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# Rickets Misdiagnosed as Arthritis-Primum Non Nocere.

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**ABSTRACT**

Vitamin D deficiency is becoming an important global problem. This case report is about a 16 year old girl with resistant pain and gradual weakness of both lower limbs of one year and inability to walk for six months inspite of steroid treatment after an initial diagnosis of Juvenile Rheumatoid Arthritis elsewhere. On presentation here, she had typical clinical picture and radiology of rickets with mild renal disease. She was put on oral calcitriol, calcium carbonate and was given weight relieving calipers .Her pain slowly got reduced and she started to stand and walk without any support. Rickets patient may present with weakness or multiple joint pains that it can mimic Juvenile Rheumatoid Arthritis. Hence there is a need to consider differential diagnosis and to re- evaluate the patient especially if there was no response to regular treatment. Magnesium added in patients who are resistant to vitamin-D therapy. Ignorance of this disease delays the diagnosis and worsens leaving permanent deformities.

**Keywords:** Hypo-vitaminosis D , Rickets Misdiagnosis, Rheumatoid Arthritis, re-evaluation

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## INTRODUCTION

Ignorance of disease manifestations often leads to a delay in the diagnosis. Sometimes such lapse can be serious enough to cause worsening of the symptoms leaving permanent deformities. We present one girl who had an unfortunate misadventure purely due to ignorance of common diagnosis.

**Case report**

On 18-10-12, a 16 year old girl was brought with complaints of pain and gradual weakness of both lower limbs of one year and inability to walk for six months. She was able to pass motion and urine only using a sitting type commode. She attained menarche one year back. She initially had opinion and treatment from a general practitioner and later she was seen by a neuro physician who referred her to a rheumatologist suspecting a Juvenile Rheumatoid Arthritis. She was put on steroids for a period of one year. Her condition worsened and thus she was brought to our centre.

During her examination she could not even stand without a support. She had bilateral genu valgus, knobbly wrists and stunted growth as seen in figures 1,2and 3. Her spinal examination did not reveal any specific tenderness. Her deep tendon reflexes were normal and the power of upper and lower limb muscles was normal. We clinically suspected rickets and multiple epiphyseal dysplasias. X-ray of her knees showed a gross genu valgus and wide splaying of the physeal plate. In the pelvis X-rays there was wide splaying of the upper physis of femur

**Figure1: Patient needed help to even stand. The genu valgum is obvious**

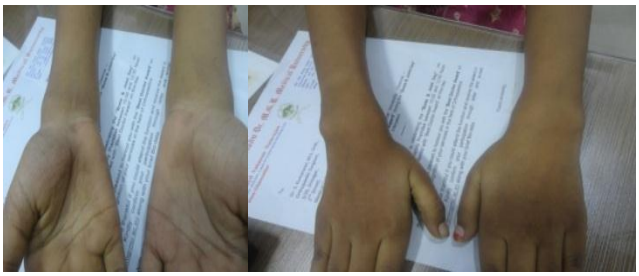

**Figure 2 and 3: Patient's wrist front and back showing the knobbly wrist.**

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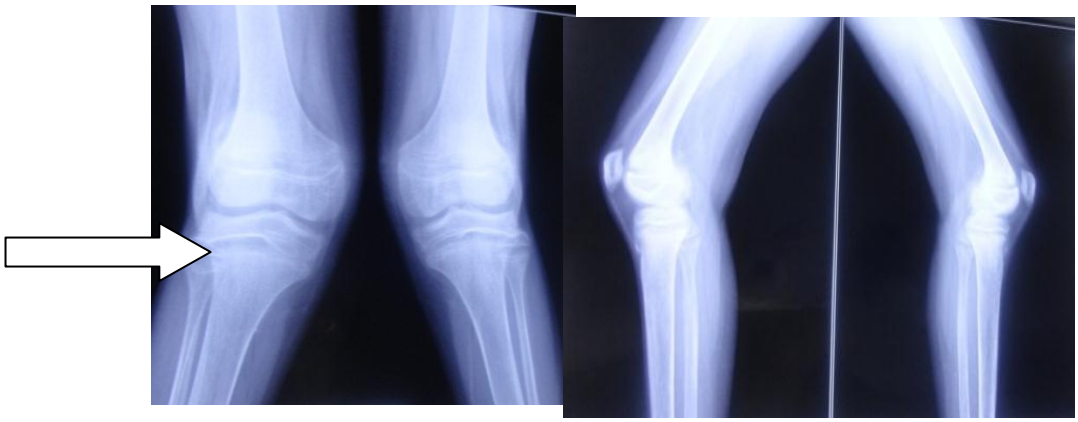

**Figure 4: X- ray of both the knees showing the genu valgum and widening of physis**

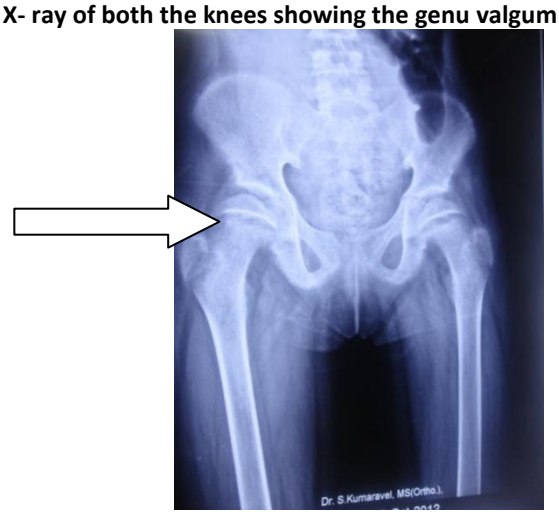

**Figure 5: The pelvis X- ray showing widening of physis marked by arrow.**

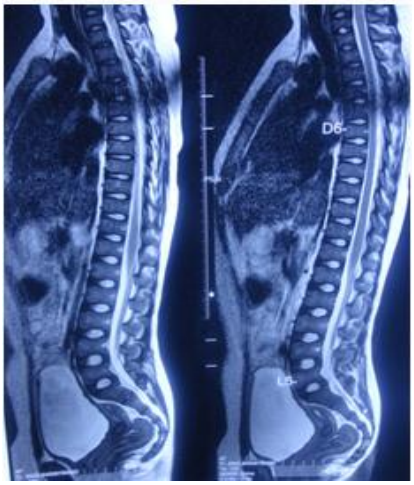

**Figure 6: Her MRI of spine showing the increased disc height simulating a cod fish appearance.**

MRI of her spine (figure 6) was nonspecific with increased disc height. The finding was widening of the disc spaces (typical cod fish appearance) exactly as described for osteomalacia. Figure 5. Otherwise there was no specific compression of neural elements. The curvature and alignment of the dorso lumbar vertebra were normal. There was no lytic or destructive lesion or infective or neoplastic lesion**.** There was no intra extra spinal mass or compression of thecal sac.

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Ultrasound of her abdomen showed bilateral chronic medical renal disease grade 2 with her kidney size towards the lower limit of normal; other organs were normal. In the blood investigations, her Erythrocyte Sedimentation Rate was normal; her vitamin D level was 3ng/ml her blood urea was 71.83mg/dl ; creatinine was 2.16mg/dl (both elevated); her ALP was 41.49; Creatine Phosphokinase was normal; intact parathyroid level was 731.4(14-72pg/ml) ;Serum Calcium 7.3mg/dl (8.5-10.1mg/dl );phosphorus 2.5 mg /dl (normal is 2.5-4.9mg/dl). Plain CT scan of thyroid gland showed normal density with no parathyroid adenoma detectable. She also had mild UTI and her urine culture grew E.Coli sensitive to Ciprofloxacin. She had nephrology, urology and neurologist opinions. After discussion with the nephrologist, she was put on oral calcitriol 0.25mg with oral calcium carbonate 500 mg two times daily, oral Nucarnit 500mg once daily, oral soda bicarbonate two times daily. She was advised one tablet of paracetamol if she had pain. She was given weight relieving calipers as out-patient.

After two months when she was reviewed, her pain slowly disappeared and she started to stand and walk without any support. Her intact parathyroid level reduced to 48.1(14-72pg/ml) and her serum calcium increased to 8.2mg/dl 8.5-10.1mg/dl, Phosphorus increased to 3.7 mg /dl (2.5-4.9 mg/dl) her vitamin D level was 21ng/ml. Her renal parameters were continuously monitored. Her protein was 6.78 g/dl and albumen was 4.35 g/dl. Later at five months follow up, she did not have any bone pain or local tenderness. She was able to stand un-aided and walk with support. After two years, she was still under drugs (latest follow up) she had no pain in her limbs and can walk on her own without any support and was doing her activities herself. She started to go to school independently. Her only problem was getting up from the ground level due to the genuvalgum. This is being planned for Macewan's osteotomy. She did not have any other disability. The drugs were prescribed by the nephrologist, the second author.

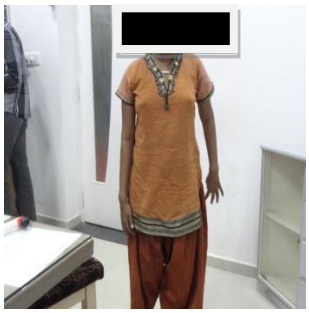

**Figure 7. Patient standing without any support**

## DISCUSSION

We are not sure if this girl had primary renal rickets misdiagnosed as arthritis or a separate steroid induced renal disease independent of rickets. The clear resolution of the paper is to consider differential diagnosis and to re- evaluate the patient especially if there was no response. For example the physician who first saw the patient might have re-thought his diagnosis after a few weeks of steroids itself, as the child did not respond to even high dose steroids which might have reduced the swelling and pain in Juvenile Rheumatoid Arthritis. The response of the disease to our treatment with vitamin D and calcium stopped us from proceeding to do a renal biopsy.Since the symptoms of osteomalacia like pain in the limbs and occasionally muscle weakness take these children to the neurologist whose apparently diagnosed joint conditions – e.g. as arthritis due to the widespread bone pain.

In the children with rickets, serum calcium and phosphorus are usually low and serum alkaline phosphatase is elevated reflecting the new bone formations in young children with the growing ends or physes are widened. Bone biopsy is usually not needed. Vitamin $D_3$ (cholecalciferol) which is easily absorbed form gut is selected as the dietary supplementation for achieving adequate levels of vitamin D. Such oral supplementations are found to be superior to unprotected exposure to sunlight which may predispose to skin malignancies. Vitamin D insufficiency is frequently seen in UK population. The findings differ according to their age e.g. bony deformities like that seen in rickets or hypocalcaemia as seen in infancy and childhood, and or

with pain in muscles and bone and weakness in adults. Interestingly apart from skeletal manifestations cardiovascular disease, type 2 diabetes, several cancers, and autoimmune conditions are related to vitamin D insufficiency. Factors that prevent the sunlight exposure to the skin can reduce vitamin D production in the body. These include clothing that covers the skin for religious purposes, more skin pigmentation, sunscreen, bedridden patients. Oral vitamin D may also be ineffective in mal-absorption, renal and liver disease, and with concomitant antiepileptic use. Usually an assay of serum 25-hydroxyvitamin D (25-OHD) is useful in clinching the diagnosis. Rickets and osteomalacia should be treated with high strength calciferol for at least 12 weeks, vitamin D supplements should be continued after this time.[1] Skin pigmentation is by natural selection to adjust levels of pigmentation to levels of UV radiation available at particular latitude. High UV radiation near the equator and led to the evolution of dark, photo protective, eumelanin-rich pigmentation. And in low level of UV radiation, to sustain cutaneous photosynthesis of vitamin $D_3$the skin pigmentation is less[2].

Wrickken means 'to twist', the word rachitic is possibly created due to the resemblance in sound. Soranus of Ephesus of Alexandria was the earliest person to describe clinical signs of rickets in Roman children [3].

If there is no improvement with calcium and vitamin D, Magnesium dependent Vitamin D-resistant rickets should be borne in mind. Two interesting cases of rickets associated with hypomagnesaemia are reported. These two children, one age two and the other age five, presented with typical rickets, had massive doses of vitamin D, i.e. 600,000 IU of Vitamin D daily for ten days. They did not have any improvement even after six weeks. These two children showed an excellent response to oral magnesium-chloride supplementation and the rickets rapidly resolved. Thus serum-magnesium levels should be measured in all cases of rickets and magnesium supplementation is thought, particularly in those patients who are resistant to vitamin-D therapy[4].

Hypo-vitaminosis D is rising again as a most important health crisis worldwide. If 75nmol /L of serum 25(OH) D levels were a reference level then all regions i.e. Asia, Europe, Middle East and Africa, Latin America, North America, and Oceania, were having insufficient levels of vitamin D. But even levels below 25 nmol/L are frequent in South Asia and the Middle East through a survey of published literature. Factors like extreme age, female sex, living away from the equator, longer winter and relatively less sunlight exposure, more melanin in skin, diet with poor vitamin D or food not fortified with vitamin D ,cause lower levels of 25(OH) Vitamin D[5].There is an advice from American Academy of Pediatrics (AAP ) to supplement in most children who consume milk, doubling the amount of vitamin D babies and children to 400 International Units (IU) per day[6].

Coming to the issue of misdiagnosing a case of rickets or osteomalacia, very rarely one sees such misdiagnosis. This is because of presentations of the patient with weakness or multiple joint pain that can simulate a Juvenile Rheumatoid Arthritis[7].Sometimes Lyme disease can be misdiagnosed as Juvenile Rheumatoid Arthritis[8].Children with acute lymphatic leukemia may present like juvenile chronic arthritis are presented. All had symptoms and signs for at least 4 months before leukemia was diagnosed and in two the full blood count was normal at presentation. The importance of a high index of suspicion is emphasized, particularly if the white cell count is low [9, 10].The exact diagnosis of leukemia is delayed as evaluation is concentrated on arthritis. Early institution of steroids can also not only delay diagnosis further but also reduce the response to chemotherapy. There is also an emphasis on early bone marrow examination if there are any atypical features of juvenile idiopathic arthritis and certainly before starting steroids or cytotoxic agents. [10]

## CONCLUSION

Basic mistakes in diagnosis cause grave residue to the patient. Awareness of common orthopedic disease manifestations is a must to pass the basic medical degree. There should be an element of soul-searching on the part of educationist. From the above deliberation we only imagine similar cases are possible in future. *Primum non nocere.*

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