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CALCIFICATION OF PERIPHERAL ARTERIES AND DUAL-ENERGY X-RAY ABSORPTIOMETRY IN PATIENTS UNDERGOING RENAL REPLACEMENT THERAPY

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Vascular calcification is common in patients with chronic kidney disease and in kidney transplant recipients. It leads to increased arterial stiffness, left ventricular hypertrophy, complicates formation of arteriovenous fistula for hemodialysis, decreases coronary artery perfusion, and generally increases cardiovascular morbidity and mortality. Vascular calcification affects arteries of all sizes – starting from the intimal and medial layers of the arterial wall. In clinical practice, several non-invasive imaging techniques have been used to evaluate the location and severity of vascular calcification. There is a report positing a possibility of evaluating vascular calcification by dual-energy x-ray absorptiometry (DXA). This paper presents the experience of successful diagnosis of peripheral arterial calcification by DXA in kidney transplant recipients and end-stage renal disease patients.

Keywords: vascular calcification, dual-energy X-ray absorptiometry, chronic kidney disease, kidney transplantation.

Vascular calcification (VC) is a degenerative vascular disease affecting the main branches of the arterial vasculature. It is associated with aging. VC can occur in either the intimal (atherosclerosis) or medial (arteriosclerosis) layers of the arterial wall. First described by Virchow in 1863, intimal calcification develops at the site of an atherosclerotic plaque, in which differentiation of osteogenic cells is induced as a result of changes in lipid accumulation, pro-inflammatory cytokines and apoptosis. Intimal calcification is more common in large and medium-sized arteries, leading to a narrowing of the vessel lumen, lower perfusion and organ ischemia. The most important risk factors for its development are the traditional cardiovascular risk factors: age, male gender, hypertension, smoking, diabetes mellitus, and chronic kidney disease (CKD) [1, 2].

Medial calcification was first described by German pathologist Mönckeberg in 1903; it is characterized by injury to the muscular middle layer of the walls of arteries. It is not accompanied by a narrowing of the vessel lumen, but increases its stiffness, and can be localized in all types of arteries. Arteriosclerosis can develop in the absence of atherosclerosis, more especially in diseases that are associated with serious metabolic changes, such as diabetes mellitus and CKD. In the latter case, vascular calcification increases with decreasing renal function. Potential risk factors for its development in CKD include hyperphosphatemia, excessive calcium intake (calcium-based phosphate binders), prolonged dialysis, vitamin D deficiency, elevated fibroblast growth factor 23, inflammatory cytokines, immunosuppressive therapy in direct and indirect renal transplant recipients with direct and indirect effects, as well as inadequate inhibition of the mineralization process due to reduced fetuin-A, matrix Gla-protein, osteoprotogerin, osteopontin, and inorganic pyrophosphate [3–6]. However, most CKD patients have both types of vascular calcification due to the presence of both risk factors. However, medial calcification is the main form that affects arteries of all sizes – from small arterioles to the aorta [1, 2, 4].

VC prevalence in CKD is high both in its early stages and among dialysis patients. Studies in patients following kidney transplantation showed no signs of VC regression. Moreover, they showed that in some cases, VC continues to progress, although this process can occur at a lower rate than in dialysis therapy [3, 6–8].

The pathogenesis of medial vascular calcification in CKD patients is a complex multifactorial process, involving chronic systemic and local inflammation, metabolic disorders and genetic abnormalities that mainly affect calcium and phosphate homeostasis [4, 5, 9, 10]. Currently, VC in CKD is seen as an active or passive complex process closely resembling skeletal bone formation. One component of active vascular calcification process involves reprogramming and transdifferentiation of vascular smooth muscle cells into osteoblast-like cells. It has been shown that the main cause of phenotypic transformation of vascular smooth muscle cells is calcium and phosphate metabolism impairment, manifested by hypercalcemia and hyperphosphatemia. The resulting osteoblast-like cells produce and secrete extracellular matrix vesicles, containing phosphate and calcium minerals in the form

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of hydroxyapatite. Vesicles are released from osteoblast-like cells in response to increased intracellular calcium and serve as a focus for subsequent mineralization. In addition, hyperphosphatemia induces vascular smooth muscle cell apoptosis, since these cells cannot adapt to a hyperphosphate environment. Undergoing apoptosis, vascular smooth muscle cells secrete apoptotic bodies from their surface, which, along with matrix vesicles, act as centers for calcium and phosphate deposition. A passive process involves deposition of minerals into the vascular wall from the extracellular fluid surrounding the vascular smooth muscle cells. In addition, it is assumed that osteoblast-like cells secrete factors that reduce the number and/or activity of osteoclast-like cells in the vascular wall, which would ensure resorption of minerals [1, 2, 9].

In real clinical practice, VC (atherosclerosis and/or arteriosclerosis) in CKD patients is associated with development/progression of cardiovascular disease – acute myocardial infarction, coronary heart disease, acute cerebrovascular accident, left ventricular hypertrophy, cardiac rhythm disturbance, including fatal, circulatory failure, which are the most important cause of cardiovascular mortality, several times higher than that in other risk groups. Peripheral arterial calcification complicates formation of constant vascular access (arteriovenous fistula) for renal replacement therapy – hemodialysis. It can also create difficulty/inability to perform vascular anastomosis in kidney transplantation [1, 7, 11].

There are several non-invasive imaging methods available for topical diagnosis of VC – from the simple to the very complex. These include standard radiography (limbs, pelvis, abdominal cavity in lateral projection), electron beam and multispiral computed tomography, magnetic resonance imaging, and duplex ultrasound. Each of the above VC detection methods has a different informational value, sensitivity, accessibility, reproducibility and safety levels. However, none of these methods can clearly differentiate between intimal and medial calcification [12, 13]. There have been separate attempts to evaluate vascular calcification using dual energy X-ray absorptiometry (DXA) since the density of the calcified arterial wall corresponds to the density of bone tissue [14]. In [15], DXA was performed in end-stage kidney disease patients, who were being treated by programmed hemodialysis and continuous ambulatory peritoneal dialysis and in kidney transplant recipients. This was to measure bone mineral density (BMD) in standard skeleton sections (distal forearm, proximal femur, lumbar spine), diagnose secondary or primary osteoporosis and evaluate the effectiveness of surgical treatment of hyperparathyroidism (HPT). It turned out that in some patients, the DXA examination allows to visualize peripheral arterial calcification.

The aim of this paper is to demonstrate the possibility of detecting peripheral arterial calcification and analyzing BMD during bone DXA all at the same time.

CLINICAL OBSERVATIONS

1. Patient C. (b. 1962)

Chronic glomerulonephritis (without histological confirmation) and stage-3 CKD were diagnosed in 2005. Since chronic renal failure was progressing, the patient was placed under continuous ambulatory peritoneal dialysis in spring 2008. Cadaveric kidney transplantation was performed in fall 2010. Primary graft function. Upon discharge from the hospital (a month later), blood biochemistry analysis parameters were: creatinine 150 μmol/L (estimated glomerular filtration rate – eGFR 46 mL/min), cholesterol 6.0 mmol/L, uric acid 284 μmol/L, ionized calcium 1.0 mmol/L, phosphorus 0.82 mmol/L (2.0 mmol/L before kidney transplantation), alkaline phosphatase 219 U/L (normal 30–280 U/L). Maintenance immunosuppressive therapy (cyclosporin A level monitored in plasma, prednisone 10 mg every other day, mycophenolate mofetil 1500 mg/day), antihypertensive therapy, statins.

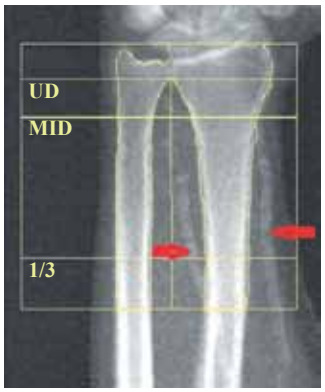
For five years, the patient's condition was satisfactory, renal graft function was stable; blood creatinine 170–200 μmol/L. In 2015, during the next ambulatory biochemical blood test: creatinine 203 μmol/L (rSCF 25 ml/min), cholesterol 4.3 mmol/L, total calcium 2.7 mmol/L, phosphorus 1.0 mmol/L, alkaline phosphatase 144 U/L (normal 60–300 U/L), parathyroid hormone (PTH) 330 pg/mL (1286 pg/mL before kidney transplantation). Anterior neck ultrasound and consultation with an endocrinologist surgeon regarding post-transplant HPT were recommended for the patient; the recommendations were not done. In 2016, type 2 diabetes was diagnosed, insulin therapy was prescribed.

Patient's condition deteriorated in fall 2017 – increasing weakness, osteoarticular pain syndrome. Was hospitalized in the kidney transplantation unit. Biochemical blood test: creatinine 206 μmol/L (eGFR 30 mL/min), total calcium 2.3 mmol/L, phosphorus 1.6 mmol/L, alkaline phosphatase 89 U/L (normal 30–120 U/L), PTH 415 pg/ml, uric acid 372 μmol/L, glucose 5.2–11.2 mmol/L, glycated hemoglobin 6.3%. Anterior neck ultrasound: in the projection of the right and left lower parathyroid glands, volumetric formations are 13 × 8 × 10 and 18 × 11 × 12 mm, respectively. DXA of standard sections of the skeleton was performed. When analyzing the BMD in the lower third of the forearm and the left proximal femur (reduced in both sections), the contours of the radial, ulnar and femoral arteries were visualized (Fig. 1). The patient was referred for a DXA of the lower third of the forearm and proximal femur on the right. This confirmed the presence of osteopenic syndrome and calcification of the radial, ulnar, and femoral arteries (Fig. 2). The

a

Name: Chizhov, Mihail Olegov
Patient ID: OHG 30206-c
DOB: 31 January 1962
Sex: Male
Ethnicity: White
Height:
Weight:
Age: 55

Referring Physician:



219 × 106

Scan Information:

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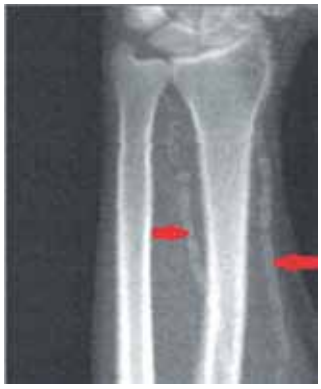
Analysis: 05 December 2017 13:58 Version 12.7
Left Forearm
Operator:
Model: Discovery A (S/N 83099)
Comment:

DXA Results Summary:

Radius	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
UD	4.48	1.23	0.274	-4.6	50	-3.8	55
MID	9.72	4.99	0.510	-3.6	72	-3.2	75
1/3	3.20	2.39	0.746	-1.3	91	-0.7	95
Total	**17.45**	**8.60**	**0.493**	**-3.7**	**72**	**-3.1**	**75**

Total BMD CV 1.0%
WHO Classification: Osteoporosis

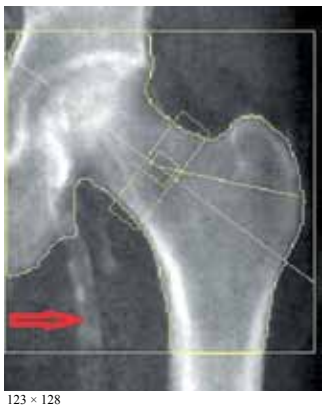
b



c

Name: Chizhov, Mihail Olegov
Patient ID: OHG 30206-c
DOB: 31 January 1962
Sex: Male
Ethnicity: White
Height:
Weight:
Age: 55

Referring Physician:



123 × 128
NECK: 49 × 15

Scan Information:

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Scan Type: x Left Hip
Analysis: 05 December 2017 13:51 Version 12.7
Hip
Operator:
Model: Discovery A (S/N 83099)
Comment:

DXA Results Summary:

Region	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
Neck	**5.98**	**3.56**	**0.596**	**-2.5**	**64**	**-1.6**	**73**
Total	49.03	35.28	0.719	-2.1	70	-1.7	74

d

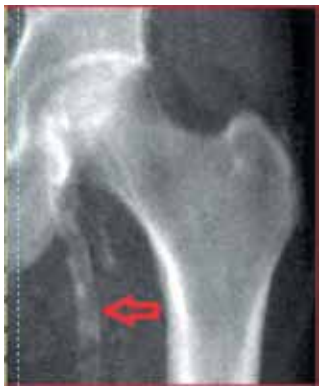


Fig. 1. DXA results for patient C.: a, b – distal bones of left forearm (arrows indicate the radial and ulnar arteries); c, d – left proximal femur (arrow indicates the femoral artery)

patient was transferred to the surgical endocrinology unit for surgical treatment of HPT.

2. Patient P. (b. 1987)

Suffers from congenital anomaly of the urinary system. Programmed hemodialysis treatment since autumn 1997 using arteriovenous fistula on the left forearm. Cadaveric kidney transplantation in 2005. In the postoperative period, delayed graft function, neo-ureteral anastomosis surgery, opening of the abscess of the anterior abdominal wall. Blood creatinine 170–180 µmol/L (eGFR 37 mL/min). Renal graft function deterioration in 2014 – proteinuria up to 4 g/day, with histological examination, glomerulosclerosis, chronic allograft nephropathy. Resumption of hemodialysis in the spring of 2015 using a permanent dialysis catheter (the formed arteriovenous fistula on the right forearm did not function). Retransplantation of cadaveric kidney on November 25, 2017. Primary graft function (Table 1).

A year later, ultrasound and CT scan of the anterior surface of the neck were performed. Two palpable abnormalities with a diameter of 10 mm were visualized in the projection of the lower poles of the thyroid gland. DXA of standard parts of the skeleton was performed, osteopenic syndrome was diagnosed in the lower third of the left forearm and the left proximal femur. The contours of the radial and ulnar arteries were visualized in both forearms, extraosseous calcified lesions on the left (in the area of aneurysmal expansion of the vascular anastomosis), femoral artery was visualized on the left thigh (Fig. 3). Consultation by an endocrinologist surgeon regarding post-transplant HPT was recommended.

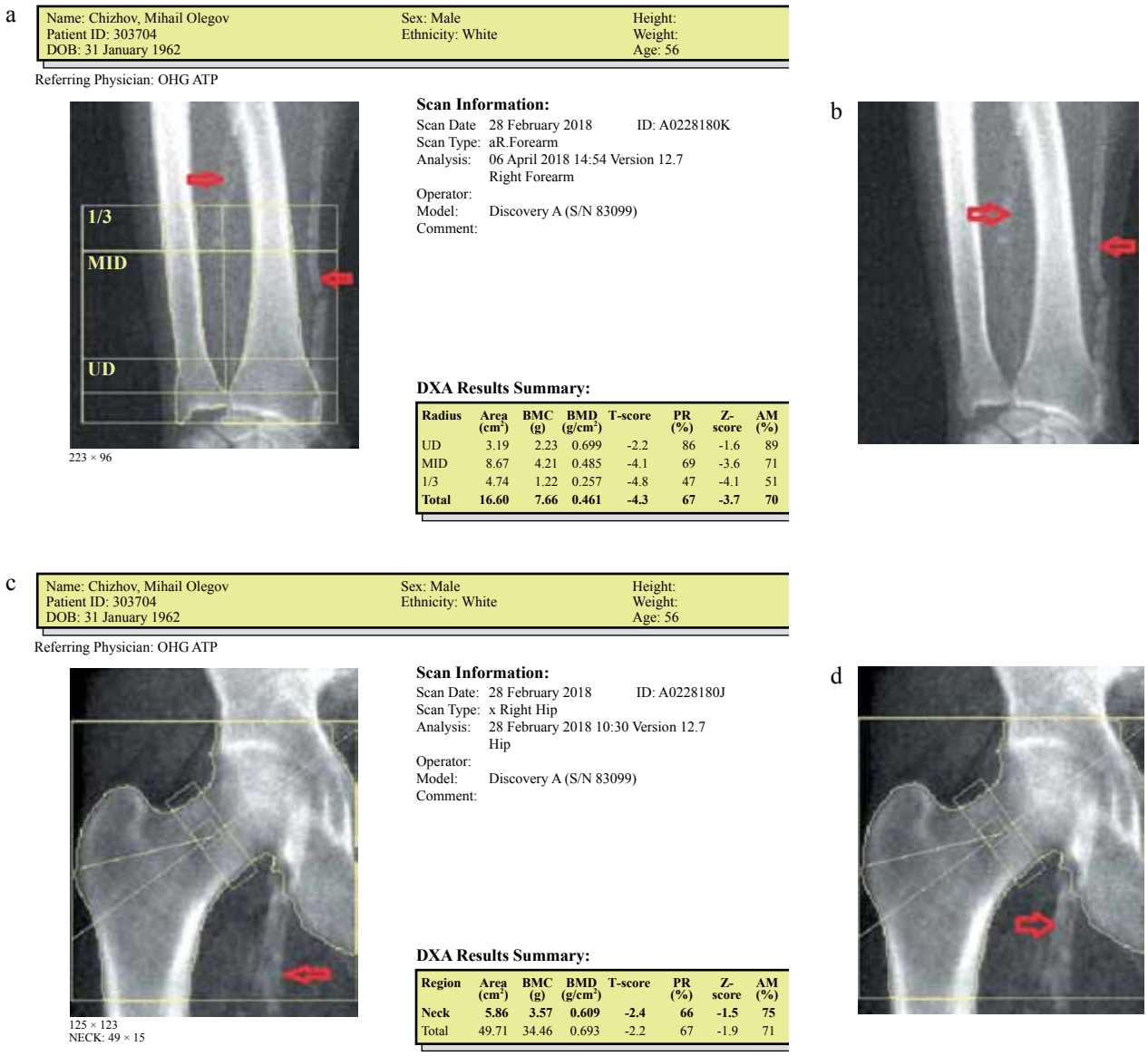


Fig. 2. DXA results for patient C.: a, b – distal bones of right forearm (arrows indicate the radial and ulnar arteries); c, d – right proximal femur (arrow indicates the femoral artery)

Table 1

Dynamic biochemical test results for patient P.

Blood indicator	Cadaver kidney retransplantation on November 25, 2017		
	Before (October 2017)	After 1 month.	After 1 year
Creatinine, μmol/L	868	80	100
Urea, mmol/L	19.6	11.6	6.6
GFR (estimated), mL/min	–	85	65
Hemoglobin, g/l	122	114	140
Total blood calcium/serum albumin, mmol/L	2.25	2.4	2.4
Phosphorus, mmol/L	2.57	1.0 (norm 0.81–1.45)	1.35 (norm 0.84–1.6)
Parathyroid hormone, pg/ml	695	180	204
Alkaline phosphatase, U/L	–	45 (norm 26–115)	113 (norm 3–258)
Cholesterol, mmol/L	–	5.2	5.7
Glycated hemoglobin, %	–	5.1	5.6

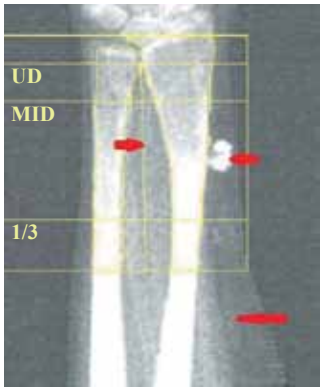
a

Name: Petrikov, Alexandr Alekseev
Patient ID: 361114
DOB: 21 July 1987

Sex: Male
Ethnicity: White

Height: 157.0 cm
Weight: 49.0 kg
Age: 31

Referring Physician: OHG ATP



194 × 94

Scan Information:

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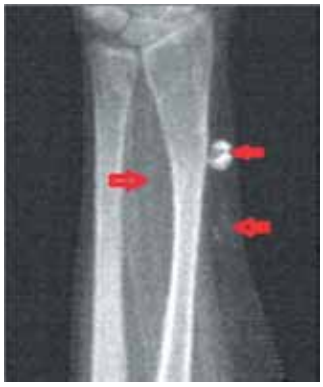
Scan Type: a L.Forearm
Analysis: 20 September 2018 10:52 Version 12.7.4.2
Left Forearm
Operator:
Model: Discovery A (S/N 83099)
Comment:

DXA Results Summary:

Radius	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
UD	3.46	0.78	0.227		41	-5.2	42
MID	6.11	2.33	0.381		54	-6.0	54
1/3	2.00	1.03	0.517		63	-5.6	64
Total	**11.56**	**4.14**	**0.358**		**52**	**-6.2**	**53**

b



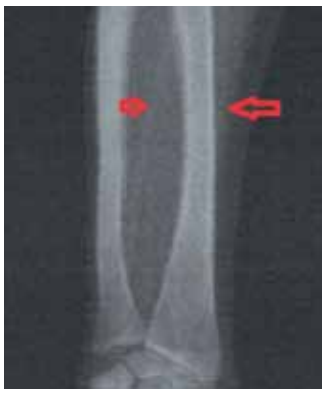
c

Name: Petrikov, Alexandr Alekseev
Patient ID: 361114
DOB: 21 July 1987

Sex: Male
Ethnicity: White

Height: 157.0 cm
Weight: 49.0 kg
Age: 31

Referring Physician: OHG ATP



Scan Information:

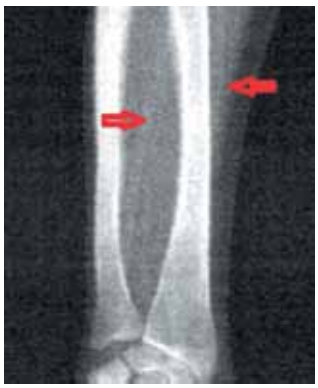
ScanDate: 20 September 2018 ID: A092Q180A
Scan Type: aR.Forearm
Analysis:

Operator:
Model: Discovery A (S/N 83099)
Comment:

DXA Results Summary:

Result data not available

d



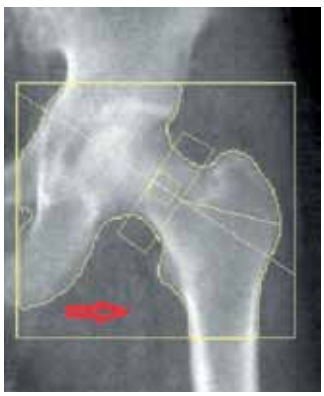
e

Name: Petrikov, Alexandr Alekseev
Patient ID: 361114
DOB: 21 July 1987

Sex: Male
Ethnicity: White

Height: 157.0 cm
Weight: 49.0 kg
Age: 31

Referring Physician: OHG ATP



111 × 102
NECK: 49 × 15

Scan Information:

ScanDate: 20 September 2018 ID: A09201807
Scan Type: x Left Hip
Analysis: 20 September 2018 10:46 Version 12.7.4.2
Hip
Operator:
Model: Discovery A (S/N 83099)
Comment:

DXA Results Summary:

Region	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
Neck	**4.51**	**2.86**	**0.634**		**68**	**-2.0**	**70**
Total	31.10	20.36	0.654		63	-2.4	64

f

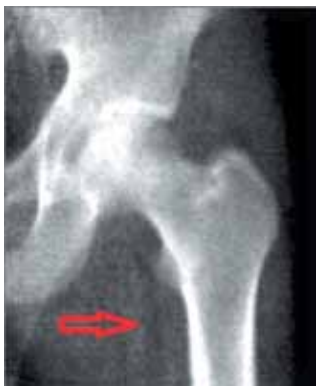


Fig. 3. DXA results for patient P.: a, b – distal part of left forearm; c, d – distal part of right forearm (arrows indicate the radial and ulnar arteries and the extraosseous calcification site); e, f – left proximal femur (the arrow indicates the femoral artery)

3. Patient O. (b. 1975)

Has been suffering from type 1 diabetes since the age of 17. Continuous ambulatory peritoneal dialysis (CAPD) since September 24, 2008 due to development of end-stage renal disease (blood urea 22.2 mmol/L, blood creatinine 538 μmol/L, eGFR 7.7 mL/min, blood hemoglobin 73 g/l). Satisfactory response to treatment, adequate CAPD program. In mid-2012, when the CAPD program became inadequate, the patient was placed under hemodialysis treatment. Repeated arteriovenous fistula formation in the lower third of the left forearm (2012), in the lower (2013) and middle third (2015) of the left shoulder using vascular prosthesis. Anemia, hypocalcemia and hyperphosphatemia were corrected (Table 2).

Persistent increase in PTH in 2012 and drug therapy were unable to normalize the function of the parathyroid gland. For two years, the patient refused surgical treatment. Parathyroidectomy was performed on June 10, 2014: three enlarged parathyroid glands were removed, the upper right gland was not found. As part of comprehensive examination, DXA of right distal forearm (arterial fistula was done on the left forearm) and the left proximal femur (Fig. 4) was performed before surgery. Osteopenic syndrome was diagnosed in both sections, while ulnar, radial and femoral arteries were visualized.

DISCUSSION

Currently, it is widely believed that there is close relationship between the bone and vascular systems in CKD patients. This is reflected in the existence of the bone-vascular axis phenomenon. It is confirmed by reports that the degree of calcification of large and medium arteries is negatively associated with BMD and positively associated with a higher rate of prevalent vertebral fractures [2, 16]. Reports also indicate that vascular calcification does not depend on the variant of renal osteodystrophy but is due to predominance of bone resorption over bone formation, and that serum phosphorus is a connecting link. In the case of adynamic bone disease, hyperphosphatemia can be the result of dietary phosphorus intake against a background of low bone turnover. With secondary HPT, on the contrary, phosphorus is released from bone due to high bone turnover [17].

Diagnosis of peripheral arterial calcification in CKD patients is very crucial in real clinical practice. When it is detected with high degree of probability, you can predict calcification of the coronary arteries, and, accordingly, fatal and non-fatal cardiovascular events [12, 18]. Experts discuss the benefits and methods of routine screening for vascular calcification as there is no convincing evidence that routine testing of this condition helps detect CKD. According to international guidelines – Kidney Disease: Improving Global Outcomes (KDIGO) – the evidence for recommendation of vascular calcification screening using lateral abdominal radiography is graded as 3C (weak and low quality of evidence) [19]. It is likely that in some dialysis patients and kidney transplant recipients, peripheral arterial calcification can be diagnosed via DXA, as our observations show.

DXA is today's established standard for measuring BMD and diagnosing primary and secondary osteoporosis. According to KDIGO guidelines, revised and updated in 2017, BMD testing is indicated for patients with CKD stages 3–5, including the dialysis population, with signs of mineral and bone disorder (MBD) and/or risk factors for osteoporosis, as there is evidence that decreased bone mineral density increases bone fracture risks [20]. HPT is one of the clinical variants of MBD in CKD and the main risk factor for formation of osteopenic syndrome (secondary osteoporosis) in these patients. It seems appropriate for all dialysis patients and kidney transplant recipients with HPT of varying severity to undergo DXA, and repeatedly for those with prolonged HPT. Measuring BMD, especially over time, will allow not only to evaluate the effectiveness of HPT therapy, but sometimes to detect peripheral arterial calcification. In patients with CKD and secondary HPT, detecting a progressive decrease in BMD and visualizing calcified arteries may be an additional argument in favor of surgical treatment of HPT. This is exactly what happened in our patients.

Table 2

Dynamic biochemical test results for patient O.

Blood parameters	Observation years									
	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017
Creatinine, μmol/L	570	500	880	840	810	680	860	720	774	902
Urea, mmol/L	11.8	9.9	11.8	12.9	13.7	16.2	20.3	7.2	10.9	10.1
Hemoglobin, g/l	97	112	107	109	97	113	132	115	121	128
Total blood calcium/serum albumin, mmol/L	2.1	2.1	2.1	2.1	2.2	2.2	2.3	2.2	2.0	2.2
Phosphorus, mmol/L	1.9	1.6	2.0	2.1	2.0	2.2	1.8	1.7	0.8	0.57
Parathyroid hormone, pg/ml	276	364	335	705	1114	1256	1754	234	190	147
Alkaline phosphatase, U/L (norm 30–120)	96	114	146	180	252	196	400	160	–	–
Cholesterol, mmol/L	5.9	6.0	5.5	6.7	6.6	4.5	4.9	4.1	–	–
Glycated hemoglobin, %	10.3	8.3	9.1	9.4	8.1	–	–	7.5	–	–

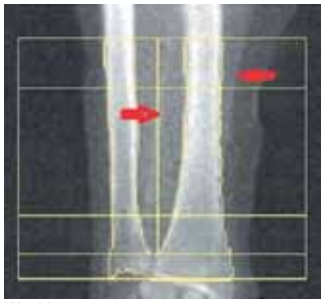
a

Name: Orlova, Natalya Anatole
Patient ID: OHG
DOB: 28 March 1975

Sex: Female
Ethnicity: White

Height:
Weight:
Age: 39

Referring Physician:



228 × 96

Scan Information:

Scan Date: 15 April 2014 ID: A04151415
Scan Type: a R.Forearm
Analysis: 12 July 2018 16:06 Version 12.7
Right Forearm
Operator:
Model Discovery A (S/N 83099)
Comment:

b

DXA Results Summary:

Radius	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
UD	2.67	1.33	0.498	-3.3	72	-2.9	74
MID	6.60	2.76	0.418	-3.5	69	-3.1	71
1/3	3.26	0.90	0.275	-2.9	62	-2.7	64
Total	**12.53**	**4.99**	**0.398**	**-3.4**	**69**	**-3.1**	**71**

c

Name: Orlova, Natalya Anatole
Patient ID: OHG
DOB: 28 March 1975

Sex: Female
Ethnicity: White

Height:
Weight:
Age: 39

Referring Physician:

113 × 111
NECK: 48 × 15

Scan Information:

ScanDate: 15 April2014 ID: A04151414
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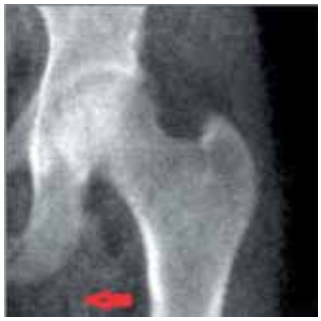
Analysis: 12 July 2018 16:08 Version 12.7
Hip
Operator:
Model: Discovery A (S/N 83099)
Comment:

d



DXA Results Summary:

Region	Area (cm^2)	BMC (g)	BMD (g/cm^2)	T-score	PR (%)	Z-score	AM (%)
Neck	**5.06**	**3.00**	**0.593**	**-2.3**	**70**	**-2.0**	**72**
Total	32.54	22.99	0.707	-1.9	75	-1.8	76

Fig. 4. DXA results for patient O.: a, b – right distal forearm (arrows indicate the radial and ulnar arteries); c, d – left proximal femur (the arrow indicates the femoral artery)

DXA of the distal forearm and proximal femur was performed in all patients as part of comprehensive examination before surgical treatment of severe HPT. This was to evaluate BMD. The scanning revealed not only a significant decrease in BMD in these bone sections (respectively 1/3 radius and total hip –2.2 SD and –2.1 SD according to T-criterion in the first patient, –5.6 SD and –2.4 SD according to the Z-criterion in the second patient and –2.7 SD and –1.8 SD according to the Z-criterion in the third patient). It also quite clearly visualized the radial ulnar and femoral arteries, which clearly indicates calcification. Forearm arterial calcification was what caused the unsuccessful formation of the arteriovenous fistula in the second patient and multiple formation of arteriovenous fistulas in the third patient. One of the real risk factors for vascular calcification in all patients should be the duration of CKD before dialysis, long-term renal replacement therapy (hemodialysis and kidney transplantation in the first two patients, peritoneal dialysis and hemodialysis in the third patient), as well as suboptimal renal graft function (in the first two patients) [6, 21]. In the third patient, diabetes mellitus, which was the main disease, played a major role in development of vascular calcification. Poor glycemic control (glycated hemoglobin >7.5%) observed in our patient was a serious factor in the development/progression of peripheral arterial calcification – hyperglycemia was shown to directly induce phenotypic transformation of vascular smooth muscle cells into osteoblast-like cells. It is very likely that in the first patient, post-transplant diabetes mellitus was also a serious factor for the development/progression of peripheral vascular calcification. Diagnosis 1.5 years before DXA and 6.3% glycated hemoglobin do not exclude longer existence of diabetes with episodes of decompensation. Finally, secondary HPT (hyperphosphatemia), its inadequate conservative correction and

delayed surgical treatment, played a crucial role in the development/progression of vascular calcification in all cases [8, 9]. Most likely, all patients have both vascular calcification variants – atherosclerosis and arteriosclerosis. Visualization of forearm arteries and femoral artery along the entire length in the form of fairly uniform linear cords with simultaneous presence of separate denser areas could serve as a confirmation of combined intimal and medial vascular calcification.

The clinical cases presented suggest that DXA could be used for distal forearm and proximal femur not only for measuring bone mineral density but also for detecting peripheral arterial calcification.

The authors declare no conflict of interest.

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