Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/16321
DOI URL: http://dx.doi.org/10.21474/IJAR01/16321

RESEARCH ARTICLE

ADYNAMIC OSTEOPATHY: ABOUT A CASE WITH REVIEW OF THE LITERATURE

Tangara M.[1], Traore O.[2,4], Kouma A.[3],Toure A.[1], Kodio A.[1], Coulibaly N.[1], Djiguiba K.[1], Coulibaly M.[4], Maiga D[1], Dolo M.[1], Sidibé M.[1], Sangare N.[1], Coulibaly K.[1], Samaké M.[1], S.Y Seydou[1], Bagayoko O.L[4], Thiam A.[4] and Yattara H.[1]

1. Nephrology and Hemodialysis Department of the University Hospital Center of "Point G" Bamako-Mali.
2. Radiology Department of the University Hospital Center of "Point G" Bamako-Mali.
3. Radiology Department of the Hospital Center Mother-Child "Luxembourg" Bamako-Mali.
4. Radiology Department of the Medicalclinic "Marie Curie" Bamako-Mali.

Manuscript Info

Manuscript History
Received: 20 December 2022
Final Accepted: 24 January 2023
Published: February 2023

Key words:-
Adynamic Osteopathy, Renalfailure, Hypoparathyroidism, Sickle Cell Disease And Pregnancy

Abstract

We report the case of a 42-year-old housewife patient whowasadmitted for impairedrenalfunction at 1246 µmmol/l with a clearance of 3.07ml/min/1.73m2, with the aim of to show a rare case of adynamicosteopathy with review of the literature. The clinical examination on admission hadfound a patient altered, impotent, pain in bothlowerlimbs, dehydrated, pale, BP=170/90mmhg, temperature at 36°7, heart rate at 100bts/min. Biologically:anemiawasfound at 7.2 g/dl. Hyperleukocytosis at 15400/mm3. Creatininewas 1246 µmol/l, urea 49mmol/l, uricacid 850umol/l, parathormone 58.4 pg/l and vitamin D 16 ng/l. The reprovedcalcemiawas at 2.73 mmol/l, the phosphorus at 2.69 mmol/l, the bicarbonateemia at 13 mmol/l. Blood sugarwas 6.1mmol/l, ferritinemia 1800 ng/ml, Na+=137mmol/l, K+=4.3 mmol/l, Cl-=106 mmol/l, proteinuria 6.8g/24 hours ,leucocyturia at 40000/mm3, withouthaematuria, an E.coliurinary tract infection whichwas sensitive to ciprofloxacin. Our patient had a heterozygous AC sickle cell profile, and wascarrying an evolving single-fetalpregnancy of 15 weeks +5 days on ultrasound. On the cerebral scanner, wefound a semi-recentischemic stroke in the deepterritory of the left middle cerebralartery. The patient alsounderwent five hemodialysis sessions. A biological control aftertwomonths of treatment hadfoundserumcreatinine at 212 µmol/l, urea at 40.3 mmol/l, sodium at 132.9 mmol/l, serum potassium at 5.2 mmol/, bloodchloride at 91.6 mmol/l, Magnesium at 1.1mmol/l, serum calcium=3.1mmol/l, CRP at 21mg/l, White Blood Cells at 1200/mm3, Hemoglobinlevel at 6.6 g/dl. The VS was at 90-100 mm.Urinary volume at 680ml. Adynamic osteopathyis a rare and fatal pathologythatrequiresearly treatment.

Introduction:-

Adynamic osteopathyischaracterized by a decrease in the production of bone tissue, both in terms of its formation and itsresorption (destruction of the old tissue which must bereplaced by new). Bone involvement, referred to as

Corresponding Author:- Tangara M.
Address:- Nephrology and Hemodialysis Department of the University Hospital Center of "Point" Bamako-Mali.

"Mineral and Bone Disorders in ChronicKidney Disease," isconsistentlypresent to somedegree in any patient with chronickidney disease. It persists and tends to worsenduringdialysis treatment [1]. Osteopathies are complications in patients on long-termdialysis [2]. Calcium and phosphate play a major rolein bonemineralization, theyalso have multiple functions in the body. Althoughtightlyregulated, serum phosphate concentrations varythroughout life according to physiologicalneeds. On the other hand, ionizedcalcemiaismaintainedwithin a verynarrow range of values thanks to the combined action of two hormones, parathyroid hormone (PTH) and calcitriol, an active metabolite of vitamin D [3]. Chronickidney disease (CRD) inducesmineralmetabolismdisordersleading to bonelesions and vascular calcifications which affect its vital and functionalprognosis [4]. The aim of ourworkwas to show a rare case of adynamicosteopathy with review of the literature in the therapeutic management of thispathology.

Observation:-

We report the case of a 42-year-old patient, housewifewhowasadmitted for impairedrenalfunction at 1246 µmmol/l with a clearance of 3.07ml/l. The clinical examination on admission hadfound a patient altered, impotent, pain in bothlowerlimbs, dehydrated, pale, BP=170/90mmhg, temperature at 36°7, heart rate at 100bts/min. Biologically: anemiawasfound at 7.2 g/dl. Hyperleukocytosis at 15400mm3, leukocyturiawithouthematuria. Creatininewas 1246 umol/l, urea 49mmol/l, uricacid 850 umol/l, parathormone 58.4pg/ml and vitamin D 16 ng/ml. Calcium was 2.73 mmol/l, ferritin 1800 ng/ml, phosphorus 2.69 mmol//l, bicarbonate 13 mmol/l. Blood sugarwas 6.1 mmol/l; Na+=137 mmol/l, K+=4.3 mmol/l, Cl-=106 mmol/l, proteinuria at 6.8g/24 hours, withouthematuria, an E.coliurinary infection thatwas sensitive to ciprofloxacin . **(Table 1)**

Table I:- Biologicalassessmentcarried out on admission.

Biological examination	Results
Hemoglobin level	7,2 g/dl
Leukocytes	15400mm3
Creatinemia	1246 µmol/l
Urea	49mmol/l
Uric acid	850µmol/l
parathyroid hormone	58,4pg/ml
Vitamin D	16 nmol/l
Calcemia	2,7 mmol/l
Phosphorus	2,69 mmol/l
Bicarbonate	13 mmol/l
blood sugar	6,1 mmol/l
Na+	137mmol/l
K+	4,3 mmol/l
24 hour proteinuria	6,8grams/24 hours
Leukocyturia	40000leucocyte/L
Chlorine	106 mmol/l
Ferritinemia	1800 ng/ml

Our patient had a heterozygous AC sickle cell profile, and wascarrying an evolving single-fetalpregnancy of 15 weeks +5 daysaccording to an ultrasoundperformed. **(Figure 1)**

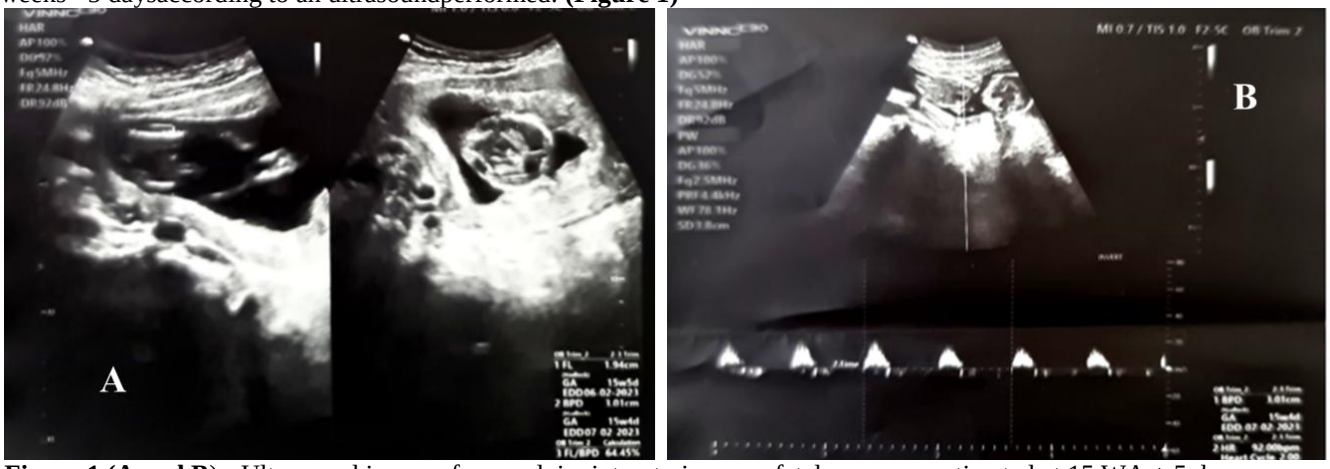

Figure 1 (A and B):- Ultrasound image of an evolvingintrauterinemonofetalpregnancyestimated at 15 WA + 5 days (weeks of amenorrhea).

On the abdominal ultrasound, bilateralrenal pain wasfoundwithoutabnormalities in the liver, spleen and gallbladder**(Figure 2)**

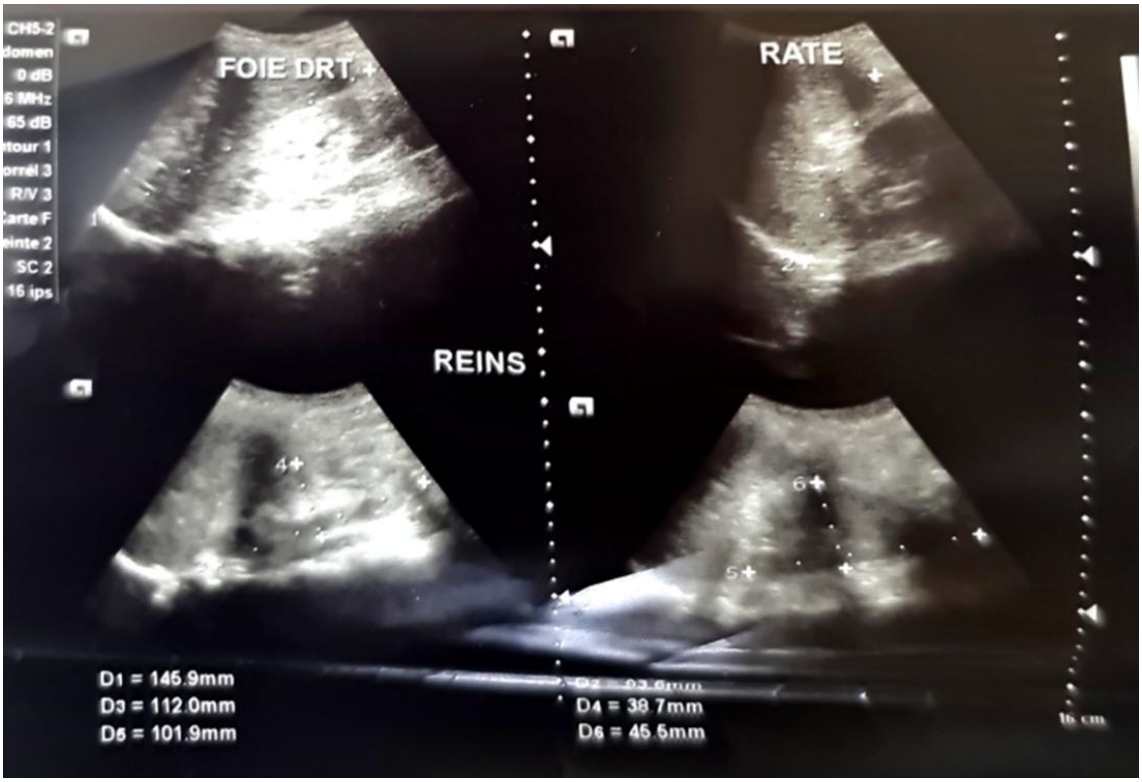

Figure 2:- Ultrasound image of bothkidneysshowingechogenickidneys with loss of its cortico-sinus differentiation suggestive of bilateralrenalsuffering.

On the cerebral scanner, wefound a semi-recentischemic stroke in the deepterritory of the left middle cerebralartery. **(Figure 3)**

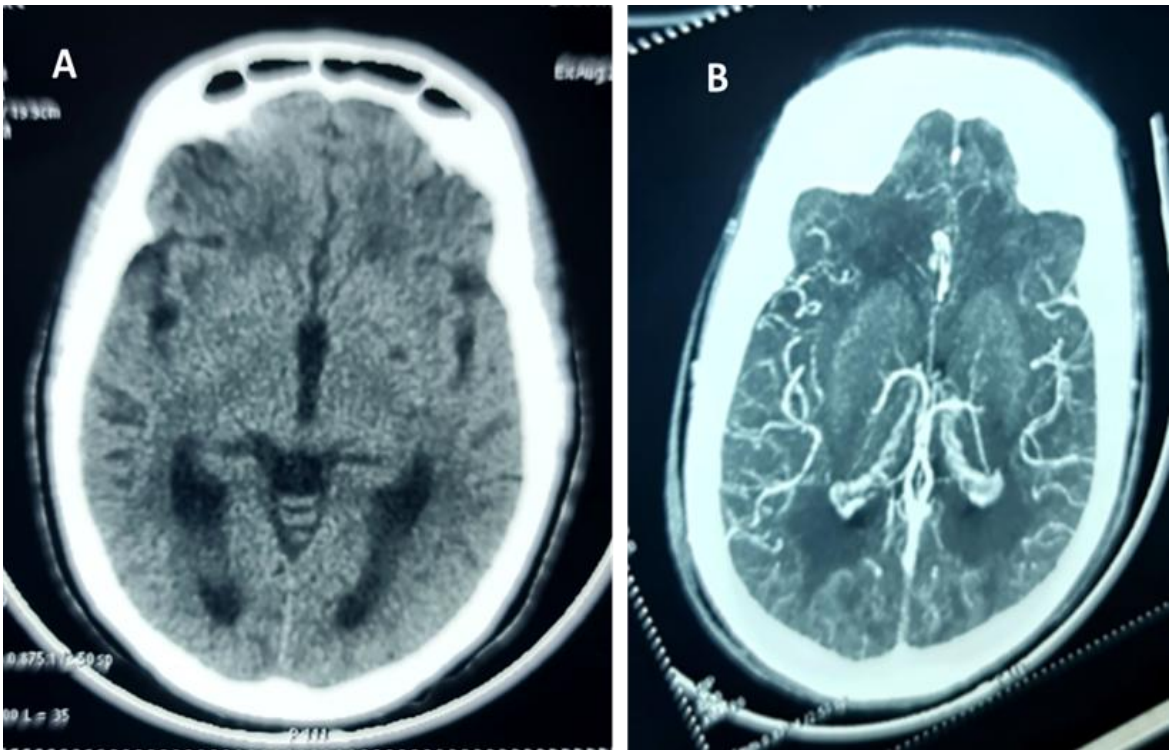

Figure 3:- Cerebral CT (A) with agiographic acquisition (B) showinglacunarleftlenticularhypodensity suggestive of an ischemiclesion of the territory of the deep middle cerebralartery.

A biological control aftertwomonths of treatment hadfoundserumcreatinine at 212 µmol/l, urea at 40.3 mmol/l, sodium at 132.9 mmol/l, serum potassium at 5.2 mmol/, bloodchloride at 91.6 mmol/l, Magnesium at 1.1mmol/l, serum calcium=3.1mmol/l, bicarbonatemia at 19 mmol/l, CRP at 21mg/l, White Blood Cells at 1200/mm3, Hemoglobinlevel at 6.6 g/dl. Proteinuria for 24 hourshadreturned to 0.42 g/24 hours with normal urinarysediment and diuresismaintained at 950 ml per 24 hours. **(Table II)**

Table II:- Biologicalassessmentcarried out after 2 months of treatment.

Biological examination after 2 months treatment	Results found
Creatinemia	212 µmol/l
Urea	40,3 mmol/l
Kalemia	5,2 mmol/l
Chloremia	91,6 mmol/l
Magnesium	1,1 mmol/l
Calcemia	3,1 mmol/l
White globule	1200mm3
Hemoglobin level	6,6 g/dl
Proteinuria	0.42 grams/24 hours

Discussion:-

A-Pathopathologicalmechanism:

Calcium and phosphates togetherform hydroxyapatite crystalswhich, deposited on the collagen matrix, ensure the texture of the bone tissue of vertebrates. Divalent calcium Ca++ isinvolved in nerve conduction, muscle contraction, coagulation, cell differentiation and intracellularsignaling**[1].** The phosphate anion plays a role in energy exchanges, certain enzymaticactivities, the synthesis of nucleicacids, the acid-base balance and the intracellular signal. Phosphocalcicregulationiscarried out by calcitonin, vitamin D and parathyroid hormone. Thesethree hormones have their point of impact in the intestines, kidneys and bonesthuscontrolling the entry, the exit, the stores of calcium and phosphorus**[3].** The onlyregulatory signal perceivedis the variation in serum calcium **[1,5].**Surgery and non-calcium derivatives are involved in the treatment of secondaryhyperparathyroidism**[6].**

B-Adynamic Osteopathy (Adynamic Bone)

Defined by a decrease in the rate of bone formation. There is a decrease in the formation of osteoid substance and mineralization. The osteoidborders are of normal or lowthickness**[7,8].** It ispresentin 15 to 50% of patients depending on the series. The main etiologywas, a few yearsago, aluminumosteopathy (aplasticbone), linked to bone intoxication with inhibition of calcium deposits on the osteoid matrix. It was due to the presence of aluminum in the hemodialysis baths or the use of alumina gels **[9].**Poisoningiscurrently rare since the introduction of non-aluminum calcium and non-calcium phosphorussequestrants. The accumulation of iron in the bonesisresponsible for a lowbone focus and a lowparathyroid hormone. The risk factors for adynamicosteopathy are currentlyage, corticosteroidtherapy, diabetes, peritonealdialysis, excessive suppression of parathormone secretion by calcium salts, 1-alpha hydroxylderivatives of vitamin D and severephosphorus restriction.Adynamic osteopathyisassociated with an increasedrisk of hypercalcemia and calcificationVascular. Histologically, the mineralized surfaces are collapsed, the rate ofmineralizationis normal or low, withoutanyincrease in osteoid surfaces, whichdifferentiatesitfromosteomalacia. There are few or no osteoblastsalong the bonytrabeculae and osteoclasticreabsorptionisalsoreduced or absent. This idiopathicadynamicosteopathy**[10],** like aluminumadynamicosteopathy, isaccompanied by normal alkalinephosphatemia and sometimes a tendency to hypercalcemiaundervitamin drifts or high doses of calcium carbonate. It isobserved in patients who have a normal or lowlevel of circulatingparathyroid hormone **[11].** Bone ironoverload can beassociated with adynamicbone**[12].**Diabetes and hypogonadism are othercontributing factors. Our patient diedfollowing a multiparametricattack and slow treatment.

C-Imaging:

In imaging, dual-photon absorptiometry (DXA) assessesbonemineraldensity (BMD). The National Institute of Health (NIH) defined, in 2000, new diagnostic criteria for osteoporosis, introducingbonequalitycriteria (microarchitecture, geometry and mineralization) in addition to the quantitative criterionrepresented by BMD **[7, 8]**.. New bone imaging techniques have therefore been developed, with the aim of exploringbonequality. Can wehope for an improvement in the therapeutic management of patients with CKD with these new tools and betterprediction of fracture risk, knowingthat 50% of hemodialysis patients will have at least one fracture **[7,9].**

Conclusion:-

Disorders of mineral and bonemetabolism are associated with a high risk of mortality and an increasedrisk of fracture in a context of reversible clinical and paraclinicaldisturbances. However, treatmentsexist, such as calcium salts, active derivatives of vitamin D, surgery and more recentlycalcimimeticswhichmakeit possible to control parathormoneemia. However, itis important to rememberthat excessive parathyroid hormone suppression shouldbeavoided in dialysis and predialysis patients, so as not to promote the development of adynamicosteopathy**.**

References:-

1.NK Man, Paul Jungers. Disorders of calcium phosphate metabolism and osteoarticularcomplications: July 14, 2007.

2.Mahamadou Diarra. Evaluation of the Treatment of End-Stage ChronicKidney Disease by HemodialysisfromJanuary 1 to December 31, 2008 in the Nephrology and Hemodialysis Department of the Point-G University Hospital: FMOS medicalthesis University Year 2008-2009 – Bamako-Mali No. 340

3.Marie Courbebaisse, Jean-Claude Souberbielle. Phosphocalcicbalance:regulation and exploration: Nephrology and Therapeutics 7(2011) 118-138.

4-Fabrice MacWay. Pathophysiology of renalosteodystrophy: Journal of Rheumatism 79S (2012) A18-A.
5.Ziad A. Massy and Tilman B. Drueke. Parathyroid hormone, simple hormone or uremictoxin? : Nephrology and Therapeutics 7 (2011) 1-4.
6.Geoffrey A et coll. Cinacalcet for Secondary Hyperparathyroidism in patients Receiving Hemodialysis. n engl j med 350;15 / www. nejm.org april 8,2004.
7-Panel Justine Bacchetta.Imaging in patient with chronic kidney disease: A new tool for managing renal osteodystrophy?Volume 5, Issue 1, February 2009, Pages 25-33https://doi.org/10.1016/j.nephro.2008.04.008Get rights and content
8.K. Atsumi et al.Risk factors for vertebral fractures in renal osteodystrophy: Am J Kidney Dis(1999) livre.
9.S.A. Jamal et al. Low bone mineral density and fractures in long-term hemodialysis patients: a meta-analysis: Am J Kidney Dis(2007) livre.
10-T Bardin M C Solal RenalosteodystrophyMusculoskeletal system treaty: 14-027-L-10 1996
11-Cohen-Solal ME, Sebert JL, Boudaillez B, Marie A,Morinière P et al.Comparison of intact, midregion, and carboxy-terminal assays of parathyroid hormone for the diagnostic of bone disease in hemodialyzed patients.J Clin EndocrinolMetab1991; 73:516-524
12-Van de Vyver WJ, D'Haese PC, De Broe ME. Iron over load and bone disease in chronic dialysis patients.Nephrol Dial Transplant 1990; 5: 781-787.