patients described by Maroteaux had no significant clinical problems. A similar patient was described by Curry

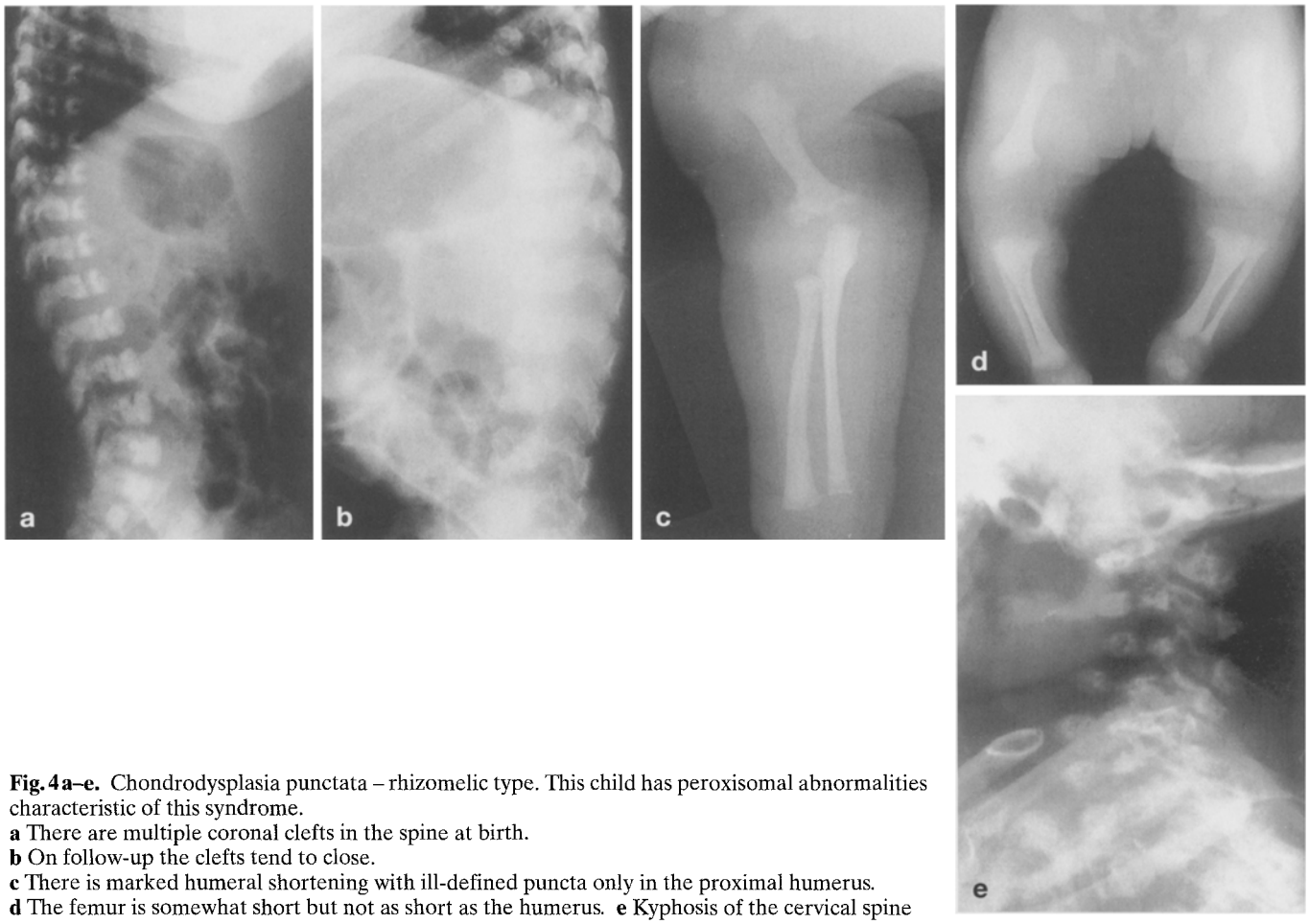

Fig. 4a–e. Chondrodysplasia punctata – rhizomelic type. This child has peroxisomal abnormalities characteristic of this syndrome.
a There are multiple coronal clefts in the spine at birth.
b On follow-up the clefts tend to close.
c There is marked humeral shortening with ill-defined puncta only in the proximal humerus.
d The femur is somewhat short but not as short as the humerus. **e** Kyphosis of the cervical spine

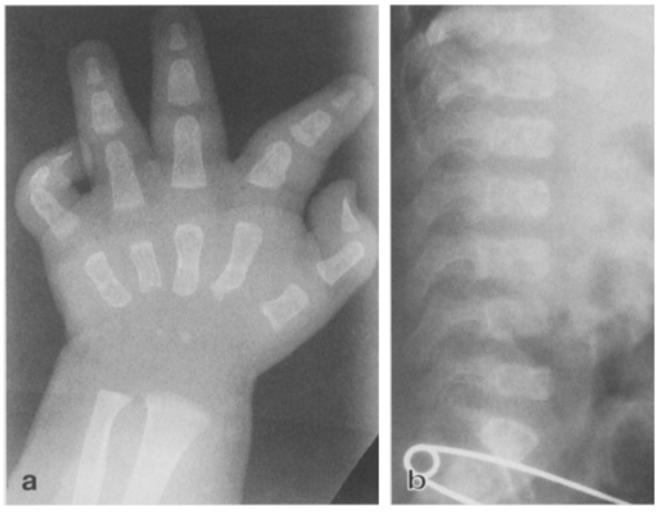

Fig. 5a,b. Metacarpal type of chondrodysplasia punctata (in the same child as in Fig. 1). **a** Shortening of the metacarpals and the proximal phalanx of the index finger. **b** Coronal clefts in the spine which are partly closing at this time

[17], but that patient had ichthyosis and was mentally retarded. Curry's patient had a deletion of the terminal X_p, whereas the karyotype in the Maroteaux cases was normal. In Maroteaux's cases the prognosis was relatively good, and it is felt that the patients had a benign type of chondrodysplasia punctata. Some of the patients described by Sheffield et al. had a very similar facial appearance to patients with the brachytelephangic form and may have been affected by this disorder [15]. Other cases of chondrodysplasia punctata with an X_p deletion have been described [18–20] with more severe findings than in the original Maroteaux cases. Ballabio et al. [18] also reported on an infant with a terminal deletion of X_p in whom the findings were similar to those recorded by Maroteaux. His patient had steroid sulfatase deficiency in the fibroblasts and also had associated ichthyosis. Wulfsburg et al. [21] also reported on a boy with short stature, developmental delay and hypoplasia, in all of which he was similar to the Maroteuax cases, and shortening of the distal phalanges. He also had ichthyosis. He had an inherited X-Y translocation associated with steroid sulfatase deficiency.

Distal phalangeal hypoplasia is the most characteristic radiological sign of this form of chondrodysplasia punctata. The distal phalanges of the fingers have a triangular appearance with the apex proximal. Eventually, as the child gets older, the phalanges appear short with irregular metaphyses and wide epiphyses. Such cases were reported by Lawrence et al. [22]. The phalangeal anomaly is a useful diagnostic sign of this disease in

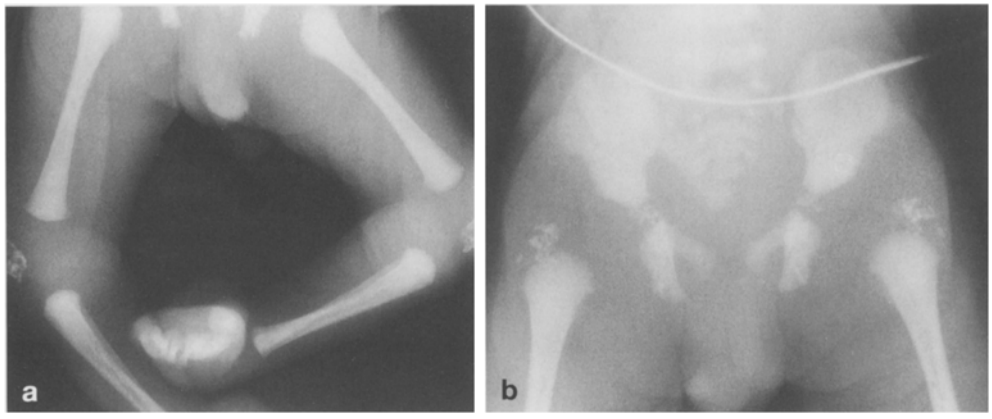

Fig. 6 a, b. Zellweger syndrome. **a** There are multiple puncta in the patellae. No puncta are seen in the knee epiphyses. **b** Few puncta are present in the hips and pelvic region. In other cases only the patellae may be affected

children over 3 or 4 years of age when the puncta have disappeared.

All of the cases described by Maroteaux were in males, so the hypothesis was that this is an isolated mutation of the X_p localized gene.

Sheffield-type chondrodysplasia punctata

Sheffield et al. [15] described a heterogeneous group of patients with punctate epiphyses who had puncta particularly in the calcaneus and spine with a benign course. In four of their cases there was evidence of distal phalangeal hypoplasia. These cases probably included some that have recently been described as a brachytelephalangic type. Others had coronal clefts and can probably be classed among the metacarpal type. Thus, the Sheffield type is probably not a specific entity but rather a heterogeneous group of disorders that can be included with other entities.

Pacman dysplasia

Shohat et al. [23] described a single case of peculiar bone dysplasia with stippling in many areas and short bowed bones with periosteal cloaking. On bone histology, osteoclasts had an unusual appearance reminiscent of the Pacman figures in computer games.

Radiologically, the entire coccygeal and sacral regions were replaced by stippling. There was dense stippling in the thoracic region as well. The epiphyses of the proximal femur, talus, calcaneus, and cuboid, as well as the bones of hand, showed considerable stippling. There was wide periosteal cloaking of many bones and poor ossification. The femora were bowed, but other bones less so. There was superior inferior sagittal clefting in the AP views in the upper spine.

Atypical forms

Many atypical forms of chondrodysplasia punctata have been reported that do not fit well into the standard patterns [22]. Cases with unilateral involvement have been seen; in others only a single ray of the hand is involved. Yet another case had marked shortening of the femora.

Zellweger syndrome (cerebro-hepato-renal syndrome)

This is another peroxisomal disorder, like rhizomelic chondrodysplasia punctata, that has associated puncta. Affected infants are severely hypotonic and have a characteristic facies with a high forehead, hypertelorism, epicanthic folds, and shallow supraorbital ridges. They may have Brushfield spots. Club foot deformity is common. The condition is inherited as an autosomal recessive. The infants usually die early in infancy. Many other body systems are involved, particularly the brain and kidneys.

Radiological findings include the presence of many puncta, which are particularly common in the patella [24] (Fig. 6). Patellar puncta also occur in other forms of chondrodysplasia punctata, particularly the rhizomelic form, but are much less frequent. If they are seen primarily in the patella, the diagnosis of Zellweger syndrome should be considered. Another radiologic finding is cortical cystic disease in the kidneys. Cysts may be present even when there are no patellar calcifications [25]. The bell-shaped thorax that is seen in affected children may be related to the flaccidity [26]. Brain manifestations include migrational disorders which are diagnosable using MRI.

Warfarin embryopathy

Maternal use of warfarin or other coumarin derivatives during pregnancy produces a bone dysplasia that is similar to chondrodysplasia punctata [27, 28]. It is most similar to the brachytelephalangic type, both in the clinical manifestation and in the radiological findings. In both these conditions the appearance of the nose and calcification of trachea are very similar. Some calcification may be present in the nose, and nasal calcifications have been reproduced in animals [2]. The exposure to coumarin derivatives such as warfarin between the 6th and 9th weeks of gestation produces the embryopathy [28].

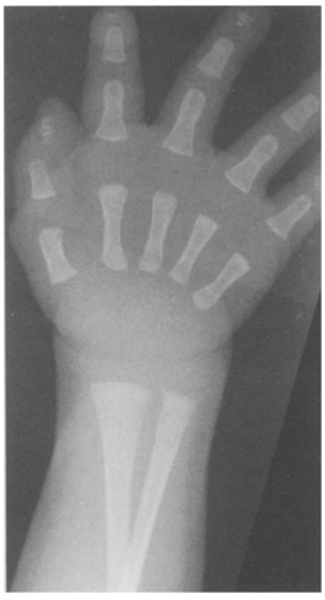

Fig. 7. Warfarin embryopathy. Note the markedly shorter distal phalanges with a triangular shape

Radiologically, stippling is seen in bone ends and around the spine. It may be indistinguishable from that found in chondrodysplasia punctata. The distal phalanges are very short, often shaped like an inverted triangle with the apex proximally, very similar in appearance to the hand of the brachytelephalangic type (Fig. 7). The short distal phalanges are apparent early in gestation. They have been seen in a 17-week abortus [29].

Multiple vitamin K-dependent coagulation factor deficiency

An appearance similar to warfarin embryopathy has also been seen in association with congenital deficiency of multiple vitamin K-dependent coagulation factors [30]. On the radiographs there was mild stippling in the perilumbar and perisacral region, and no other areas of stippling were present. In the hands, marked hypoplasia of distal phalanges of all the fingers were seen, very similar to that seen in warfarin embryopathy. With growth the distal phalanges remain quite hypoplastic.

In Summary, punctate epiphyses are seen in a variety of disorders, as listed in Table 1. Differentiation of the various forms depends on the pattern of clinical and radiologic abnormalities. Prenatal diagnosis is possible by recognition of the facial abnormality or limb abnormalities in some cases [31]. Postnatally, diagnosis is easiest in the first 2 or 3 years; when the puncta disappear it may be more difficult. Not all puncta are due to a bone dysplasia. Puncta in the tarsal bones only are seen in many other disorders such as fetal alcohol syndrome [32], the trisomies [33] as well as most of the nonchondrodysplasia syndromes listed in Table 1.

Acknowledgement. My thanks to Sally Gartman for typing the manuscript.

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