

Rickets

Uptodate: Clinical Nutrition

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Produced by Trevor A Scott.

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Black and white Duration: 00:24:47:19

00:00:00:00

<Opening titles, including short sequence showing an Asian man in a turban holding a small baby>

<Professor CF Stroud over further shots of Asian man with baby whilst a doctor demonstrates one of the effects of low blood calcium on the baby's hand reflexes>

The baby who you're looking at on your screen at the moment is very significant for two reasons. Firstly, he is, as you can see from the father if not from the baby, an Asian child. And secondly because the doctor is just about to demonstrate a rare, but nevertheless interesting, physical sign which shows that the baby has a low blood calcium. The sign which is being demonstrated is the carpal part or carpopedal



spasm in which, if the venous return from the arm is obstructed, the muscles go into spasm causing extension of the phalangeal joints and flexion at the carpophalangeal joints. And you can see that happening at the moment as the cuff is blown up and the blood returned from the arm is obstructed. The fingers are now extended because of spasm of the extensor muscles and flexed at the carpophalangeal joints because of spasm of the flexor group of muscles in the forearm. The reason for this baby having a low blood calcium is because the child also has rickets. I would stress this also is a rare sign, not one that we see very often but I think it is an interesting one by which to introduce our programme on nutritional rickets.

<Stroud to camera, then over still photographs of children with different degrees of severity of rickets, then back to camera>

Rickets is, of course, a very traditional disease in paediatrics. This is because in the 19th century it was very common indeed and this high prevalence of rickets in the community continued right into the 1920s. Indeed, Frederick Sterling, in the book which he wrote in 1924 said that 44% of children under 3 brought to hospital in London suffered from rickets, while John Thomson in Edinburgh, amongst children of the same age, found levels of rickets, the prevalence of rickets to be over 50% in children of that age going to hospitals in Glasgow. It's rather strange that at that time nobody knew the cause of rickets though now it seems very obvious for us to say it is due to vitamin D deficiency.

The rickets seen at that time also was very severe, and this was well shown in a photograph which was taken by Dr Harriet Chick, Dame Harriet Chick, who was the person who first showed that ultraviolet light cured rickets and who led the way to the discovery of vitamin D. These children were children whose photograph was taken in Vienna in the 1920s about the same time as Still wrote his book, and as you see their deformities are really gross. Nowadays the degree of deformity you see in nutritional rickets in children of the same age is rather less. And in this photograph you see the rather mild knock knee or genu valgum and, of course, by the time this deformity is seen by the parents and doctors nowadays, the condition of rickets is identified, diagnosed and treated. We do, however, have the very interesting situation that in



our country, rickets is almost entirely seen among some of the immigrant populations. It began to be reported around about the 1960s, both in London and Glasgow, and it was, with interest, that we noticed that most of the children we were seeing with rickets were from immigrant communities and now it has become quite obvious that they are nearly all from the Asian community.

For this reason we thought we would go to Hillingdon where Dr Tucker, with the help of Dr Haas, looks after a large Asian community and it was not with very much difficulty that Sam Tucker was able to produce for us a few cases of rickets, including the one you have already seen, but also an older child which we can use to demonstrate to you some of the clinical signs of rickets by which we make our clinical diagnosis.

00:05:44:00

<Short film featuring staff and patients at the Hillingdon Clinic. Opening scene shows Dr Sandhu sitting at a table with Dr Haas, a female infant and her father>

<Sandhu>

And the next child is 20 months old and called Binda Bhatti[?] and she, in fact, only came to the country a year ago and soon after that was found to have clinical rickets in the clinic, or rickets was suspected by the doctor who saw her in the clinic, and was sent up to your clinic and Dr Haas will tell you what we found.

<Dr Haas over shots of the girl's face, a photograph of her hands, then Haas shown seated at table reading from notes with girl in background>

That's right, she actually presented here at the age of 20 months to our clinic with clinical signs of rickets. The actual signs were an open anterior fontanelle, some swelling of the wrists distally, some evidence of a rickety rosary which could be felt and quite a lot of bowing of the tibia. There was no spinal deformity at that time, she



also clinically was anaemic, quite pale. The test confirmed our clinical impression. The haemoglobin, in fact, was not as low as one might have thought, it was 9.6, but very much an iron deficiency anaemia with a very small MCV of 55 and the biochemical picture of classic rickets with an alkaline phosphatase of 65.1 and a calcium, in fact, which was not all that low about 2.39. The X-rays I'm not going to be able to show you are as good as you're going to see as examples of this problem.

<Camera pulls back to show opening scene of doctors and infant with her father seated at the table>

So she has a combined nutritional problem of iron deficiency anaemia, which is extremely common in these children, and classical rickets.

<End of film clip>

<Stroud to camera, then over x-rays of the legs and hands of a child with rickets before and after vitamin D>

Dr Haas mentioned that this child had abnormal x-rays and, indeed, if we look at the x-rays of a child with rickets we can see what gives rise to this swelling of the wrists and to the bowing of the legs. If you look at these x-rays of fairly severe nutritional rickets in a child, you will notice that the area between the diaphysis and the epiphysis of all the bones is very much increased. And particularly around the knee there's a great fuzziness at the end of the diaphysis of the femur and the tibia and at the ankles, in addition to an inward bending of the ankles, there is cupping of the ends of the bones. And the reason for this is that owing to the lack of vitamin D, calcium for various reasons is not available and it is very difficult for the body to lay down calcium in the developing cartilage which is the process by which bones grow. As a result there is a great hypertrophy of the cartilage which is the cause of the bumps which occur at the wrist and the knees and at the ankles in these children.

When vitamin D is provided, as we can see in the next x-rays, on your left as you look at the screen is the x-ray of an older child with rickets and you notice that the



end of the ulna is cupped and there is a widening of the epiphyseal line due to the difficulty the body has in laying down calcium. Whereas, on your right, as you look to the screen is the same child after a period of vitamin D therapy, which obviously went on for a long time because there has been development of bone age, but also you'll notice that the ulna is now normal, the epiphyseal line is thin and there is the normal emphasis of the line at the end of the diaphysis. So that healing has taken place as a result of this child receiving vitamin D.

Well, obviously the most important thing to decide in any child with rickets, if we find that the changes are due to a lack of vitamin D, the most important thing to find out in every child with rickets is: why is this child short of vitamin D? And therefore, we have to make an enquiry about all sorts of things including the child's diet and the child's cultural way of life.

<Back to short film and earlier first scene, now camera focused on child's father talking to Sandhu>

<Child's relative speaks, in Punjabi>

<Sandhu>

In fact he says that she did have some meat and a small amount of omelette and some milk before, but the amount she eats has increased since she's been treated. It's difficult to assess how much she was getting.

<Haas, off camera>

Well, obviously not enough.

<Relative mutters to Sandhu, she translates>

He says her health has improved remarkably.



<Haas off camera>

She's certainly much happier.

<End of film clip>

00:11:24:00

<Dr J Stephen to camera, then over table listing amounts of vitamin D in commonly eaten foods>

We heard Dr Sandhu talking to the child's father about her diet and this was very important from the point of view of her anaemia. But when it comes to her rickets we have to remember that there is very little vitamin D in our diet, not only in Asian diets but in all our diets and the table shows some of the amounts of vitamin D in the more common foods that we eat.

Cod liver oil contains a lot of vitamin D, but of course this can't be regarded as a food, it is only taken as a supplement. Herrings, kippers, sardines or any kind of fatty fish contain quite a bit of vitamin D. Eggs, butter, liver are not very good sources but contain a little. But milk, even in the summer months, only contains 0.5 of a microgram per 100 grams and this is a very small amount and this is a very common mistake that milk is a good source of vitamin D and it is very often thought that this is where we get it from. The last three foods on the table, margarine, milks or infant foods and some yoghurts are actually fortified with vitamin D and have it added artificially so that margarine is a very good source of vitamin D and better than butter.

So, since there are these very small amounts in our diets, where do we get our vitamin D from, and why don't the rest of us suffer from rickets?

<Stephen, standing, refers to chart showing how vitamin D from either diet or sunshine is metabolised by the body>



Well, there is one other very important source of vitamin D and that is by the action of sunshine on the skin. The sunshine acts on a substance called 7-dehydrocholesterol which is a pre-cursor of vitamin D. So that whether vitamin D comes either from the diet or from the skin, it is then transported in the blood to the liver where it forms a substance called 25-hydroxycholecalciferol, this is then transported further to the kidney where another hydroxyl group is added and dihydroxycholecalciferol is formed. There are several of these dihydroxycholecalciferols, but the one that we're interested in, which acts on rickets and helps to cure rickets, is called 1-25dihydroxycholecalciferol where the hydroxyl group has been added in the 1 position. Now, when the blood calcium level falls, which is very important for the body, the parathyroid gland is stimulated to produce parathyroid hormone which then stimulates the kidney to produce this particular 1-25-dihydroxycholecalciferol. This, in turn, acts on bone to mobilise calcium from the bone, and it stimulates the absorption of calcium from the intestine so that there is more calcium available for the blood, the blood calcium rises, this action of the parathyroid hormone is switched off and the secretion of this 1-25- dihydroxycholecalciferol is reduced. So that the mineralisation of bone can continue and the absorption of calcium in the bone goes back to normal.

00:16:39:00

<Stroud to camera>

Well Joan has explained to you how complicated is the metabolism of vitamin D. We should perhaps return to consider the most vulnerable community in our country, that is the Asian community, and say why should it be that they in particular are picked out for vitamin D deficiency.

There are three possibilities. One is that their diet is peculiar in that they eat a lot of things called chapattis which contain a large amount of a substance called phytate which bind with calcium in the gut and prevents it being absorbed properly. Secondly, as you have seen, applies perhaps to all of us, in their diet they lack vitamin D. And thirdly, perhaps because of their own cultural way of life and their clothing, living in a country where there is little sunlight, they are lacking the sunshine which would, in



fact, provide them with vitamin D through the skin. My own view is that it is a combination of the last two, together with the fact that many of the mothers don't speak English and don't take advantage of all the welfare services which are available to them.

However, any child in our country would develop rickets if he didn't get enough vitamin D and therefore we should look at what sort of children and what sort of people, doctors in general should pay particular attention to, to make sure that they're not dealing with cases of vitamin D lack. And I would pick out five major groups and I would add, once again, that particular care and attention should be paid to these groups in the Asian community.

The first group to be considered are pregnant mothers because recently, in Glasgow, Ford has shown rickets occurring in babies in utero who are being born to mothers who themselves are vitamin D deficient. And it is of interest that in the original work on rickets Mellanby found that he could not make baby rats develop rickets unless he had made their mothers vitamin D deficient beforehand. And so it is that all pregnant mothers should take adequate amounts of vitamin D and, in particular, pregnant mothers who come from the Asian community. Then, of course, naturally enough we come to newborn babies. It is the hope of all paediatricians in this country, even if not of all mothers in this country, that newborn babies should be fed for the first few months of life on breast milk. And reading the old books we're told guite often that rickets is rare in breastfed babies. You know from the list of vitamin D levels, vitamin D contents of various foods given to you by Dr Stephenson, that ordinary cow's milk is a bad source of vitamin D. But recently, Elsie Widdowson has found in human breast milk, vitamin D sulphate, which is a soluble form of a vitamin D-like substance and it may be, and it's perhaps too early to say yet, that this is the means by which breast fed babies obtain some protection against rickets and perhaps it is because Asian mothers in particular become vitamin D deficient while carrying their babies and while breast feeding that their breast milk is not as protective to their babies as is the milk of other people. This is the subject of a great deal of research at the moment and if one were too dogmatic it would certainly be possible to be wrong.



<Stroud over opening film of Asian man with baby, then back to camera>

Then thirdly, of course, the period of infancy. It is during this period that there is very rapid growth, a lot of calcium is needed and rickets is a disease of growing bones. And so, it's not surprising that the next group to be considered are the group of adolescents at the pre-pubertal spurt and, of course, in the Asian adolescent this is the time when the girl in particular is put into traditional clothing and the amount of ultraviolet light getting through to the skin is markedly diminished and perhaps a few years later she becomes a vitamin D deficient mother who will deliver a baby who may occasionally have rickets at the time of birth and who may well develop rickets as a breast fed baby at the age of 6 months as we have seen quite frequently. The fifth group I would mention is the group commonly known as the geriatric group, the group to which we're all heading ourselves, and we can't really talk about rickets in this group because their bone is not growing and rickets is a disease of growing bones.

<Stroud over illustration of two old women sitting on a park bench, then old couple – one with a broken leg, then to camera>

But certainly we know that perhaps because old people don't expose themselves to the sun and go out as much as they did when they were younger. Or perhaps because there's some alteration in the way that they hydroxylate the vitamin D precursors, we know that they are very susceptible to vitamin D lack causing osteomalacia and, indeed, some series of investigations have shown that old people who receive vitamin D supplements are less likely to get conditions such as fractured neck of femur than those who have a low intake of vitamin D in their diet.

00:22:20:00

<Stroud to camera>

Well, these are roughly the sort of points we should bear in mind whenever we look at a community for whom we have responsibility. But, more important than ever is



how do we prevent it? Certainly we can't make people with a deeply felt cultural belief in the way they should dress and behave alter the way they dress and behave and therefore there's no way of increasing their exposure and there's no way that I know of of increasing the amount of sunlight and therefore all we are left with is increasing the amount of vitamin D which gets into their body through their mouth. And we can't do that by altering their diet very easily unless we get them to take large amounts of margarine. We can, however, give them supplements of vitamin D. And it is, to my mind, a tragedy that for many years now certain groups of immigrant communities in our country have suffered from rickets, that the surveys done of these groups in some detail have always showed very high levels of biochemical rickets, if not obvious clinical rickets, and all of this could have been prevented if we could have got every member of those communities taking 3 or 400 units of vitamin D each day. And so, as doctors, I think we should recognise this as a major public health problem of our immigrant communities and try to make sure that all the people in these communities, especially the vulnerable groups I have mentioned, should be given each day a dose of vitamin D, up to about 400 units per day, because then I think we would very largely have solved the problem of rickets in our immigrant community.

<End credits over further scenes from short film at Hillingdon Clinic, Dr Sandhu speaking to a female holding a small infant>

<Sandhu speaks to female in Punjabi>

<Sandhu to Haas, off camera>

She sits fully clothed in the garden. You wouldn't find many Indian ladies sitting out in their bikinis, they would be fully clothed.

<End of film clip and final end credit>