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Primary intracranial peripheral primitive neuroectodermal tumor: lessons from an exceptionally rare neoplasm. Illustrative case

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BACKGROUND The primary intracranial peripheral primitive neuroectodermal tumor (pPNET) is a lesion subtype within the Ewing sarcoma family of tumors. pPNETs are extremely uncommon pathologies, accounting for 0.03% of intracranial tumors and 1% to 2% of Ewing sarcoma cases. Given its histological aspect similar to other highly proliferative malignant neuroectodermal neoplasms, pPNET merits extensive workup for accurate diagnosis and treatment.

OBSERVATIONS A 36-year-old male presented to the emergency department with a 1-year history of headaches in the right frontoparietal area, generalized tonic-clonic seizures, and a history of the resection of a tumor labeled as a meningioma 5 years before admission. He was neurologically intact. Brain magnetic resonance imaging revealed a heterogeneous focal lesion of 25 × 35 × 23 mm with a necrotic center and neoformative appearance in the right frontal cortex. The patient underwent multimodal treatment with gross-total resection, radiotherapy, and chemotherapy. Histopathological examination results supported the diagnosis of pPNET. At the 2-year follow-up, the patient had no new-onset symptoms, and brain imaging revealed absent signs of tumor recurrence.

LESSONS The present case describes an extraordinary pPNET case, initially confounded as a clear cell meningioma. Managing pPNET requires thorough investigation, careful differentiation from similar neuroectodermal lesions, and multimodal treatment to improve the patient's prognosis.

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KEYWORDS Ewing sarcoma; primitive neuroectodermal tumor; intracranial peripheral primitive neuroectodermal tumor; brain tumor; pPNET

The Ewing sarcoma family of tumors (ESFT) comprises a series of small round-cell neoplasms, including Ewing sarcoma (EWS), primitive neuroectodermal tumor (PNET), Askins tumor, PNET of the bone, and extraosseous Ewing sarcoma (ESS).[1] Within the ESFT spectrum, the primary intracranial peripheral primitive neuroectodermal tumor (pPNET) represents an extremely rare tumor.[2–4] Typically, pPNET can arise from the meninges and be confounded with other small, poorly differentiated round-cell tumors such as medulloblastoma, atypical teratoid/rhabdoid tumor, and lymphoma.[3,4] pPNET frequently affects children and young adults, whereas middle-aged adult and elderly populations have shown a low prevalence.[5–7] Symptoms from this condition are unspecific, vary depending on the tumor location, and can be secondary to mass effect (e.g., seizures, neurological deficits, vomiting, and headaches).[5]

The scarcity of reports, the broad differential diagnosis, and optimal therapy are among the most notable challenges in understanding pPNET comprehensively.[2,5,8–12] The case study herein ameliorates the paucity of evidence by illustrating a relapsing pPNET, initially misdiagnosed as a clear cell meningioma, that merited further multimodal treatment. In addition, we present a literature review of dura-based pPNETs in adults and discuss preceding cases with a puzzling differential diagnosis.

Illustrative Case

A 36-year-old male presented to the emergency department with a 1-year history of headaches and recurrent tonic-clonic seizures under treatment with valproic acid 500 mg three times

ABBREVIATIONS CT = computed tomography; ESFT = Ewing sarcoma family of tumors; ESS = extraosseous Ewing sarcoma; EWS = Ewing sarcoma; GTR = gross-total resection; MRI = magnetic resonance imaging; NICU = neurointensive intensive care unit; PNET = primitive neuroectodermal tumor; pPNET = peripheral primitive neuroectodermal tumor; STR = subtotal resection.

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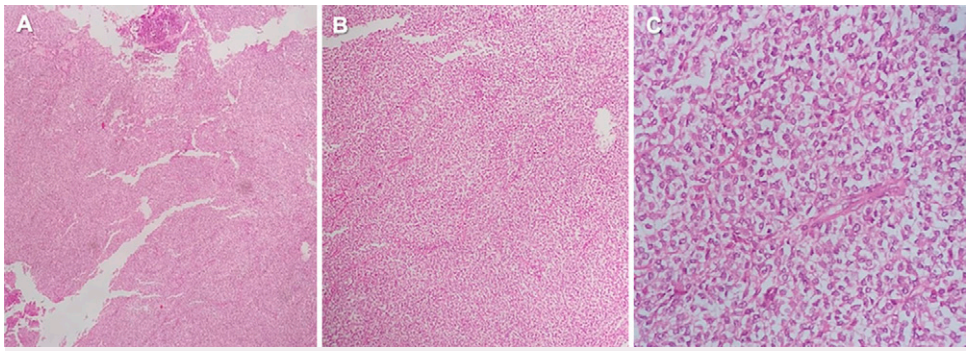

FIG. 1. A histopathological report from an external facility described a specimen with high cellularity and rounded cells of clear cytoplasm. Hematoxylin and eosin, original magnification ×50 (**A**), ×100 (**B**), and ×400 (**C**).

per day and levetiracetam 1,000 mg twice per day. His past history was relevant for a right frontal craniotomy performed 5 years before admission for resection of a frontal tumor, whose histopathological diagnosis was clear cell meningioma grade II (Fig. 1). The patient did not receive adjuvant therapy or attend further follow-up consults at the external facility until admission to our surgical center.

On neurological examination, the patient had a Glasgow Coma Scale score of 15, isochoric pupils reactive to light, and no motor deficits. Revision of the histopathological report from the outside hospital revealed a rounded cell and light cytoplasm, suggestive of a subtype of ESFT lesion.

Head computed tomography (CT) without contrast showed a heterogeneous and irregular lesion in the superior frontal gyrus with a hypodense core, hyperdense periphery, and surrounding edema (Fig. 2A–C). Brain magnetic resonance imaging (MRI) with gadolinium revealed a ring-enhancing cortical lesion with an evident neoformative component of 25 × 35 × 23 mm in the right frontal lobe (Fig. 2D–F). After a discussion of the prognosis and treatment options with the patient, he agreed to undergo excision. The patient was positioned in a standard position, followed by a right craniotomy, tumor exeresis, and heterologous duroplasty. In the operative field, the lesion appeared moderately vascularized, measuring 25 to 30 mm, and infiltrated the dura mater.

The patient tolerated the procedure well and was transferred to the neurointensive care unit (NICU) for clinical monitoring. Postoperatively, the patient was stable and neurologically intact. Brain imaging suggested complete excision of the tumor (Fig. 2G–I). The histopathological and immunohistochemistry results supported the diagnosis of pPNET (Fig. 3) with positive expression of CD99, EMA, vimentin, and cytokeratin and negative expression of PGAF, LCA, RP, and synaptophysin, with Ki-67 levels of 5% to 10%. After recovery in the NICU, the patient received chemotherapy with vincristine 2 mg, doxorubicin 140 mg, and cyclophosphamide 2,300 mg in nine alternating cycles during the induction phase and then five cycles for the consolidation phase. Furthermore, adjuvant radiotherapy was utilized in the excision site with three-dimensional conformal radiation (54 Gy over 30 fractions). At the 1- and 2-year follow-ups, the patient had no new-onset symptoms, and brain MRI studies showed no signs of tumor recurrence (Fig. 4).

Patient Informed Consent

The necessary patient informed consent was obtained in this study.

Discussion

Accounting for 0.03% of intracranial tumors and 1% to 2% of EWS cases, pPNETs are extremely rare malignant neoplasms.[2,6,13–17] Demographically, children and adolescents are commonly affected.[2,17–19] MRI findings commonly show heterogeneous and hypo- to isointense lesions on T1-weighted sequences and iso- to hyperintense lesions on T2-weighted sequences.[2] Additionally, histopathological examination of pPNET specimens often reveals small, round, darkly stained cells with hyperchromatic nuclei and a high mitosis rate.[20,21] This microscopic similarity is shared with other malignant pathologies such as anaplastic ependymoma, atypical teratoid/rhabdoid tumor, lymphoma, rhabdomyosarcoma, malignant meningioma, and other forms of highly proliferative malignant neuroectodermal tumors.[5,20,22] Given this broad differential diagnosis, the final diagnosis of pPNET requires an extensive workup, including immunohistochemistry analysis and cytogenetic examination. Immunohistochemistry analysis shows positive expression of CD99, FLI-1, NKX2.2, GFAP, and synaptophysin and negative EMA expression. In particular, CD99 is a highly sensitivity but nonspecific marker.[20] Conversely, detection of the *EWSR1* gene, stemming from the translocation t(11;22)(q24;q12), can be regarded as more specific and confirmatory.[5,8–12] Besides, it is worth mentioning that central nervous system embryonal tumors are negative for CD99 and *EWSR1* gene rearrangement, which helps to distinguish them from pPNET.[20]

Although no consensus exists about the optimal therapy for pPNETs, treatment options include resection, chemotherapy, and radiotherapy. Gross-total resection (GTR) is regarded as the cornerstone of therapy and impacts survival rates. Chen et al.[2] reported that the mean survival time with GTR was longer than with subtotal resection (STR), although this difference was not statistically significant. Thus, when feasible, GTR should be the primary goal of surgical intervention. Adjuvant radiotherapy and chemotherapy (e.g., vincristine, cyclophosphamide, doxorubicin, ifosfamide, and etoposide) have also been demonstrated to be valuable. The regimens for radiotherapy have been variable. In a review of the literature including 48 cases, Cherif El Asri et al.[5] described the use of radiation with two distinct radiation parameters: one group with a dose of 36 to 60 Gy in a conventional fractionation and another group with a

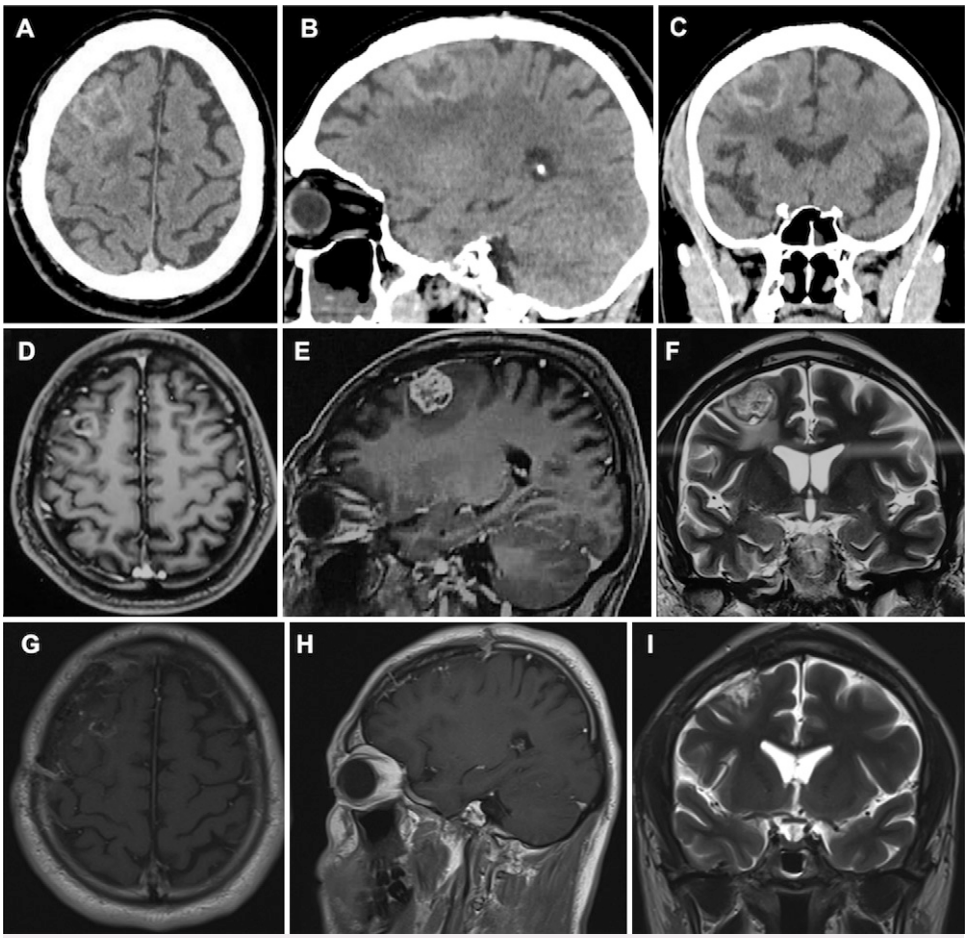

FIG. 2. Preoperative axial (**A**), sagittal (**B**), and coronal (**C**) head CT scans without contrast depict a heterogeneous and irregular lesion of the hypodense core and hyperdense periphery in the right frontal lobe. No midline deviation was present. Preoperative axial (**D**) and sagittal (**E**) MRI with gadolinium revealed a ring-enhancing lesion with a necrotic center and neoformative appearance of 25 × 35 × 23 mm in the cortical area of the frontal lobe. Preoperative coronal fluid-attenuated inversion recovery (FLAIR) sequence (**F**) depicts a heterogeneous and hyperintense lesion in the cortical frontal area. Postoperative T1-weighted (**G and H**) and FLAIR (**I**) sequences demonstrated complete tumor excision.

dose of 36 to 54 Gy for incomplete resection and 60 Gy after intralesional resection. However, there was no significant difference between the two groups, thus suggesting that the dose does not influence prognosis. Furthermore, chemotherapy as adjuvant therapy to surgery provides better survival outcomes (84%) versus the use of surgery alone (53%).[5] In the present case, multimodal treatment was effective in tumor control at the last follow-up.

We performed a MEDLINE review of articles reporting dura-based pPNET in adults over the last decade (Table 1).[2,7,18,19,23–33] Approximately 16 patients (50% female), with a median age of 33 years (range 18–56 years), were diagnosed with pPNET and followed up for a median of 12 months (range 0.2–60 months). Frequent presenting symptoms included headaches, vomiting, vision impairment, and neurological deficits. These lesions were commonly located in the frontal (25%), frontoparietal (12.5%), or frontotemporal (12.5%) regions. Because pPNET has a meningeal origin and displays a dural base on brain MRI,[18] 31% of cases mimicked the presence of a meningioma or hemangiopericytoma.[7,18,27,28,30] This finding resonates with our clinical scenario, given that an absent acknowledgment of pPNET in the differential diagnosis can impact the precision of treatment and the patient outcome.

Regarding workup strategies, immunohistochemistry analysis revealed CD99 expression in all patients, thus helping to rule out other tumors (Table 1). Less frequent markers included vimentin, synaptophysin, nonspecific enolase, S100, and nestin. Genetic sequencing and fluorescence in situ hybridization were utilized in only 44% of cases to identify the presence of *EWSR1* or translocation t(11;22). A surgical approach was undertaken in all patients, with GTR achieved in 80% of patients. Adjuvant radiotherapy or chemotherapy was reported in 87% of cases. Radiation dosages ranged from 50 to 55.8 Gy and were administered fractionally. Chemotherapy included varying regimens of vincristine, ifosfamide, etoposide, doxorubicin, cyclophosphamide, actinomycin,

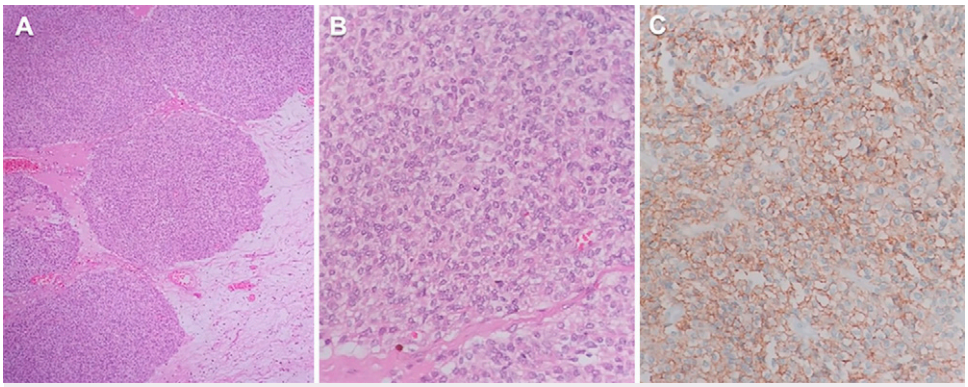

FIG. 3. Hematoxylin and eosin staining demonstrated evident signs of mitosis (**A**, original magnification ×100) with rounded cells of clear cytoplasm (**B**, original magnification ×400). Immunohistochemical staining depicted positive expression of CD99 (**C**, original magnification ×400).

adriamycin, and dactinomycin. During follow-up, 38% of patients remained asymptomatic, 25% had tumor recurrence, and 31% died.

In summary, a critical remark given our case is to provide early identification of pPNET based on clinical suspicion, awareness, and a thorough workup when discerning a meningeal lesion with malignant behavior.[12] This deliberation, coupled with an appraisal of tumor location, extent of resection, utilization of adjuvant radiotherapy and chemotherapy, and bone lysis, impact the patient's prognosis.[5]

Observations

The present case reflects the current challenge with diagnosis and optimizing the treatment of patients with pPNET; as initially diagnosed in an external facility, clear cell meningioma can confound the initial diagnosis. Furthermore, although specific treatment guidelines are lacking for this pathology, multimodal treatment with resection and adjuvant radiotherapy and chemotherapy appears to provide stable tumor growth. However, there remain undefined knowledge gaps meriting further research, such as optimal radiation dose and chemotherapy regimens.

Lessons

pPNETs are extremely uncommon pathologies with a broad differential diagnosis. An extensive workup, including brain imaging, histopathological examination, immunohistochemistry analysis, and cytogenetic analysis, aids in confirming the diagnosis. Although notable knowledge gaps exist regarding the optimal therapy, GTR and adjuvant therapy using radiotherapy and chemotherapy, when feasible, appear to provide stable tumor growth.

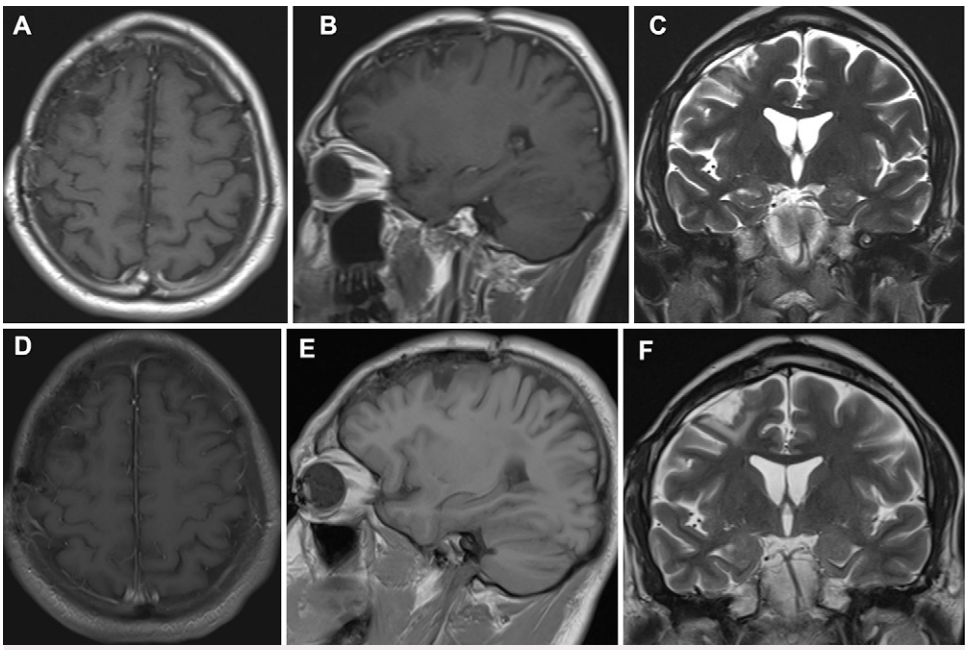

FIG. 4. One-year (**A–C**) and 2-year (**D–F**) postoperative MRI showed no signs of tumor recurrence.

TABLE 1. Literature review of dura-based pPNET in adults

Authors & Year	Age (yrs)	Sex	Presenting Symptoms & Signs	Tumor Location	Size (mm)	IHC & Genetic Analysis	EOR	Adjuvant Therapy	Tumor Recurrence	Neurological Outcome	FU
Rainov et al., 2024[23]	18	M	HA, scalp swelling	Lt frontoparietal	NR	CD99+, vimentin+, t(11;22)	GTR	Chemo: V, I, E, & Dox	No	No deficits	12 yrs
Datta et al., 2023[24]	55	F	HA, vision impairment	Lt frontal	NR	CD99+, synaptophysin+	GTR	Chemo	NR	NR	NR
Hyun et al., 2023[7]	38	F	Rt-sided tinnitus, rt hemiparesis & imbalance	Rt petrous ridge	44 × 56 × 60	CD99+, vimentin+, EWSR1+	STR	Chemo: V, A, C, I, & E; RT: 55 Gy	No	Asx	12 mos
Mohakud et al., 2022[25]	50s	F	HA, projectile vomiting, blurred vision	Lt fronto-temporal	NR	CD99+, synaptophysin+	NR	NR	NR	Death	6 days
Deshpande et al., 2021[26]	33	M	NR	Lt temporal	NR	CD99+, t(11;22)	STR	Chemo: V, A, C, Ac, I, & E; focal RT: 55.8 Gy	No	No deficits	18 mos
Jiang et al., 2020[18]	55	F	Memory decline, muscle strength decrease	Lt frontal lobe	43 × 65 × 35	CD99+, vimentin+, EWSR1+	GTR	RT: 55 Gy × 1 mo	No	Asx	18 mos
Bansal et al., 2019[27]	22	F	HA, scalp swelling	Lt parieto-occipital	NR	CD99+	GTR	Chemo & RT	No	Asx	14 mos
Chen et al., 2019[2]	23	M	Scalp swelling	Lt fronto-temporal	NR	CD99+	STR	RT: 55 Gy	NR	Death	6 mos
	43	M	Epilepsy	Rt parietal	NR	CD99+	GTR	Chemo: V, D, C, & A; RT: 50 Gy	Yes	Death	5 yrs
Sohail et al., 2019[28]	20	M	HA, drowsiness, & fever	Rt temporo-parietal	NR	CD99+, synaptophysin+, & t(11;22)	GTR	Chemo & RT	Yes	Lt-sided hemiplegia	3 mos
Singh et al., 2018[29]	18	M	Seizure	Lt frontoparietal convexity	70 × 70 × 60	CD99+, vimentin +	GTR	RT: 55 Gy	No	NR	12 mos
Kumar et al., 2017[30]	22	M	Generalized tonic-clonic seizure	Lt frontal	70 × 48 × 88	CD99+, nonspecific enolase+	GTR	Chemo & RT	No	No deficits	7 mos
Salunke et al., 2014[19]	52	M	Low-grade fever, HA, vision impairment	Occipital	NR	CD99+, vimentin+	STR	Chemo: V, A, C, E, & I; RT: 50 Gy	Yes	Death	10 mos
Cole et al., 2014[31]	51	F	Visual disturbances	Occipital	35 × 30	CD99+, vimentin+, S100+, EWSR1+	GTR	Chemo: V, C, Dox, I, & E	No	NR	24 mos
Tanboon et al., 2012[32]	22	F	HA, projectile vomiting, & blurred vision	Rt frontal	80 × 70 × 40	CD99+, synaptophysin+, vimentin+	GTR	No	Yes	Death	6 mos

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TABLE 1. Literature review of dura-based pPNET in adults

Authors & Year	Age (yrs)	Sex	Presenting Symptoms & Signs	Tumor Location	Size (mm)	IHC & Genetic Analysis	EOR	Adjuvant Therapy	Tumor Recurrence	Neurological Outcome	FU
Mellai et al., 2010[33]	56	F	HA, lt-side hemiparesis	Rt temporal	NR	CD99+, vimentin+, nestin+, EWS-FLI1 fusion	GTR	No	No	No deficits	18 mos

A = Adriamycin; Ac = actinomycin; Asx = asymptomatic; C = cyclophosphamide; chemo = chemotherapy; D = dactinomycin; Dox = doxorubicin; E = etoposide; EOR = extent of resection; FU = follow-up; HA = headache; I = ifosfamide; IHC = immunohistochemistry; NR = not reported; RT = radiotherapy; V = vincristine; + = positive.

Acknowledgments

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Rojas-Apaza, Tacas-Gil. Acquisition of data: all authors. Analysis and interpretation of data: Rojas-Apaza, Bocanegra-Becerra, Novoa-Ramírez. Drafting the article: Rojas-Apaza, Bocanegra-Becerra. Critically revising the article: Rojas-Apaza, Bocanegra-Becerra, Tacas-Gil. Reviewed submitted version of manuscript: Rojas-Apaza, Bocanegra-Becerra, Novoa-Ramírez, Latorre-Zúñiga. Approved the final version of the manuscript on behalf of all authors: Rojas-Apaza. Statistical analysis: Bocanegra-Becerra. Administrative/technical/ material support: Bocanegra-Becerra, Novoa-Ramírez, Latorre-Zúñiga. Study supervision: Rojas-Apaza, Tacas-Gil.

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