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Original Article

Prognostic Models for the Development of Diffuse Idiopathic Skeletal Hyperostosis

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Abstract

Introduction: Diffuse idiopathic skeletal hyperostosis (DISH) is a common worldwide disease in adults over 50 years of age. The clinical diagnosis at the beginning of the disease is very difficult, even impossible, without typical symptoms and image changes. Mathematical models for searching risk factors include analysing medical history data, comorbidities, biochemical and instrumental results.

Aim: The aim of the study was to analyse the demographic, clinical, biochemical, and imaging findings in patients with DISH and develop prognostic models to help identify risk factors for the disease.

Materials and methods: We analysed 124 patients with diffuse idiopathic skeletal hyperostosis treated at the Clinic of Rheumatology in St George University Hospital, Plovdiv between 2013 and 2020. All biochemical and imaging studies were performed in the facilities of the University Hospital. SPSS, ver. 26 was used for the statistical analysis.

Results: One-way analysis of history and clinical symptoms showed the highest prognostic value with OR>4 for over 50 years, mechanical pain in the thoracic and cervical spine, and Ott's symptom, OR >3 for Hirz's symptom, and OR>2 for thoracic spine stiffness, clinical evidence of spine fracture, and the Shober's symptom. We found that the highest prognostic value for the risk factors of DISH is elevated triglycerides, increased glucose, increased total cholesterol, and increased uric acid (OR over 5).

Conclusions: Our mathematical models determined the risk factors for development of DISH using different variables from the history, laboratory parameters, and imaging studies. These mathematical models are easy to apply and can be used routinely in clinical practice.

Keywords

diffuse idiopathic skeletal hyperostosis, mathematical models

INTRODUCTION

Diffuse idiopathic skeletal hyperostosis (DISH) is a common disease in adults over 50 years of age worldwide.[1-3] According to most authors, it is observed in 10% of the adult population, and its frequency is described in higher percentages in individual populations.[4,5] The disease affects mainly males and is more common with advancing age.[6,7] Although the disease has been extensively studied since 1950 and a significant number of reports have already been published, a number of DISH-related problems have not been resolved. There is no definition of the nature of the disease that is accepted by all authors. According to Resnick et al.[8] "… diffuse idiopathic skeletal hyperos-

tosis is a common ossifying diathesis in middle-aged and elderly people". It is characterized by bone proliferation on the front of the spine and the extraspinal ligaments and tendons, where they attach to the bones.[8,9] Rhotschild defines DISH as "a protective phenomenon that is likely to be a normal option for the protection of mechanical damage to spine due to an increased risk of cerebrovascular damage, fractures and the development of myelopathy."[10,11] In recent years, researchers have been looking for new clinical and laboratory criteria to be used for early diagnosis of the disease.[12,13]

The clinical diagnosis at the beginning of the disease is very difficult, even impossible, without typical symptoms and image changes.[14,15] The disease is suspected in patients aged 50 and older, men who complain of prolonged diffuse back pain, in the area of various entheses, tendons, joints, bone edges and tubers, in normal or slightly altered routine laboratory tests.[16,17] This suspicion is heightened by the establishment of muscle rigidity and restriction of movement around the pain region.[18] Sometimes the disease is painless[19-21] and is suspected in obscure dysphagia, especially dry food and head strain, accompanied by dysphonia, dyspnoea, existing myelopathy with quadriparesis or quadriplegia, cauda equina syndrome, in palpation of bone thickenings and spines, in fractures of the spine with minimal trauma or twists.[22-24]

DISH may be suspected in the presence of various risk factors: obesity, elevated BMI, type 2 diabetes mellitus or impaired glucose tolerance, hypertension, gout (hyperuricemia), hyperinsulinemia, increased pituitary growth hormone, impaired lipid metabolism (increased, triglycerides, fatty acids), etc. In case of any suspicion of the disease, it is necessary to conduct an X-ray examination of the affected area and of the middle and lower part of the thoracic spine, where the earliest characteristic radiological changes usually occur.[25-27]

Diagnosis of DISH is made using various criteria such as the following:

A. Criteria of Resnick and Nivayama[27]

B. Criteria of Julkunen et al.[28]

C. Criteria of Utsinger[29]

DISH is a disease that is unconditionally associated with other vascular and metabolic diseases.[30-33] In some patients, vascular diseases such as ischemic heart disease and transient ischemic attack precede the manifestations of diffuse hyperostosis, in others the diagnosis of one disease is an occasion to discover another by chance. In the absence of a long-term follow-up, it is difficult to establish which disease appears first, and which disease is a consequence of an already developed one.[34,35]

Mathematical models for searching diagnostic criteria include history data, comorbidities, biochemical and instrumental results to help us build the prognostic mathematical model.[36]

AIM

The aim of the study was to analyse the demographic, clinical, biochemical, and imaging findings in patients with diffuse idiopathic skeletal hyperostosis and develop prognostic models related to these clinical and laboratory results to help identify the risk factors for the disease.

MATERIALS AND METHODS

The study included 124 patients with diffuse idiopathic skeletal hyperostosis treated at the Clinic of Rheumatology in St George University Hospital, Plovdiv. All biochemical and imaging studies were conducted in the facilities of the University Hospital.

The inclusion criteria for this study were as follows:

1. Age 18 years and over.

2. Confirmed diagnosis of DISH according to the criteria of Resnick and Niwayama[15], with documented radiographic evidence of the disease. Patients presented with more than 24-month history of complaints.

3. Different duration of the disease.

4. Patients who assist in the study and provide medical documentation for concomitant diseases and previous hospitalisations.

Exclusion criteria:

1. History of psoriasis or family history of this condition.

2. History of inflammatory bowel disease.

3. History of hematological and renal diseases.

4. Cognitive impairment.

5. Presence of a neoplasm manifested in the last 5 years.

Research approach

Observational study of suitable patients from 2013-2020

The results of the patients were compared with 270 sex- and age-matched individuals with spondylosis.

Comprehensive clinical data were collected about the patients, which were coded and made available to the research team, of which patients were informed. The individual results from the obtained data, indices, and functional samples were calculated.

We measured the blood count, ESR, C-reactive protein, the biochemical indicators and the indicators of bone metabolism. All clinical and laboratory tests were performed in the Central Clinical Laboratory of St George University Hospital. Fasting blood samples were taken in the morning following strictly the manufacturer's instructions.

Readings are the results of conventional radiography of all patients from the study and from computed tomography and magnetic resonance imaging.

Statistical analysis

SPSS v. 26 was used for statistical analysis. Developing the prognostic models for DISH in the study patients goes through the following stages:

- Formation of a sample of 124 patients to identify independent, statistically significant prognostic factors for the disease onset.
- All factors that were proven to be statistically significant by the one-way analysis were analysed by a multifactor analysis. A regression model with a stepwise procedure of choice was used.
- The condition for proportionality of all covariants included in the model of the regression procedure was checked. Presence of single cases from the database was investigated, which had sharply deviating values for the covariants participating in the model, which had a strong influence on the estimation of the regression coefficients.
- Multifactor analysis of all 124 patients was repeated.

The following variables significant in the one-way analysis were tested to compile a prognostic mathematical model: anamnestic data (sudden increase in acute back pain, acute compression fracture of the thoracolumbar spine, presence of mechanical back pain, presence of combined pain and lumbar spine, stiffness in the thoracic and cervical spine), concomitant diseases (arterial hypertension, ischemic heart disease, gout, stroke, transient stroke, osteoporosis, dyslipidemia, gout, atherosclerosis), elevated values above normal of glucose, C-peptide, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, uric acid, serum osteocalcin, serum osteoprotegerin, and findings of the imaging studies (ossification of anterior, posterior, and anterior ligament, enlarged lumen of the vertebral arteries).

RESULTS

One-way regression analysis – evaluation of OR

Clinical findings, comorbidities, laboratory and imaging results were analysed using one-way regression analysis by calculating OR. One-way analysis of history and clinical symptoms showed **(Table 1)** the highest prognostic value with OR>4 for over 50 years, mechanical pain in the thoracic and cervical spine, and Ott's symptom, OR>3 for Hirz's symptom, and OR>2 for thoracic spine stiffness, clinical evidence of spine fracture, and the Shober's symptom.

When determining the OR by analysing some diseases **(Table 2)**, we found that the highest prognostic value for the risk factors of DISH was the presence of long-term treated hypertension, followed by coronary heart disease (confirmed by coronary angiography), dyslipidemia, obesity, type 2 diabetes, gout, cerebral atherosclerosis (transient ischemic attack). The table does not show these diseases or patient complaints in which the studied factor does not affect the development of the disease, such as myocardial infarction (OR=0.878) and type 1 diabetes mellitus (OR=0.978).

One-factor regression analysis did not prove prognostic value of gender, place of residence, level of education, alcohol intake and smoking for the development of DISH.

The results of the laboratory tests were also analysed using one-way regression analysis by calculating OR in the same patients **(Table 3)**. We found that the highest prognostic value for the risk factors of DISH was elevated levels of triglycerides and glucose, increased total cholesterol, and increased uric acid.

Table 1. Risk assessment of occurrence of DISH from history and clinical symptoms - one-way analysis

Factors		Control group n (%)	DISH n (%)	OR	95% CI	*P*
Age over 50 years	No	21 (7.77)	4 (3.22)	Rc (1)		<0.001*
	Yes	158 (92.3)	120 (96.7)	4.652	[2.121–7.184]	
Pain in the thoracic and cervical spine of mechanical type	No	220 (81.1)	65(52.5)	Rc (1)		<0.001*
	Yes	50 (18.9)	59 (47.5)	4.135	[2.527–6.414]	
Stiffness in the thoracolumbar spine lasting up to 10 minutes	No	239 (88.5)	91 (73.4)	Rc (1)		<0.0001*
	Yes	31 (11.5)	33 (26.6)	2.796	[1.619–4.829]	
Positive symptom of Ott	No	220 (81.1)	65(52.5)	Rc (1)		0.001*
	Yes	50 (18.9)	59 (47.5)	4.135	[2.527–6.414]	
Positive symptom of Hirz	No	205 (76.0)	91 (73.4)	Rc (1)		<0.0001*
	Yes	65 (24.0)	39 (31.45)	3.762	[1.679–5.390]	
Positive symptom of Shober	No	130 (48.1)	53 (42.8)	Rc (1)		<0.0001*
	Yes	140 (51.8)	71 (57.2)	2.413	[0.922–4.241]	

Table 2. Risk assessment of concomitant diseases for the occurrence of DISH - one-way analysis

Factors		Control group n (%)	DISH n (%)	OR	95% CI	*P*
Treated hypertension	No	164 (60.7)	13 (10.5)	Rc (1)		<0.0001*
	Yes	106 (39.3)	111 (89.5)	13.210	[7.076–24.663]	
Ischemic heart disease	No	149 (55.2)	23 (18.5)	Rc (1)		<0.0001*
	Yes	121 (44.8)	101 (81.5)	5.407	[3.239–9.027]	
Dyslipidemia	No	159 (58.8)	32 (25.8)			
	Yes	111 (41.2)	95 (76.6)	5.002	[3.332–8.809]	
Obesity class 2 and 3	No	220 (81.1)	65 (52.5)	Rc (1)		<0.0001*
	Yes	50 (18.9)	59 (47.5)	4.135	[2.527–6.414]	
Stroke and presence of cerebral atherosclerosis	No	224 (82.9)	36 (29.0)	Rc (1)		<0.0001*
	No	46 (17.03)	88 (71.0)	4.030	[3.286–6.323]	
Type 2 diabetes mellitus, oral treatment	Yes	219 (81.1)	64 (51.6)	Rc (1)		<0.0001*
	No	51 (18.9)	60 (48.4)	4.026	[2.527–6.414]	
	Yes	24 (48)	31 (56.3)	3.167	[2.234–5.991]	
Gout	No	222 (82.2)	75 (60.5)	Rc (1)		<0.0001*
	Yes	48 (17.8)	49 39.5)	3.022	[1.877–4.866]	

Table 3. Risk assessment for the occurrence of DISH from the laboratory test data - one-way analysis

Factors		Control group n (%)	DISH n (%)	OR	95% CI	*P*
Elevated glucose levels	No	205 (76.1)	13 (10.5)	Rc (1)		<0.0001
	Yes	65 (24.0)	111 (89.5)	14.335	[6.117–25.014]	
Elevated glycated hemoglobin	No	21 (55.5)	42 (70)	Rc (1)		<0.0001
	Yes	11 (44.8)	18 (30)	12.796	[6.019–13.291]	
Elevated C-peptide	No	43 (56.7)	15 (25.9)	Rc (1)		<0.0001
	Yes	33 (43.4)	43 (74.1)	11.667	[5.052–12.765]	
Elevated triglycerides	No	221 (81.9)	19 (15.3)	Rc (1)		<0.0001
	Yes	49 (18.1)	105 (84.7)	15.226	[9.121–23.224]	
Elevated total cholesterol	No	230 (85.1)	19 (15.3)	Rc (1)		<0.0001
	No	40 (14.9)	105 (84.7)	14.224	[5.234–26.012]	
Elevated levels of HDL-cholesterol	Yes	252 (93.3)	103 (83.0)	Rc (1)	*	<0.0001
	No	18 (6.7)	21 (17.0)	10.290	[5.981–13.990]	
Reduced LDL-cholesterol	Yes	44 (73.3)	14 (41.1)	Rc (1)		<0.001
	No	16 (26.7)	20 (58.9)	9.342	[5.341–12.191]	
Increased uric acid, etc.	Yes	235 (87.0)	36 (29.1)	Rc (1)		<0.0001
	No	35 (12.96)	88 (70.9)	11.236	[7.121–13.990]	

Construction of receiver operating characteristic (ROC) curves for carbohydrate profile

A ROC curve was constructed for the indicators of carbohydrate profile – serum glucose, glycated hemoglobin, and C-peptide in patients with DISH. The results are presented graphically in **Fig. 1**, the reliability is presented in **Table 4**.

Exemplary values of threshold points of the studied indicators of the carbohydrate profile and their exact mathematical expressions of specificity, sensitivity, predictive value, and accuracy are presented in **Table 5**.

Construction of ROC curves for the lipid profile

ROC curve was constructed for the indicators of the lipid profile – total cholesterol, HDL cholesterol, triglycerides in patients with DISH. The results are presented graphically in **Fig. 2**, the reliability is presented in **Table 6**.

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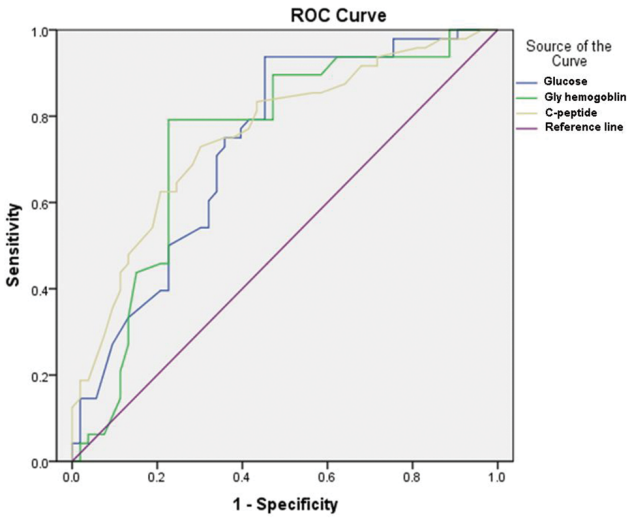

Figure 1. ROC curve for assessment of serum glucose (mmol/l), glycated hemoglobin (%), C-peptide (ng/ml) in patients with DISH.

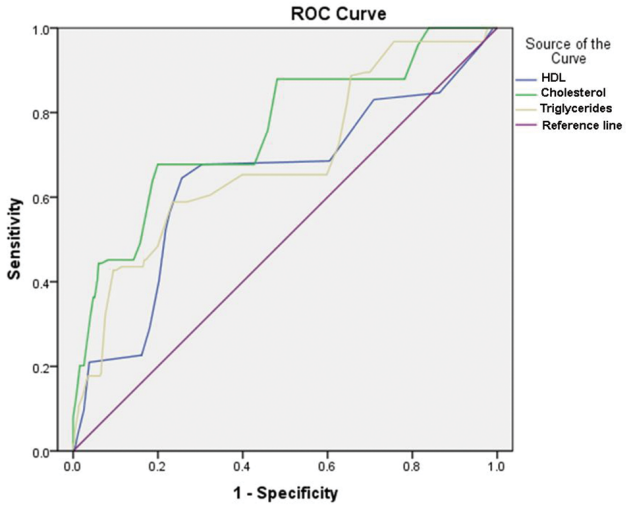

Figure 2. ROC curve for the assessment of total cholesterol (mmol/l), HDL-cholesterol (mmol/l), triglycerides (mmol/l) in patients with DISH (n=224).

Table 4. ROC indicators of serum glucose (mmol/l), glycated hemoglobin (%), C-peptide (ng/ml) in patients with DISH (n=224)

Data	Area under the ROC curve	Se	95% Confidence interval		*P*
			Lower bound	Upper bound	<0.0001
Glucose (2.8–6.1 mmol/l)	0.733	0.050	0.635	0.831	<0.0001
Glycated hemoglobin (3.5–6.3%)	0.744	0.051	0.643	0.844	<0.0001
C-peptide (0.51–2.72 ng/ml)	0.758	0.048	0.664	0.852	<0.0001

Table 5. Exemplary threshold values of the studied indicators of glucose, glycated hemoglobin, and C-peptide and ROC in patients with DISH (n=224)

Indicator	Exemplary threshold values	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Glucose (2.8–6.1 mmol/l)	9.9500	79.2%	45.3%	81.2%	75%	80%
	10.250	75%	39.6%	80.3%	72.2%	87%
Glycated hemoglobin (3.5–6.3%)	7.150	83.8%	85.1%	65.2%	65%	70%
	7.250	83.3%	83.2%	62.1%	62.2%	77%
C-peptide (0.51–2.72 ng/ml)	4.150	97.9%	86.8%	86.2%	85%	84%
	4.250	95.8%	83%	86.3%	82.2%	86%

Table 6. Indicators of ROC curve for the assessment of levels of total cholesterol (mmol/l), HDL-cholesterol (mmol/l), triglycerides (mmol/l) in patients with DISH (n=224)

Data	Area under the ROC-curve	Se	95% Confidence interval		*P*
			Lower bound	Upper bound	<0.0001
Total cholesterol (3.0–5.2 mmol/l)	0.733	0.050	0.635	0.831	<0.0001
HDL-cholesterol (1.1-1–7 mmol/l)	0.744	0.051	0.643	0.844	<0.0001
Triglycerides (0.6–1.71 mmol/l)	0.758	0.048	0.664	0.852	<0.0001

Exemplary values of threshold points of the studied indicators of the lipid profile and their exact mathematical expressions of specificity, sensitivity, predictive value and accuracy are presented in **Table 7**.

Construction of ROC curves for protein profile

ROC curve was constructed for the indicators of the protein profile – urea, uric acid, creatinine in patients with DISH. The results are presented graphically in **Fig. 3**, the reliability is presented in **Table 8**.

Exemplary values of threshold points of the studied indicators of the lipid profile and their exact mathematical expressions of specificity, sensitivity, predictive value and accuracy are presented in **Table 9**.

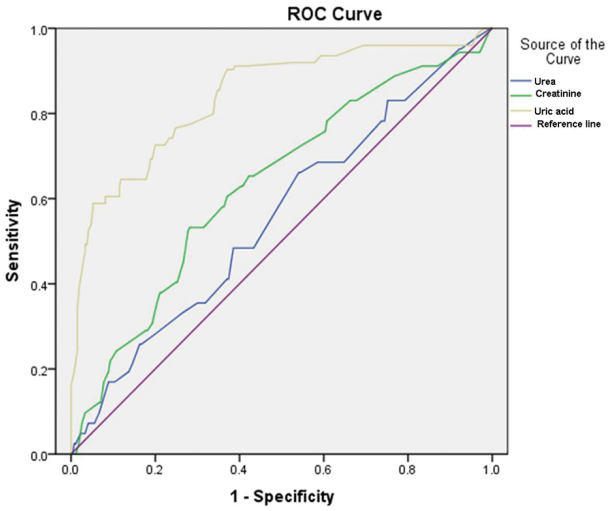

Figure 3. ROC curve for the assessment of urea (mmol/l), creatinine (μmol/l) and uric acid (μmol/l) in patients with DISH (n=224).

Table 7. Exemplary threshold values of the studied indicators of total cholesterol (mmol/l), HDL-cholesterol (mmol/l), triglycerides and ROC indicators in patients with DISH (n=224)

Indicator	Exemplary threshold values	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Total cholesterol (3.0–5.2 mmol/l)	0.750	84.7%	84.1%	85.2%	80%	89%
	0.850	83.1%	69.6%	86.3%	72.2%	76%
HDL cholesterol (1.1–1.7 mmol/l)	6.350	87.9%	48.9%	78.2%	69%	75%
	6.450	89.9%	49.8%	75.1%	71.2%	76%
Triglycerides (0.6–1.7 mmol/l)	4.350	89.5%	70%	86.2%	85%	84%
	4.450	88.7%	67.8%	86.3%	82.2%	86%

Table 8. Indicators of ROC curve for assessment of the level of urea (mmol/l), creatinine (μmol/l) and uric acid (μmol/l) in patients with DISH (n=224)

Data	Area under the ROC curve (AUC)	Se	95% Confidence interval		*P*
			Lower bound	Upper bound	<0.0001
Urea (2.6–7.2 mmol/l)	0.556	0.031	0.494	0.617	<0.075
Creatinine (74–134 μmol/l)	0.633	0.030	0.573	0.693	<0.0001
Uric acid (male 208–398 μmol/l, female 149–363 μmol/l)	0.842	0.023	0.796	0.887	<0.0001

Table 9. Exemplary threshold values of the studied indicators of urea (mmol/l), creatinine (μmol/l) и uric acid (μmol/l) and ROC indicators in patients with DISH (n=224)

Indicator	Exemplary threshold values	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Urea (2.6–7.2 mmol/l)	10.01	97%	67%	74.2%	72%	60%
	10.15	73%	56%	78.3%	63.1%	73%
Creatinine (74–134 μmol/l)	87.5	78.2%	60.7%	70.2%	65%	73%
	88.5	75.8%	60.4%	72.1%	61.2%	72%
Uric acid (male 208–398 μmol/l; female 149–363 μmol/l)	411.50	60.5%	81%	72.1%	73%	65%
	414.01	58.9%	81%	71.1%	57.2%	71%

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DISCUSSION

At the end of the 20th century, a number of factors were identified that were positively associated with the risk of developing DISH. The data about them are contradictory. From the diseases that are risky for the development of DISH, the authors take type 2 diabetes mellitus, obesity with increased BMI, arterial hypertension, coronary heart disease, atherosclerosis, gout, etc.; from the biochemical abnormalities – high serum glucose, total cholesterol, triglycerides, uric acid, some hormones and growth factors, etc.

According to our data, age over 50 years is a risk factor for the development of DISH, which is supported by the results of other authors[37], which proves that the number of patients with DISH increases with age.

The results obtained from the prognostic mathematical models based on the anamnestic data, physical examination, laboratory, and imaging findings serve to build recommendations for diagnosis based on many and different findings of the patient. Combining these results provide a clear clinical and laboratory algorithm for diagnosis and has differential diagnostic significance.

We have not found that males are at risk for developing the disease as claimed by a number of authors.[38,39]

According to the results obtained for the assessment of risk factors in DISH, we found that the altered carbohydrate, protein, and lipid metabolism, proven by some of the most common tested samples for them, are a risk factor for the disease. The developing metabolic syndrome in patients with DISH increases their cardiovascular risk and further worsens their quality of life.

Our data shows that in patients with DISH there is a change in all metabolism – protein, lipid, carbohydrate, bone, and we believe that as an element of the metabolic syndrome we should include the altered bone metabolism, which is clinically presented with diffuse hyperostosis.

Osteoporosis is not mentioned in the literature as a risk factor for the development of DISH. Our hypothesis is that osteoporosis is also a basal stimulus for the development of DISH along with the metabolic syndrome. It is no coincidence that Rhotschild notes that "DISH is a phenomenon for the protection of mechanical damage to the spine due to an increased risk of fractures, myelopathies and cerebrovascular injuries." This protection is achieved by the formation of new bone substance on the bones and the soft tissues around them. It can be assumed that the ossification of the longitudinal ligaments, as a 'splint' protects against spinal fractures and dislocation of fragments. By resisting developing osteoporosis, the body responds by increasing the function of certain hormones and growth factors, as well as the migration of mesenchymal cells into the soft tissues around the bones. The fact that the significantly higher levels of s-osteocalcin and lower levels of s-RANKL found in DISH patients is in support of this hypothesis. The protective mechanism adopted in this way explains why DISH is more common in diseases that cause certain hormonal changes with a decrease in bone strength (pituitary diseases with increased growth factor, parathyroid hyperfunction, pancreatic with increased insulin, etc.).

The results obtained from multivariate regression analysis to assess risk factors for the disease are used to make recommendations for early diagnosis.

CONCLUSIONS

Our mathematical models determined the risk factors for development of DISH using different variables from the history, laboratory parameters, and imaging studies. These mathematical models are easy to apply and can be used routinely in clinical practice.

These mathematical models could also be used for to make recommendations for early diagnosis of DISH.

Author contributions

All authors confirm participation in the design of the study, data collection, interpretation of results, data analysis, and manuscript preparation. All authors have participated in the writing of the manuscript and have given their consent to its publication.

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Competing Interests

The authors have declared that no competing interests exist.

REFERENCES

1. Miyazawa N, Akiyama I. Diffuse idiopathic skeletal hyperostosis associated with risk factors for stroke: a case-control study. Spine 2006; 31(8):E225–9.
2. Olivieri S, Angelo D, Cutro A, et al. Diffuse idiopathic skeletal hyperostosis may give the typical postural abnormalities of advanced ankylosing spondylitis. Rheumatology 2007; 46(11):1709–11.
3. Pappone N, Di Girolamo C, Del Puente A, et al. Diffuse idiopathic skeletal hyperostosis (DISH): a retrospective analysis. Clin Rheumatol 1996; 15(2):121–4.
4. Uehara M, Takahashi J, Ikegami S, et al. Impact of diffuse idiopathic skeletal hyperostosis on sagittal spinal alignment in the general elderly population: a Japanese cohort survey randomly sampled from a basic resident registry. JBJS Open Access 2019; 4(3). doi: 10.2106/

JBJS.OA.18.00062
5. Forestier J, Lagier R. Ankylosing hyperostosis of the spine. Clin Orthop Relat Res 1971; 74:65–83.
6. Alparslan L, Yu J, Weissman B. Diffuse idiopathic skeletal hyperostosis. In: Lee DM, Kiener HP, Brenner MB. Kelley's Textbook of Rheumatology. 7th ed. Philadelphia: WB Saunders/Elsevier; 2005; 1:783–5.
7. Armas J, Couto A, Bettencourt B. Spondyloarthritis, diffuse idiopathic skeletal hyperostosis (DISH) and chondrocalcinosis. Adv Exp Med Biol 2009; 649:37–56.
8. Resnick D, Guerra J, Robinson C, et al. Association of diffuse idiopathic skeletal hyperostosis (DISH) and calcification and ossification of posterior longitudinal ligament. AYR 1978; 131(6):1049–54.
9. Resnick D, Shapiro R, Weisner K, et al. Diffuse idiopathic skeletal hyperostosis – DISH (Ankylosing hyperostosis or Forestier and Rotes-Querol disease. Semin Arthr Rheumat 1978; 11(3):153–87.
10. Rhothschild B. Diffuse idiopathic skeletal hyperostosis as reflected in the paleontologic records: Dinosaurs and early mammals. Semin Arthrit Rheumat 1987; 17(2):119–25.
11. Rhothschild B. Diffuse idiopathic skeletal hyperostosis. Compr Ther 1988; 14(2):65–9.
12. Mader R. Diffuse idiopathic skeletal hyperostosis: time for a change. J Rheumat 2008; 35(3):377–9.
13. Sarzi-Puttini P, Atzeni F. New developments in our understanding of DISH (diffuse idiopathic skeletal hyperostosis). Currt Opin Rheumat 2004; 16(3):287–92.
14. Robinson PC, Wordsworth BP, Reveille JD, et al. Axial spondyloarthritis: a new disease entity, not necessarily early ankylosing spondylitis. ARD 2013; 72(2):162–4.
15. Resnick D, Niwayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). Radiology 1976; 119(3):559–68.
16. Kim SK, Choi BR, Kim CG, et al. The prevalence of diffuse idiopathic skeletal hyperostosis in Korea. J Rheumatol 2004; 31(10):2032–5.
17. Mader R. Clinical manifestations of diffuse idiopathic skeletal hyperostosis of the cervical spine. Semin Arthritis Rheum 2002; 32(2):130–5.
18. Mori K, Kasahara T, Mimura T, et al. Prevalence of thoracic diffuse idiopathic skeletal hyperostosis (DISH) in Japanese: results of chest CT-based cross-sectional study. J Orthop Sci 2017; 22(1):38–42.
19. Sacks D. Diffuse idiopathic skeletal hyperostosis (DISH). Symptoms, diagnosis and treatment. Health Amid Infi 2011; 23(2):56–60.
20. Sarzi-Puttini P, Atzeni F. New developments in our understanding of DISH (Diffuse idiopathic skeletal hyperostosis). Curr Opin Rheumat 2004; 16:287–92.
21. Schoenfeld A, Harris M, Shiel W. Diffuse idiopathic skeletal hyperostosis (cont). 2011; 10–31.
22. Sencan D, Elden H, Nacitarhan V, et al. The prevalence of diffuse idiopathic skeletal hyperostosis in patients with diabetes mellitus. Rheum Int 2005; 25(7):518–51.
23. Solaroglu I, Okutan O, Karakus M, et al. Dysphagia due to diffuse idiopathic skeletal hyperostosis of the cervical spine. Turk Neurosurg 2008; 18(4):409–11.
24. Burner T, Rosenthal A. Diabetes and rheumatic diseases. Curr Opin Rheumatol 2009; 21(1):50–4.
25. Olivieri S, Angelo D, Palazzi C, et al. Diffuse idiopathic skeletal hyperostosis. Differentiation from ankylosing spondylitis. Curr Rheumat Rep 2009; 11(5):328–31.
26. Oppenlander ME, Orringer DA, La Marca F, et al. Dysphagia due to anterior cervical hyperosteophytosis. Surg Neurol 2009; 72(3):266–70.
27. Resnick D, Novayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). Radiology 1976; 119(3):559–68.
28. Julkunen H, Heinonen O, Knekt P, et al. The epidemiology of hyperostosis of the spine together with its symptoms and related mortality in a general population. Scand J Rheumatol 1975; 4:23.
29. Utsinger P. Diffuse idiopathic skeletal hyperostosis. Clin Rheum Dis 1985; 11(2):325–51.
30. Holton KF, Denard PJ, Yoo JU, et al. Osteoporotic Fractures in Men (MrOS) Study Group. Diffuse idiopathic skeletal hyperostosis and its relation to back pain among older men: the MrOS Study. In: Seminars in arthritis and rheumatism. WB Saunders 2011; 41(2):131–8.
31. Mori K, Kasahara T, Mimura T, et al. Prevalence of thoracic diffuse idiopathic skeletal hyperostosis (DISH) in Japanese: results of chest CT-based cross-sectional study. J Orthop Sci 2017; 22(1):38–42.
32. Kim B, Moon M, Yoon M, et al. Prevalence of diffuse idiopathic skeletal hyperostosis diagnosed by whole spine computed tomography: a preliminary study. Clin Orthop Surg 2018; 10(1):41–6.
33. Littlejohn G, Smythe H. Marked hyperinsulinemia after glucose challenge in patients with diffuse idiopathic skeletal hyperostosis. J Rheumatol 1981; 8(6):965–8.
34. Romero-Vargas S, Zárate-Kalfópulos B, Otero-Cámara E. The impact of body mass index and central obesity on the spino-pelvic parameters: a correlation study. Eur Spine J 2013; 22(4):878–82.
35. Allen RJ, Pardo MS. The problematic value of mathematical models of evidence. J Leg Stud 2007; 36(1):107–40.
36. Westerveld L, Van Ufford H, Verlaan J, et al. The prevalence of diffuse idiopathic skeletal hyperostosis in an outpatient population in the Netherlands. J Rheumatol 2008; 35(8):1635–8.
37. Rhotschild B, Grogon D. Diffuse idiopathic skeletal hyperostosis, treatment and management. Medscape 2011; 31.
38. Baxi V, Gaiwal S. Diffuse idiopathic skeletal hyperostosis of cervical spine. An unusual cause of difficult fiber optic intubation. Saudi J Anaesht 2010; 4(1):17–9 39.
39. Le HV, Wick JB, Van BW, et al. Diffuse idiopathic skeletal hyperostosis of the spine: pathophysiology, diagnosis, and management. J Am Acad Orthop Surg 2021; 29(24):1044–51. doi: 10.5435/JAAOS-D-20-01344.

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Прогностические модели развития диффузного идиопатического скелетного гиперостоза

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Резюме

Введение: Диффузный идиопатический скелетный гиперостоз (ДИСГ) является распространённым во всём мире заболеванием у взрослых старше 50 лет. Клинический диагноз в начале заболевания очень труден, даже невозможен без типичных симптомов и изменений картины. Математические модели поиска факторов риска включают анализ данных анамнеза, сопутствующих заболеваний, результатов биохимических и инструментальных исследований.

Цель: Цель исследования состояла в том, чтобы проанализировать демографические, клинические, биохимические и визуализационные данные у пациентов с ДИСГ и разработать прогностические модели, помогающие определить факторы риска заболевания.

Материалы и методы: Мы проанализировали 124 пациентов с диффузным идиопатическим скелетным гиперостозом, лечившихся в Клинике ревматологии Университетской больницы Святого Георгия в Пловдиве в период с 2013 по 2020 год. Все биохимические и визуализирующие исследования проводились в условиях университетской больницы. SPSS, ver. 26 использовали для статистического анализа.

Результаты: Однофакторный анализ анамнеза и клинических симптомов показал наибольшую прогностическую ценность при OR>4 в течение более 50 лет, механической боли в грудном и шейном отделах позвоночника и симптоме Отта, OR>3 для симптома Герца и OR>2 для тугоподвижности грудного отдела позвоночника, клинических признаков перелома позвоночника и синдрома Шобера. Мы обнаружили, что наибольшей прогностической ценностью факторов риска ДИСГ является повышенный уровень триглицеридов, повышенный уровень глюкозы, повышенный уровень общего холестерина и повышенный уровень мочевой кислоты (OR более 5).

Заключение: Наши математические модели определили факторы риска развития ДИСГ с использованием различных переменных из анамнеза, лабораторных параметров и исследований изображений. Эти математические модели просты в применении и могут рутинно использоваться в клинической практике.

Ключевые слова

диффузный идиопатический скелетный гиперостоз, математические модели