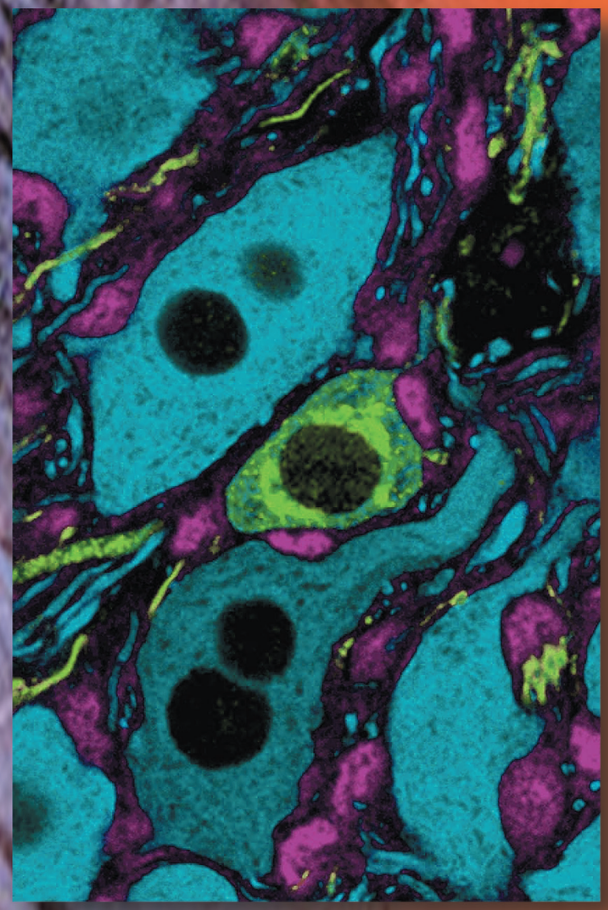
MAY 2021 VOL. 42 No. 2 Pages 43 - 69 AUSTRALIAN JOURNAL OF MEDICAL SCIENCE

May 2021
Vol. 42
No. 2

AUSTRALIAN JOURNAL OF

Medical Science

2021



CASE STUDY

Severe RhD haemolytic disease of the foetus and newborn in RhD negative multigravida

ORIGINAL ARTICLE

The diagnostic challenge in a giant midline neck mass: spindle cell pleomorphic lipoma versus soft tissue tumour

REGULAR FEATURES

GENE THERAPY CAN ADDRESS THE ROOT CAUSE OF MONOGENIC DISEASES

Novartis is at the forefront of gene therapy, an exciting new branch of medicine.

Monogenic diseases are inherited conditions caused by a deletion or mutation in a single gene. Gene therapy delivers a new or working copy of this gene into human cells, making it possible to treat the root cause of the disease and halt their progression.

With the first two of these pioneering therapies now available in Australia, together we have the opportunity to transform the lives of patients.

Discover more at
www.medhub.com.au

NOVARTIS

AU-15620. March 2021

Australian Journal of Medical Science

ADMINISTRATION

AIMS National Office

Chief Executive: Mr Michael Nolan, BAppSc BA MPhil GradCertMgt FAIMS MASM

Design, formatting and administration: Ms Simona Adochiei
Email: programs@aims.org.au
Website: www.aims.org.au
Telephone: 61 7 3876 2988
Address: PO Box 1911 Milton Qld 4064 Australia

The Australian Journal of Medical Science is the official publication of the Australian Institute of Medical and Clinical Scientists (AIMS).

Circulation 2000 per issue. The Journal is circulated to members in pathology laboratories, universities and research institutes throughout Australia and overseas.

Annual subscription rates are available from AIMS National Office.

Article reprints may be organised on request from AIMS National Office.

Advertising rates are available from AIMS National Office.

Abstraction of the Australian Journal of Medical Science is through the following serial catalogue listings: Australasian Medical Index, Chemical Abstracts, and EMBASE/Excerpta Medica.

The Australian Journal of Medical Science is included on the Australian Research Council ERA 2018 Journal List.

ISSN 1038-1643

Printed by Westminster Eagle Eye Printing, PO Box 161, Paddington Qld 4064.

Design Cover and layout design by Kim Brown, 23 Denman St Exeter SA 5019 kimbo@internode.on.net. Cover photograph courtesy of Prof Ian Gibbins, Flinders Medical Centre, Adelaide.

CONTENTS

May 2021 Vol. 42 No. 2

Case Study

Severe RhD haemolytic disease of the foetus and newborn in RhD negative multigravida 44

Tsz Ki Yip

Original article

The diagnostic challenge in a giant midline neck mass: spindle cell pleomorphic lipoma versus soft tissue tumours 52

Nor Rahimah Aini

Regular Features

Journal-based CPD No. 76 58

Journal-based CPD No. 77 59

Books for review 61

Instructions to authors 63

Australian Council for Certification of the Medical Laboratory Scientific Workforce

68

***Instructions to authors** are available on the AIMS website www.aims.org.au*

Severe RhD haemolytic disease of the foetus and newborn in RhD negative multigravida

Tsz Ki Yip[1,2]

[1] *School of Biomedical Sciences, Faculty of Health, Queensland University of Technology, Brisbane, Queensland*

[2]*QML Pathology, Department of Haematology, Murarrie (Central) Laboratory, Brisbane, Queensland*

Abstract

RhD haemolytic disease of the newborn (RhD HDFN) involves the destruction of foetal red cells by maternal IgG antibodies that enter the foetal circulation during pregnancy. The patient is a RhD-positive male born at 39 weeks due to suspected RhD isoimmunisation. The mother is a RhD-negative 34-year-old female with history of vaginal spotting and missed abortions. History of anti-D prophylaxis was denied but the antibody screening was positive for anti-D at 28 weeks gestation. Severe anaemia with reticulocytosis, positive direct antiglobulin test and anti-D elution from cord blood supported RhD HDFN. The mother required anti-D injection post-delivery and exchange transfusion with phototherapy for the neonate. It was later confirmed that the clinician was not informed of the occurrence of a feto-maternal haemorrhage. This case demonstrates the importance of anti-D prophylaxis after potential sensitising events and the patient's ability to recognise abnormalities and inform their clinician in preventing severe RhD HDFN.

Keywords: *haemolytic disease of newborn, erythroblastosis foetalis, newborn haemolytic disease, rhesus factors, blood group incompatibility*

Introduction

RhD haemolytic disease of the newborn (RhD HDFN) involves the destruction of foetal red cells (RBCs) by maternal IgG antibodies that enter the foetal circulation during pregnancy (Delaney and Matthews 2015). This occurs when a RhD-negative woman carries a RhD positive foetus due to the RHD gene inherited from the father (Hassan *et al* 2019).

If primigravida, and there is no history of sensitisation, the baby is unlikely to be affected as a weak primary response involves elevated IgM antibodies which cannot cross the placenta and is therefore insufficient to cause haemolysis (Hassan *et al* 2019). However, feto-maternal haemorrhage (FMH) events can lead to the development of IgG anti-D if no anti-D prophylaxis administration was given (Hassan *et al* 2019). Once IgG anti-D from the mother crosses the placenta, RhD-positive foetal RBCs become sensitised and are cleared from the circulation by splenic macrophages resulting in haemolysis and hyperbilirubinaemia (Keohane *et al* 2016).

RhD HDFN is now uncommon due to the introduction of anti-D immunoglobulin prophylaxis in the mid-late 1900s. Whilst the survival of affected foetuses is now greater than 90% and routine anti-D prophylaxis is common, cases of RhD HDFN are still reported. We describe a case of RhD HDFN in a newborn of a 34-year-old woman with a history of missed abortions. The case illustrates the importance of anti-D prophylaxis after potential sensitising events in primigravida and multigravida women and the consequences of the lack of patient-clinician communication resulting in severe HDFN.

Address correspondence to:
Tsz Ki (Charlotte) Yip
School of Biomedical Sciences, Faculty of Health,
Queensland University of Technology,
Brisbane QLD 4000
E mail: tk.charlotte.yip@gmail.com

Case history

The patient is a RhD-positive newborn male delivered via a caesarean section at 39 weeks gestation due to suspected RhD isoimmunisation from the mother. The mother is a RhD-negative 34-year-old female with clinical notes of vaginal spotting and a history of missed abortions. The mother denied history of any anti-D prophylaxis.

Results

The mother was confirmed to be AB RhD-negative, with negative antibody screens up until 28 weeks gestation.

Table 1. *Cumulative full blood count results of the patient. Note the marked anaemia with marked macrocytosis at birth and 6 hours after birth. Results for 1 day and 3 days are post-transfusion results. Flagged results are in red. Reference ranges were obtained from Tran (2019).*

Parameter	At Birth	6 hours	1 day	3 days	Reference Range
Haemoglobin (g/L)	54 (---)	56 (---)	149	155	145 – 225
RCC (x10^{12}/L)	0.9 (---)		4.3	4.6	4.0 – 6.6
Haematocrit (L/L)	0.14 (---)		0.42	0.46	0.45 – 0.67
MCV (Hassan *et al*)	157 (+++)		98	100	95 – 121
MCH (pg)			35	34	31 – 37
MCHC (g/L)			354	336	310 – 365
RDW (%)		23.8	31.5		11.0 – 15.5
Platelet Count (x10^{9}/L)	105 (-)	98 (--)	N/A	56 (--)	150 - 550
WCC (x10^{9}/L)	35.3 (++)	30.1 (++)	28.2 (++)	33.0 (++)	9.0 – 34.0
Neutrophils (x10^{9}/L)	15.2 (+)	9.9	12.4 (++)	16.5 (++)	5.0 – 21.0
Lymphocytes (x10^{9}/L)	6.0	6.3	5.6	7.9	2.0 – 11.0
Monocytes (x10^{9}/L)	1.8	0.6	2.5	4.0 (++)	0.2 – 2.5
Eosinophils (x10^{9}/L)	1.41 (++)	2.41 (++)	1.41 (++)	1.32 (++)	0.1 – 1.0
Basophils (x10^{9}/L)	0.00	0.30 (+)	0.00	0.00	0 – 0.20
Metamyelocytes (x10^{9}/L)	4.59	5.72	3.38	0.33	
Myelocytes (x10^{9}/L)	3.88	2.11	0.28	0.99	
Promyelocytes (x10^{9}/L)	0.35	0.90	0.00	0.33	
Blasts (x10^{9}/L)	2.12	1.81	2.54	1.65	
Band Forms (x10^{9}/L)	2.12	0.00	2.82	2.64	0 – 1.4
NRBC/100WBCs	388	628	750	465	0 – 7
Reticulocytes (%)	24.2	56.8			3.0 – 7.0

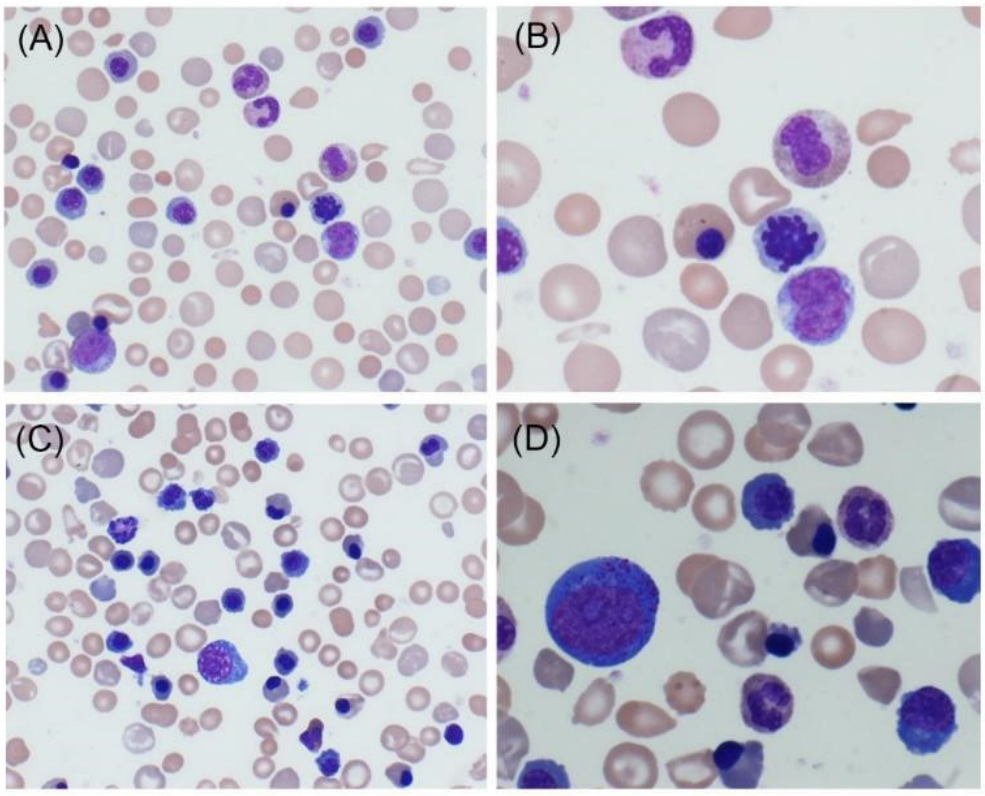


Figure 1. *(A-B) Blood film showing erythrocytes at various maturation stages and apoptotic bodies present at birth of the RhD HDFN neonate. (A&B: Giemsa/Wright stain; x500 and x1000 respectively). (C-D) Blood film showing abundance in nucleated red cells, marked polychromasia and early myeloid cells 6 hours after birth. (C&D: Giemsa/Wright stain; x500 and x1000 respectively). All photomicrographs were taken on a Nikon DSRi2 microscope camera..*

At 28 weeks, antibody screening was positive and anti-D was identified. The anti-D quantitation was <4 IU/mL, which suggested an unlikely risk of RhD HDFN. The anti-D titre was reported to have increased to 4096 by 38 weeks. Paternal antigen typing indicated the father as type A RhD-positive with RhD homozygosity.

From 28 weeks to 39 weeks, the mother and foetus were closely monitored and supported until delivery was appropriate. The full blood count results at birth showed a marked anaemia with normochromic RBCs, mild anisopoikilocytosis, numerous round macrocytes and marked polychromasia (Table 1). A moderate leucocytosis due to mild neutrophilia and moderate eosinophilia was present along with a mild thrombocytopenia. The blood films, in figs 1A-D, showed an abundance of nucleated red cells (nRBCs) at various stages of maturation, and early myeloid cells with a calculated immature:total (IT) ratio of 0.50.

Biochemical testing indicated markedly elevated creatinine and urate, along with increased bilirubin, lactate dehydrogenase and liver enzymes. A low bicarbonate with an increased anion gap and hypoalbuminaemia was also reported. Macroscopically the specimen was moderately haemolysed, severely icteric and slightly turbid. Hyperbilirubinaemia of >400 umol/L (normal reference range: 17-170 umol/L) 2 days after birth was confirmed to be predominantly unconjugated.

Blood banking results confirmed type A RhD-positive for the foetus, and an antibody identification of anti-D resulting in a strong (4+) positive IgG specific response in the direct antiglobulin test (DAT). Cord blood elution was also consistent with strong anti-D sensitisation on neonatal RBCs.

Blood investigations (severe anaemia with reticulocytosis, positive DAT, mother with anti-D isoimmunisation and high quantification of anti-D) supported the diagnosis of severe HDFN due to anti-D isoimmunisation. The mother received 1 vial of prophylactic anti-D within 72 hours post-delivery. Two paediatric packs of O RhD-negative packed cells were transfused to the neonate under Australian and New

Zealand Society of Blood Transfusion guidelines to treat the anaemia, and phototherapy was required for treatment of hyperbilirubinaemia. Monitoring of potential sepsis was undertaken as required and fortunately the condition of the newborn improved.

Discussion

In severe RhD HDFN, the free haemoglobin that results from the spleen trying to clear maternal IgG anti-D bound RBCs is metabolised into bilirubin which is conjugated by the liver. However the haemolysis is so severe that extramedullary haemopoiesis in the liver and spleen occurs resulting in hepatosplenomegaly. In conjunction the connection from the maternal circulation is severed at birth leading to the inability of the foetal liver to maintain bilirubin breakdown (Delaney and Matthews 2015). Bilirubin-induced encephalopathy becomes a risk factor for the neonate, along with kernicterus due to the undeveloped neonatal blood brain barrier that allows for water insoluble unconjugated bilirubin to pass and bind to lipids in the brain (Urbaniak and Greiss 2000). Additionally, hepatic failure leading to hypoalbuminaemia results in oedema and ascites (Delaney and Matthews 2015). In the worst-case scenario, the heart struggles to maintain adequate cardiac output resulting in death (Delaney and Matthews 2015).

It is important to prevent RhD isoimmunisation to avoid erythroblastosis foetalis and kernicterus in neonates affected by RhD HDFN. As a prevention of RhD isoimmunisation, many countries including Australia have guidelines regarding to administration of antenatal and postnatal anti-D immunoglobulin prophylaxis in all non-sensitised RhD-negative women (Hassan *et al* 2019). Based on The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) Guidelines for the use of RhD immunoglobulin (Anti-D) in obstetrics in Australia, pregnant RhD-negative women are given 625 IU RhD prophylaxis injected intramuscularly in week 28 and 34 of gestation (Royal Australian and New Zealand College of Obstetricians and Gynaecologists 2019). A history of missed abortions should correlate with a history of anti-D prophylaxis, however this was denied by the mother in the case described here (Royal Australian and New Zealand College of Obstetricians and Gynaecologists 2019). Past antibody screens may indicate that she was either sensitised previously with low numbers of RhD-positive RBCs that resulted in antibodies too low to detect *in vitro*; or that she developed anti-D in the third trimester. Neither scenario can be excluded, as it was later confirmed that the mother had a feto-maternal haemorrhage during late pregnancy without informing the clinician.

The incidence of FMH is high in each trimester with a 56%, 63% and 71% chance respectively (Geaghan 2011). One study shows that foetal red cell volumes of 0.07 mL, 0.08 mL and 0.13 mL respectively, are sufficient in causing sensitisation (Geaghan 2011). However it has been reported that as little as 0.01 mL with potent Rh antigenicity is sufficient for sensitisation to occur (Geaghan 2011). Without prophylaxis, isoimmunisation occurs in approximately 16% of RhD-negative mothers carrying a RhD-positive foetus (Geaghan 2011).

Prevalence of isoimmunisation varies significantly in women of childbearing potential and the possibility varies between populations based on the prevalence of blood group antigens and the barriers in prenatal testing. Frequency of RhD negativity is estimated at 15-17% among Europeans and North Americans, 3-8% in Africans and Indians, and 0.1-0.3% in the Asian population (Webb and Delaney 2018). However despite its low RhD negativity prevalence, the rates of anti-D in women of childbearing potential are as high as 2-12% in Africa (Webb and Delaney 2018). This is due to limitations in prenatal testing in contrast to a separate study in the United States that reports a rate of RhD isoimmunisation of 1.5% third trimester prophylaxis reducing this figure to approximately 0.1% (Geaghan 2011).

It has been reported that 83% of severe HDFN cases are due to previous pregnancy, which emphasises the importance of anti-D prophylaxis in not only multigravida women but also primigravida women post-delivery and after potential sensitising events (Webb and Delaney 2018). Recognition of potential sensitising events including miscarriages, trauma, missed abortions and transfusions should be diligently recorded, along with prophylactic administration. Additionally, the importance of patients being able to recognise abnormalities in pregnancy, and the ability to trust in their clinicians is critical in identifying and preventing severe RhD HDFN.

Acknowledgements

The author would like to thank Mr Fabian Lim (QML Pathology, Haematology) and Mrs Lyndall Dial (QML Pathology, Haematology) for their approval in choosing this case study. Gratitude is extended to Dr Jeremy Robertson (QML Pathology, Haematology) and Mrs Andrea Shaw (QML Pathology, Haematology) for their assistance in gathering results for this case study. Thank you also to Mr Steven Weier (Queensland University of Technology) and Mr Paul Shield (Queensland University of Technology) for their encouragement and feedback in the writing of this case study and its submission.

References

Delaney M & Matthews DC 2015. Hemolytic disease of the fetus and newborn: managing the mother, fetus, and newborn. *Hematology Am Soc Hematol Educ Program* 2015: 146-51.

Geaghan SM 2011. Diagnostic laboratory technologies for the fetus and neonate with isoimmunization. *Semin Perinatol* 35: 148-54.

Hassan MZ, Iberahim S, Abdul Rahman WSW, Zulkafli Z, Bahar R, Ramli M, Mohd Noor NH & Mustaffa R 2019. Severe anti-D haemolytic disease of fetal and newborn in rhesus D negative primigravida. *Malays J Pathol* 41: 55-58.

Keohane EM, Smith L & Walenga JM 2016. Extrinsic Defects Leading to Increased Erythrocyte Destruction—Immune Causes. In: KEOHANE, E. M. (ed.) Rodak's Hematology: Clinical Principles and Applications. 5th ed. St. Louis, Missouri: Elsevier Saunders.

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG). Guidelines for the use of Rh(D) Immunoglobulin (Anti-D) in obstetrics in Australia. https://www.ranzcog.edu.au/RANZCOG_SITE/media/RANZCOG-MEDIA/Women's%20Health/Statement%20and%20guidelines/Clinical-Obstetrics/Guidelines-for-the-use-of-Rh(D)-Immunoglobulin-(Anti-D)-(C-Obs-6)-Review-November-2015.pdf?ext=.pdf Accessed June 14, 2019.

Tran, D. Haematology Reference Range- Index. http://qml-intranet.hc.int/Document?2756 Accessed June 21, 2019.

Urbaniak SJ & Greiss MA 2000. RhD haemolytic disease of the fetus and the newborn. *Blood Rev* 14: 44-61.

Webb J & Delaney M 2018. Red Blood Cell Alloimmunization in the Pregnant Patient. *Transfus Med Rev* 32: 213-219.


What doesn't kill you makes you stronger!
AIMS Tropical Division
Annual Scientific Meeting
Special Guest Speaker
Dr Norman Swan
celebrityspeakers
June 11th - 13th 2021
The Ville Resort Townsville
Come and enjoy the tropics this winter
Don't forget to mention the conference when booking
AIMS Tropical Division
Australian Institute of Medical and Clinical Scientists
Abstracts due April 1st 2021

NSW North Coast Division Conference 2021

"Always look on the bright side of life.....with Medical Science !!!"

*** *Save the date* ****

12 - 14 November 2021

Venue (to be advised), Armidale, NSW

EVENT INFORMATION & REGISTRATION DETAILS COMING SOON.....

Dates: 12-14 November 2021

Armidale

Registrations Open

20th Jul 21 12:00 PM

Registrations Close

30th Oct 21 11:30 AM

Cost TBA

AIMS NSM 2021

EARLYBIRD REGISTRATIONS

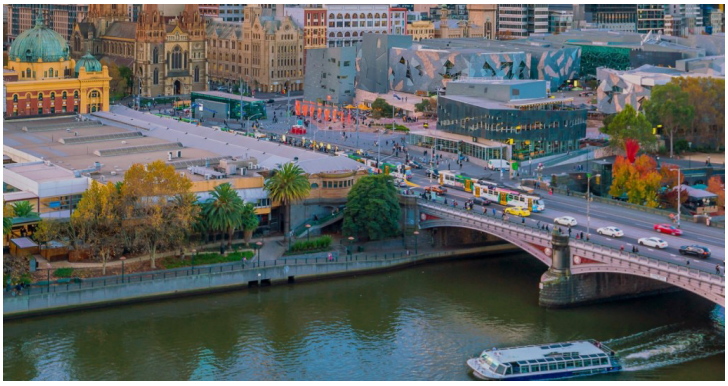
NOW OPEN!!

Earlybird registrations close on 30 June 2021

https://aomevents.eventsair.com/aims-national-scientific-meeting-2021/

AIMS 2021 will be a hybrid event, comprising both an in-person meeting and a virtual meeting. The meeting will still be held over three days, incorporating a comprehensive program that will appeal to all laboratory staff including phlebotomists, pre-analytical staff, technicians, scientists, clinical scientists, medical staff, and laboratory managers. Going hybrid will allow people who, for a variety of reasons, will not be able to join the in-person meeting in Melbourne.

Virtual delegates will be able to join the online sessions at scheduled times, while still being able to pose questions and interact with the presenters. Recorded sessions will also be available on demand post meeting.



Australian Institute of
Medical and Clinical Scientists
National Scientific
Meeting 2021

2021
RE-VISION
FOR THE FUTURE
30 AUG - 1 SEPT

The diagnostic challenge in a giant midline neck mass: spindle cell pleomorphic lipoma versus soft tissue tumours

Nor Rahimah Aini [1], Nurul Atiah Mohd Ali [2], Maryam Ahamad Sharifuddin [2],

Sharifah Emilia Tuan Sharif [2], Norhafiza Mat Lazim[1]

[1] Department of Otorhinolaryngology- Head & Neck Surgery, School of Medical Sciences, Universiti Sains Malaysia, Health Campus, Kubang Kerian, Kelantan, Malaysia

[2] Department of Pathology, School of Medical Science, Universiti Sains Malaysia, Health Campus, Kubang Kerian, Kelantan, Malaysia

Abstract

Spindle cell/pleomorphic lipomas are uncommon subtypes of benign lipomatous tumours. They typically occur in the subcutaneous fatty tissue of the posterior neck, back and shoulder. They rarely affect the anterior neck region, especially excessive sizes (greater than 10 cm in length or weighing over 1000 grams). In a long-standing case it is critical to exclude soft tissue tumours or malignant transformation or liposarcoma because of different therapeutic approaches. Thus detailed histopathological examination is a pre-requisite. We report a rare case of a giant spindle cell/pleomorphic lipoma of the anterior neck. The tumour was successfully removed surgically. The histopathology revealed spindle cell/pleomorphic lipoma with the presence of atypical cells. The diagnostic challenge is highlighted, especially the difficulties in distinguishing spindle cell/pleomorphic lipoma from liposarcoma as this influences the different treatment regimens required. No recurrence has been noted up to eight months after surgery and the patient has had an excellent functional and cosmetic outcome.

Keywords: spindle cell/pleomorphic lipoma; liposarcoma; neck mass

Introduction

Adipocytic tumours represent an extremely heterogenous category of clinical and morphologically distinct lesions. These types of tumours often present a diagnostic challenge, especially when dealing with a rare subtype that includes spindle cell/pleomorphic lipomas (SC/PLs). Lipomas are frequently identified in the head and neck area as soft tissue tumours. The diagnosis of atypical lipomatous tumours (ALT) should always be excluded when the tumour reaches an abnormal size of greater than 10 cm in length or weighing more than 1000 grams (Copcu and Sivrioglu 2005). There is a high likelihood of malignant transformation in a long-standing mass, which determines specific treatment approaches. A rare case of a giant spindle cell/pleomorphic anterior neck lipoma has been identified and, to our knowledge, it is the first reported in the literature for this rare lipomatous tumour entity with abnormal size in the anterior region of the head and neck.

Address correspondence to:
A/P Dr Norhafiza Mat Lazim
Department of Otorhinolaryngology
School of Medical Sciences,
Universiti Sains Malaysia,
Health Campus, Kubang Kerian, Kelantan, Malaysia
E-mail: norhafiza@usm.my

Case report

A 49-year-old man presented with a two year history of a painless anterior neck swelling which was slowly progressing in size. The lesion started at the left submental region and extend to the right submental region as the mass grew bigger. Local examination revealed a huge well-defined anterior neck mass measuring 37cm x 27cm in transverse and longitudinal dimensions, respectively. The mass extended from the submental region, inferiorly to the cricoid cartilage, and laterally up to the anterior border of sternocleidomastoid muscle. The mass appeared soft to firm, mobile, non-tender and not attached to the underlying skin (Figure 1). No other lymphadenopathy was noted.

Contrast enhanced computer tomography (CT) of the neck showed a large well- defined, minimally enhancing,

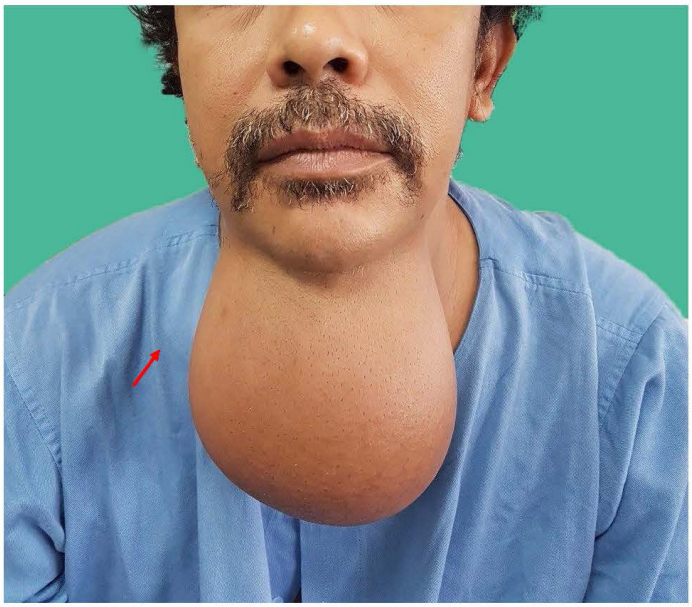


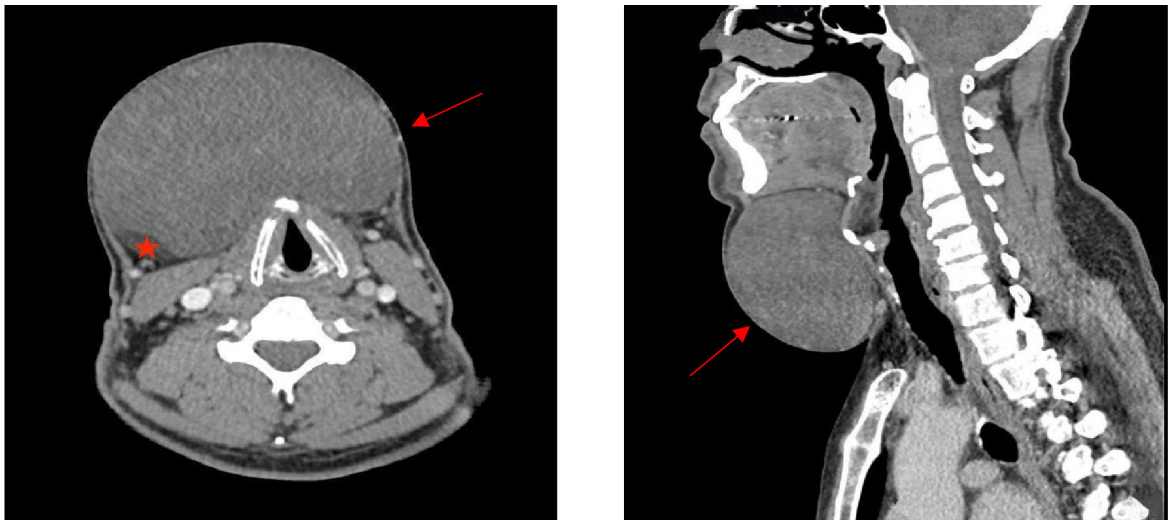
Figure 1. *Pre-operative photograph of patient with huge submental mass (red arrow).*

slightly heterogenous mass at the anterior aspect of neck and extending from the submandibular region to the supraclavicular region. The mass measured approximately 11.7cm x 10.3cm x 7.5cm and had fat components; calcifications were not seen (Figure 2). No clear fat plane was seen between the mass and platysma muscle anteriorly and posteriorly the mass mildly compressed the larynx to the left.

A surgical excision was performed under general anaesthesia. A planned skin excision was marked on the skin surface. The anterior neck was exposed and the skin incision following the skin tension lines was made. The platysma muscle was divided and superior and inferior flaps were raised. A well capsulated mass was located at midline of the anterior neck. The base of the mass extended from the hyoid bone superiorly to the thyroid cartilage inferiorly and laterally to the medial border of sternocleidomastoid muscle. No cervical node enlargement was noted and the muscular diaphragm of mouth was preserved. After deliverance of the mass, the inferior flap was brought over the superior flap and the redundant skin was then removed.

Post-operative histological examination showed a round intact encapsulated mass, measuring 12.6cm x 11.0cm x 7.0cm and weighing 1083grams. The outer surface was smooth with prominent vasculature (Figure 3). Dissection revealed a circumscribed and encapsulated lesion composed of predominantly homogenous greyish white, myxoid to gelatinous material. A few small yellowish foci ranging from 1.0-1.4 cm were seen. There were no solid areas or papillary lesions noted. Microscopically, the lesion is was composed of two main components comprising adipocytes and spindle cells. The adipocytes were predominantly uniform with focal areas of slight variation in sizes, in the background of myxoid to pinkish collagen embedded spindle cells. The spindle cells were bland with no specific pattern. Scattered floret cells with occasional cells showing mild atypia were seen , however, no definite lipoblasts were detected. Thick ropey collagen and scattered mast cells were seen in the background which were the clue for the diagnosis (Figure 4). In some areas, loose myxoid stroma with prominent, arborizing, thick walled blood vessels were noted. The spindle cells showed diffuse and strong positivity for CD34, but negative for S100.

The post-operative course was uneventful and the patient was discharged on the third postoperative day. He was seen at one week postoperatively with no functional impairment and good cosmetic result (Figure 5). He had no evidence of recurrence up to twelve months postoperatively.



A B

Figure 2. *Contrasted Enhanced Computer Tomography imaging. A large well-defined minimally enhancing slight heterogenous mass at anterior aspect of neck with fat components (red asterisk *) in axial (A) and sagittal view (B).*

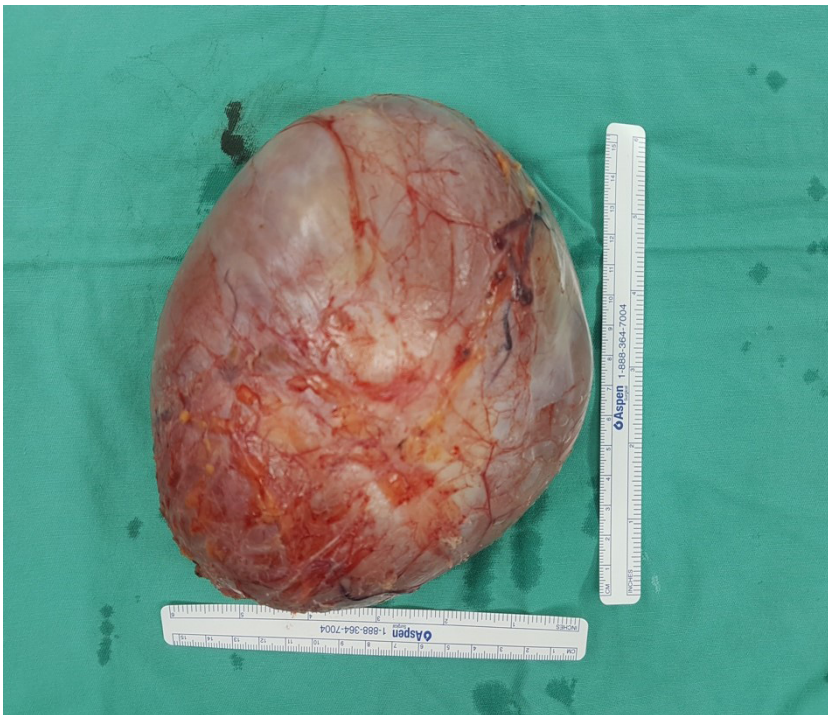


Figure 3. *On gross dissection, the mass appeared round with capsule intact. The outer surface was smooth with prominent vasculature.*

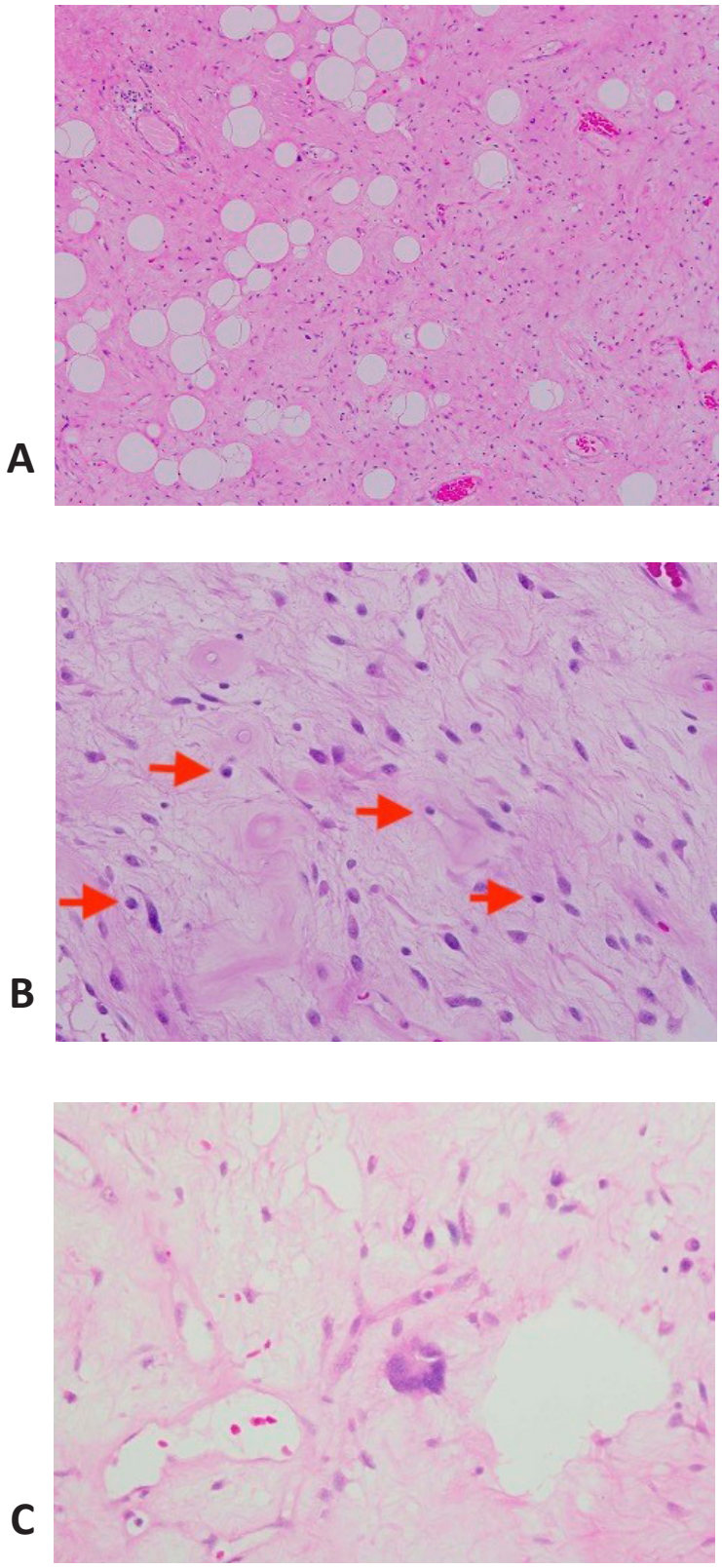


Figure 4. *Microscopic features of the case presented. (A) Admixture of two components which are adipocytes and spindle cells (red arrow) (H&E, X200). (B) The spindle cells are bland-looking and embedded within a myxoid background. Thick rope-like collagen and mast cells (arrows) are also seen (H&E, X400). (C) Occasional floret cells are present (H&E, X400).*

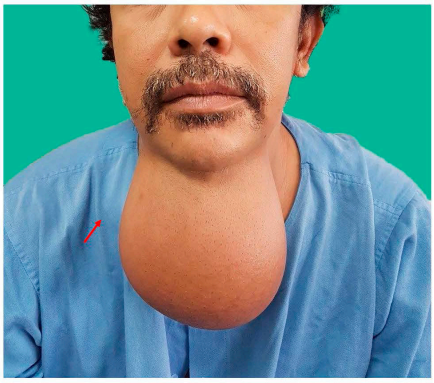


Figure 5. *Three- month follow up photographs. The previous incision scar is currently well covered with a beard.*

Discussion

Spindle cell lipoma is a discrete lipoma subtype with a distinct morphology and molecular profile. Several genetics studies in the 1990s showed that spindle cell lipoma and pleomorphic lipoma share the same defects, specifically losses of 16q and 13q (Van Treeck and Fritchie 2019). Recent cytogenic research revealed that the loss of protein expression of retinoblastoma (Rb) associated with 13q deletions found in SC/PLs helped to distinguish SC/PLs from ALT or other histologic mimics, such as hibernoma, deep angiomyxoma and angiomyofibroblastoma (Chen *et al* 2012).

The majority of SC/PLs usually occur in males between the fourth and eighth decades of life as a solitary, painless, subcutaneous mass in the posterior neck, shoulder and upper back, whereas 20% of SC/PLs occur elsewhere including the extremities, face and trunk (Goldblum *et al* 2013). Most tumours appear as a well circumscribed subcutaneous mass and seldom exceed 6 cm. Liposarcoma, on the other hand, occurs more often as a larger mass at a deep position, such as in the retroperitoneum or in the musculature of the lower extremities with infiltrative borders (Shmookler and Enzinger 1981). In contrast with the posterior neck, the existence of this tumour in the anterior neck is rare and to our knowledge, only one case has been recorded in an almost similar tumour position (Munakata *et al* 2020). Although the tumour may appear to be aggressive due to its enormous mass and unusual location, the microscopic features actually portray a non-aggressive result instead, which is indeed a diagnostic challenge for this particular case.

SC/PL tend to be firm, yellow-pink to yellowish-grey, well circumscribed and often encapsulated macroscopically. Usually, the average size is 4-5 cm in diameter, although there was a case recorded up to 14 cm (Eryilmaz *et al* 2007). Microscopically, SC/PLs have diversity in morphology and can display characteristics of spindle cell lipomas and pleomorphic lipomas. The real problem is when just one predominant characteristic is displayed. The function of immunohistochemistry (IHC) is crucial and significant when this is the case. SC/PL generally displayed a good diffuse IHC profile of CD34 positive/S-100 negative. Occasional lipoblasts may be observed in SC/PL, but there is typically no infiltrative development, necrosis, atypical spindle cells, bizarre pleomorphic cells, pleomorphic lipoblasts and substantial mitotic activity (Van Treeck and Fritchie 2019). The lipomatous atypical spindle cell tumour is the new terminology that was recently introduced to describe SC/PL with atypical spindle cells, loss of Rb gene and immunonegative for MDM2/CDK4 by IHC or fluorescence in-situ hybridization (FISH). It was therefore inferred that this was the SC/PL spectrum, but not ATL by molecular classification (Cheah *et al* 2015). This case, however, has a typical SC/PL characteristic with the inclusion of adipocyte-spindle cell admixture in the collagen context. No compelling evidence of atypical stromal cells, infiltrative growth patterns, necrosis or repeated mitosis was found in uncommon places, despite the large scale tumour.. In this case the presence of an encapsulated lesion, dense ropey collagen, distributed mast cells backed by CD34 positive/S-100 negative IHC profile strongly supports the diagnosis of SC/PL (Cheah *et al* 2015).

As to the difference in the ratio of adipose and non-adipose elements, SC/PL has variable imaging appearances and no pathognomonic for diagnosis (Bancroft *et al* 2003). Several

imaging characteristics have been shown to increase the probability of sarcoma-containing fat, such as large lesion size (more than 10 cm), thick septation, nodular or mass-like areas, and decreased macroscopic fat composition (less than 75%), although calcification is three times more likely to occur in liposarcoma in CT scans (Jelinek *et al* 2020; Younan *et al* 2018). Lee *et al* (2013) recorded almost identical CT scan images to those we obtained; well-defined, non-infiltrative, heterogeneous hypodense mass with limited enhancement, lack of calcification and associated cervical lymphadenopathy (Lee *et al* 2013). Imaging characteristics and demographics, however, are not sufficient to distinguish between SC/PL and the ALT. The diagnosis should be determined immunohistopathologically with perhaps ancillary cytogenetic analysis in problem cases (Younan *et al* 2018).

The gold standard treatment procedure for SC/PL is surgical excision with clear margins. With low recurrence rate, surgery provides excellent results (Digregorio *et al* 1992). Some literature indicates simple enucleation and blunt dissection, especially when dealing with the position of the tumour near vital structures (Bryant 1987). Because of its benign nature, radical deforming surgery must not be considered for SC/PLs (Digregorio *et al* 1992). This differs from the therapeutic approach for liposarcoma where the only curative means for localised disease is complete surgical resection. The outcome of adjuvant therapy including radiation, chemotherapy and targeted drug therapy for metastatic and unresectable disease remains controversial (Guan *et al* 2015). A distinction between SC/PL and liposarcoma is therefore important in order to prevent needless overtreatment.

Conclusion

In summary, we report a rare case of giant SC/PL of the anterior neck. The varied morphology and structural characteristics with SC/PL's make both imaging and immunohistopathology a diagnostic challenge. To distinguish this tumour from liposarcoma, a biopsy with ancillary laboratory assessment tools and immunohistochemical staining is indicated. This is important because the management of benign lipoma varies from liposarcoma the latter involving clearance of the neck nodes as well as adjuvant radiation.

Acknowledgements

Nil.

References

Bancroft LW, Kransdorf MJ, Peterson JJ, Sundaram M, Murphey MD, O'Connor, MI. 2003. Imaging characteristics of spindle cell lipoma. *Am JRoentgenol* **181:** 1251-54.

Bryant J. 1987. Pleomorphic lipoma of the bulbar conjunctiva. *Ann Ophthalmol* **19**: 148-49.

Cheah A, Billings S, Goldblum J, Hornick J, Uddin N, Rubin B. 2015. Spindle cell/pleomorphic lipomas of the face: an under-recognized diagnosis. *Histopathology,* **66***:* 430-37. doi: 10.1111/his.12548.

Chen BJ, Mariño-Enríquez A, Fletcher CD, Hornick JL. 2012. Loss of retinoblastoma protein expression in spindle cell/pleomorphic lipomas and cytogenetically related tumors: an immunohistochemical study with diagnostic implications. *Am J Surg Pathol* **36***:* 1119-28.

Copcu E, Sivrioglu N. 2005. Posterior cervical giant lipomas. *Plast Reconstr Surg 115*: 2156-57.

Digregorio F, Barr RJ, Fretzin DF. 1992. Pleomorphic lipoma: case reports and review of the literature. *J Dermatol Surg Oncol* **18** 197-203.

Eryilmaz M, Yigit T, Ozturk G, Safali M, Aksu AY. 2007. Giant spindle cell lipoma of the posterior neck. *Dermatol Surg* **33**: 1258-1261, doi: 10.1111/j.1524-4725.2007.33263.x

Goldblum JR, Weiss SW, Folpe AL. 2013. *Enzinger and Weiss's Soft Tissue Tumors E-Book.* Philadelphia: Elsevier.

Guan Z, Yu X, Wang H, Wang H, Zhang J, Li G, Cao J, Teng L. 2015. Advances in the targeted therapy of liposarcoma. *Onco Targets Ther* **8:** 125.

Jelinek J, Wu A, Wallace M, Kumar D, Henshaw R, Murphey M, Van Horn A, Aboulafia A. 2020. Imaging of spindle cell lipoma. *Clin Radio* l75: 396.e15-396.e21.

Lee HK, Hwang SB, Chung GH, Hong KH, Jang KY. 2013. Retropharyngeal spindle cell/pleomorphic lipoma. *Korean J Radiol,* **14**: 493-496. doi: 10.3348/kjr.2013.14.3.493.

Munakata K, Asoda S, Usuda S, Miyashita H, Soma T, Hasegawa T, Ogawa C, Ohyama S, Yamauchi T, Kawaida M. 2020. A case of spindle cell/pleomorphic lipoma on inferior border of the mandible. *J Oral Maxillofacial Surg Med Pathol,* **32**, 49-52.

Shmookler BM, Enzinger FM. 1981. Pleomorphic lipoma: a benign tumor simulating liposarcoma. A clinicopathologic analysis of 48 cases. *Cancer,* **47:** 126-33.

Van Treeck BJ, Fritchie KJ. 2019. Updates in spindle cell/pleomorphic lipomas. *Semin Diagn Pathol,* **36**: 105-11. doi: 10.1053/j.semdp.2019.02.005.

Younan Y, Martinez A, Reimer N, Edgar M, Gonzalez F, Umpierrez M, Subhawong T, Singer AD. 2018. Combined classical spindle cell/pleomorphic lipoma spectrum imaging and clinical data. *Skeletal Radiol* **47**: 51-59.

Australian Professional Acknowledgement of Continuing Education (APACE)

3 APACE credits per set of questions will be awarded if at least 8 out of 10 questions are answered correctly.
24 credits maximum per accreditation period claim.

Journal-based CPD No. 76
Page 1 of 1

Questions relating to the article *'Severe RhD haemolytic disease of the foetus and newborn in RhD negative multigravida'* at page 44 of this issue.

1.	RhD Haemolytic Disease of the Newborn (RhD DHFN) involves the destruction of foetal red cells by maternal IgG antibodies that enter the foetal circulation during pregnancy.	True/False
2.	This case can not demonstrate the importance of anti-D prophylaxis after potential sensitising events.	True/False
3.	Feto-maternal haemorrhage (FMH) events can lead to the development of IgG anti-D if no anti-D prophylaxis administration is not given.	True/False
4.	RhD HDFN is now uncommon due to the introduction of anti-D immunoglobulin prophylaxis in the mid-late 1800s.	True/False
5.	The case illustrates the importance of anti-D prophylaxis after potential sensitising events in primigravida and multigravida women.	True/False
6.	From 22 weeks to 35 weeks, the mother and foetus were closely monitored and supported until delivery was appropriate.	True/False
7.	Biochemical testing indicated markedly elevated creatinine and urate, along with increased bilirubin, lactate dehydrogenase and liver enzymes.	True/False
8.	Frequency of RhD negativity is estimated at 15-17% among Europeans and North Americans, 3-8% in Africans and Indians, and 0.1-0.3% in the Asian population.	True/False
9.	The importance of patients being able to recognise abnormalities in pregnancy, and the ability to trust in their clinicians is critical in identifying and preventing severe RhD HDFN.	True/False
10.	Prevalence of isoimmunisation varies significantly in women of childbearing potential and the possibility varies between populations based on the prevalence of blood group antigens and the barriers in prenatal testing.	True/False

Name: _____

Email: _____

Please photocopy this page or print it from the electronic AJMS which is stored in the AIMS 'Member Centre' under the heading 'Journal' at www.aims.org.au. ***Circle your answers, then scan, and enter them as activities in the APACE diary under 'My CPD record'.***

https://www.aims.org.au/online-diary

Australian Professional Acknowledgement of Continuing Education (APACE)

3 APACE credits per set of questions will be awarded if at least 8 out of 10 questions are answered correctly.
24 credits maximum per accreditation period claim.

Journal-based CPD No. 77
Page 1 of 1

Questions relating to the article *'The diagnostic challenge in a giant midline neck mass: spindle cell pleomorphic lipoma versus soft tissue tumours'* at page 52 of this issue.

1.	Spindle cell/pleomorphic lipomas are uncommon subtypes of malign lipomatous tumours.	True/False
2.	Spindle cell/pleomorphic lipomas typically occur in the subcutaneous fatty tissue of the posterior neck, back and shoulder.	True/False
3.	The histopathology revealed spindle cell/pleomorphic lipoma with the presence of atypical cells.	True/False
4.	Adipocytic tumours represent an extremely heterogenous category of clinical and morphologically distinct lesions.	True/False
5.	Lipomas are frequently identified in the head and neck area as soft tissue tumours.	True/False
6.	The mass measured approximately 11.7cm x 10.3cm x 7.5cm and had fat components; calcifications were present.	True/False
7.	The anterior neck was exposed and the skin incision following the skin tension lines was made.	True/False
8.	A cervical node enlargement was noted and the muscular diaphragm of mouth was preserved.	True/False
9.	Post-operative histological examination showed a round intact encapsulated mass, measuring 12.6cm x 12.0cm x 7.0cm and weighing 1083 grams.	True/False
10.	SC/PL tend to be firm, yellow-pink to yellowish grey, well circumscribed and often encapsulated macroscopically.	True/False

Name: _____

Email: _____

Please photocopy this page or print it from the electronic AJMS which is stored in the AIMS 'Member Centre' under the heading 'Journal' at www.aims.org.au. ***Circle your answers, then scan, and enter them as activities in the APACE under 'My CPD record'.***

https://www.aims.org.au/online-diary

Australasian

Professional

Acknowledgement of

Continuing

Education

Recognition of
Continuing Professional Development

Australian Institute of
Medical and Clinical Scientists

www.aims.org.au www.apace.org.au

The APACE (Australasian Professional Acknowledgement of Continuing Education) scheme is a voluntary programme that recognises continuing education, formal courses and a wide range of professional activities which contribute to your professional growth.

The healthcare industry is undergoing rapid change. We are expected to keep our knowledge and skills up to date to enable us to perform to the highest professional standard. The APACE scheme provides a method by which your professional activities are recognised.

Changes to APACE due to COVID-19 pandemic

An APACE Certificate is usually awarded on attaining 100 CEU credits within a two year period.

However, with limited opportunities for CPD due to covid-19 restrictions, the time frame has been extended to three years. Also, the limits for on-line learning, webinars, structured and journal reading have been removed.

These changes will remain in place until CPD activities return to normal across the country.

https://www.aims.org.au/apace

BOOKS FOR REVIEW

Following is a list of books available for review by resource consultants and members of the Institute with particular expertise in the field. The reviewer is invited to retain the complimentary copy of the book once the review is received.

As per our agreement with the book publishing companies, complimentary books are submitted to the Institute provided that all reviews are published in the Australian Journal of Medical Science. These reviews must be of a high quality as buying decisions and the reputation of the book and author are important considerations.

Books not requested will be allocated at discretion of the Editors for the Australian Journal of Medical Science. Reviews should be 300 to 700 words depending on the volume of the book. Time limit for return of review is **six** weeks.

Please send your request to: Australian Institute of Medical Scientists PO Box 1911 Milton Qld 4064
Tel: (07) 3876 2988 Fax: (07) 3876 2999 Email: programs@aims.org.au

1. **Bifidobacteria: Genomics & Molecular Aspects** edited by B. Mayo, & D. Van Sinderen. Caister Academic Press. xii + 260 pages.

2. **Medicine and Sport Science Volume 55: Cytokines, Growth Mediators & Physical Activity in Children during Puberty** edited by J. Jurimae, A.P. Hills & T. Jurimae. Karger. viii+178 pages.

3. **Digestive Diseases The Keys to IBD 2010: Treatment, Diagnosis & Pathophysiology.** Edited by G. Rogler & W. Sandborn. Karger. 188 pages.

4. **Else Kröner-Fresenuis Symposia Volume 1: Molecular Mechanisms of Adult Stem Cell Aging** edited by K.L. Rudolph. Karger. xii+108 pages**.**

5. **Endocrine Development Volume 24: Hormone Resistance and Hypersensitivity** edited by M. Maghnie, S. Loche, M. Cappa, L. Ghizzoni & R. Lorini. Karger. viii + 160 pages.

6. **Frontiers of Hormone Research Volume 41: Endocrine Tumor Syndromes and Their Genetics** edited by C.A Stratakis. Karger. xii + 187 pages.

7. **Frontiers of Hormone Research Volume 39: Kallmann Syndrome & Hypogonadotropic Hypogonadism** edited by R. Quinton. Karger. x+174 pages.

8. **Generic: The Unbranding of Modern Medicine** by Jeremy A. Greene. John Hopkins University Press. 368 pages.

9. **Human Pathogenic Fungi: Molecular Biology and Pathogenic Mechanisms** edited by Derek J. Sullivan & Gary P. Moran, Caister Academic Press. x + 342 pages.

10. **Internal Medicine: A Doctor's Stories** by Terrence Holt. Black Inc. 273 pages.

11. **Intolerant Bodies: A Short History of Autoimmunity** by Warwick Anderson and & Ian R. Mackay. John Hopkins University Press. 250 pages

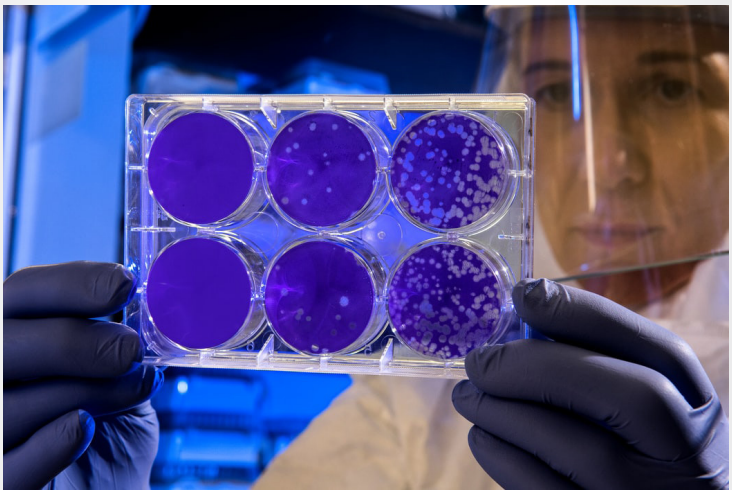
12. **Lyme disease and relapsing fever spirochetes** edited by Justin D. Radolf and D. Scott Samuels. Caiser Academic Press. 760 pages

13. **More Than Hot: A Short History of Fever** by Christopher Hamlin. John Hopkins University Press. 400 pages.

14. **Pediatric and Adolescent Medicine Volume 19: Metabolic Syndrome and Obsesity in Childhood and Adolescence** edited by W. Kiess, M. Wabitsch, C. Maffeis, A.M. Sharma. Karger. x + 202 pages.

15. **Phage Therapy - Current Research and Applications** edited by Jan Borysowski, Ryszard Miedzybrodzki & Andrzej Gorski. Caister Academic Press. 368 pages.

16. **Shigella: Molecular and Cellular Biology** edited by William D. Picking & Wendy L. Picking. Caister Academic Press. 280 pages.



Australian Institute of Medical Scientists

Immunohaematology Quality Assurance Program

Meet your accreditation requirements with the AIMS Immunohaematology Quality Assurance Program (QAP)

- Designed to suit both small and large laboratories;
- Includes blood grouping, antibody screening/identification and compatibility testing;
- Runs bi-monthly starting in July at the beginning of each financial year;
- The reports provide participants' own results along with graphical representation of results of their peers (de-identified) allowing for easy comparison and analysis.

For enrolment enquiries please contact the AIMS National Office:

E-mail: programs@aims.org.au

Phone: (07) 3876 2988

Postal Address: PO Box 1911, MILTON QLD 4064, AUSTALIA

Or contact Steve Mackay:

E-mail: aimsqap@dspl.com.au

AUSTRALIAN JOURNAL OF MEDICAL SCIENCE

Instructions to authors

The following instructions are based on the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals", also known as the Declaration of Vancouver, and on the *Australian Government Style manual: for authors, editors and printers*, 6th edition, 2002. URLs were correct on September 29th, 2008.

Manuscripts that do not fully comply with the following 'Instructions to Authors' may be returned for revision before they are considered for publication.

The *Australian Journal of Medical Science (AJMS)* will consider for publication any paper relevant to the field of Medical Science. Disciplines include blood banking, clinical biochemistry, haematology, histopathology, immunology, microbiology and molecular biology. Areas of general interest to medical laboratory scientists, including toxicology, epidemiology, public and community health, and professional and management issues will also be considered.

Papers published in the *AJMS* are in the form of:

- Review Articles
- Original Articles
- Brief Communications
- Technical Notes
- Case Studies
- Letters to the Editor
- Book Reviews

Articles submitted for publication are understood to be offered only to the *AJMS* and those accepted become the property of the *AJMS*.

All individuals listed as authors must have made a substantial contribution to the conception and design of the study, the acquisition of data or the analysis and interpretation of data; the drafting of the article or revising it critically for important intellectual content; and final approval of the version to be published. The corresponding author must take responsibility for obtaining permission from all the authors for the submission of any version of the manuscript and for any changes in authorship.

When the manuscript is submitted the authors must disclose any potential conflict of interest and/or commercial support.

Requirements & preparation of manuscripts

General

Articles should be submitted in electronic format to programs@aims.org.au. If an article is too large to be submitted by email, it should be submitted on an or USB stick.

Number pages consecutively commencing with the title page.

Arrange the article in the following sequence:

- Title page
- Abstract and key words
- Main Text
- Acknowledgements
- References
- Tables - each table, complete with title and footnotes, on a separate page
- Legends for illustrations.

Authors should ensure that their manuscript communicates their ideas and concepts simply and clearly so that the article is easily read and understood. Authors are strongly recommended to refer to the recommendations on reporting standards as outlined in the statements and checklists of the CONSORT group (see: http://www.consort-statement.org/) and similar groups such as STARD (see: http://www.stard-statement.org/). The principles outlined in these standards may be used as general guidelines and not just as applied to clinical trials and diagnostic studies.

Title page

The title of the article should not exceed three lines (40 characters per line), including punctuation and spacing. All authors must be identified on the title page (e.g., William Smith, Susan Yeo, ..."). Where applicable, the title page should also include the name of the institution with which each author is affiliated and to which the work should be attributed. In the case of multiple authors, the name, postal address, email address, telephone and facsimile number of the author responsible for correspondence relating to the manuscript should be indicated.

Abstract & keywords

The abstract should be approximately 150 words and should make sense when read alone or in conjunction with the article. The abstract should be a concise overview that describes the important details of the article including the purpose of the study/investigation, basic procedures (study subjects/experimental animals/observational and analytic methods) and the results and principal conclusions. New and important aspects of the work and its implications may also be included. References should not be included.

Three to ten keywords may be listed. Authors are advised to comply with the terms from the Medical Subject Headings (MeSH) list from Index Medicus (see http://www.nlm.nih.gov/mesh/). Keywords should be given below the Abstract.

Text

The style of writing should conform to acceptable English usage. Do not use slang, medical jargon or unnecessary abbreviations. Accepted spelling is the first choice given in the latest edition of the Macquarie Dictionary.

Wherever possible, observational or experimental articles should be divided into sections headed:

- Introduction
- Materials and methods
- Results
- Discussion
- References

For other types of articles such as commentaries, reports and reviews, use an appropriate format or consult the Editors for guidance. Do not include a separate section for conclusions, these should be given in the discussion.

Introduction

Clearly state the purpose of the article leading the reader from the known to the unknown. Summarise the rationale for the study and state the question to be answered as appropriate. Give only strictly pertinent references, and do not review the subject extensively.

Materials & methods

Present the materials and methods in a logical sequence. Describe the selection of the observational or experimental subjects (patients or experimental animals, including controls) clearly. Notification of ethics approval must be given where relevant. Identify the methods, apparatus and procedures in sufficient detail to allow other workers to reproduce the results. Give references to established methods, including statistical methods. Adequately describe new or substantially modified methods. Identify precisely all drugs and chemicals used, including generic name(s), dosage(s), and route(s) of administration. Do not identify patients or hospitals without consent.

Results

Present the results in the same sequence as given in the Materials and methods; use tables and illustrations where these will help the reader understand the work being presented. Do not repeat in the text all the data in the tables or illustrations.

Discussion

Indicate the new and important aspects of the study and emphasise the conclusions that follow. Do not repeat in detail data given in the Results section and do not add new data. Include in the Discussion the implications of the findings and their limitations and compare the observations to other relevant studies. Recommendations may be included if appropriate. Link the conclusions with the goals of the study and answer the experimental question stated in the Introduction. However, avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such.

Acknowledgements

Acknowledge individuals who have made substantial contributions to the study including technical work and financial support. Authors are responsible for obtaining consent from all the individuals acknowledged by name as inclusion may be interpreted as an endorsement of the article's contents.

References

The AJMS uses a modified Harvard System (author-date system).

Throughout the body of the manuscript cite the author/s name and the publication year in parentheses as in the following examples:

(i) Research in this area (Jones 1999) ...

(ii) It has been successfully demonstrated that (Smith and Brown 1981; Auteur 1995; Scienziato *et al* 2007).

(iii) Following further investigation, Wetenschapper (2002 highlighted the difficulties inherent in...

Where there are three or more authors, acknowledge only the first author, e.g., (Smith *et al* 2007). For two authors the following style should be used: (Smith and Brown 2007).

The reference list should be in the format described below. Journal titles should be abbreviated in Index Medicus format (see: ftp://nlmpubs.nlm.nih.gov/online/journals/ljiweb.pdf) using standard abbreviations from the ISSN List of Title Word Abbreviations (see: http://www.issn.org/en/node/344) All authors should be given in the reference list.

Do not use abstracts as references. "Unpublished observations" and "personal communications" may not be used as references, although references to written, not verbal, communications may be cited (in parentheses) in the text. Include in the references manuscripts accepted but not yet published, designate the journal followed by "in press" (in parentheses). Information from manuscripts submitted but not yet accepted should be cited in the text as "unpublished observations" (in parentheses).

Examples of the correct form for references are given below:

Journal Reference:

Stein MK, Downing RW, Rickels K 1978. Self-estimates in anxious and depressed outpatients treated with pharmacotherapy. *Psychol Rep* 43: 487-492.

Personal Author(s) of a book:

Osler AG 1976. *Complement: mechanisms and functions*. Englewood Cliffs: Prentice-Hall.

Editor, Compiler, Chairman as Author:

Rhodes AJ, Van Rooyen CE, comps. 1968. *Textbook of virology: for students and practitioners of medicine and the other health sciences*. 5th ed. Baltimore: Williams and Wilkins.

Chapter in Book:

Weinstein L, Swartz MM 1974. Pathogenic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: WB Saunders; 457-472.

Online documents:

National Center for Biotechnology Information. OMIM: online Mendelian inheritance in man. http://www.ncbi.nlm.nih.gov/omim. Accessed February 25, 2007.

Tables

Number tables consecutively with Arabic numerals and supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes, not in headings. Explain in footnotes all non-standard abbreviations used in each table.

For footnotes, use the following symbols in this sequence:

* † ‡ § ¶ ** ††

In preparing tables, consideration should be given to the page width of the Australian Journal of Medical Science. All tables should be prepared for publication vertically. In the text, cite each table in consecutive order, and mark in the margin of the text its approximate location.

If data from another published or unpublished source is used, written permission must be obtained and a copy must accompany the manuscript.

Illustrations

Colour illustrations may be submitted on a CD. Images should be scanned at a minimum of 300 dpi.

When plotting points, the following symbols are preferred:

In most instances, figures will be reduced to one column in width. All letters and numbers should be drawn to be at least 1.5 mm high after reduction, symbols at least 1.0 mm. Titles for illustrations belong in the legends for illustrations and not on the illustrations themselves.

Photomicrographs must have internal scale markers and the magnification must be stated. Symbols, arrows, or letters used in the photomicrographs should contrast with the background.

Cite each figure in the text in consecutive order, e.g., "Figure 1 illustrates …" or "… as shown (Figure 2)". If a figure has been published, acknowledge the original source and submit with the manuscript written permission from the copyright holder to reproduce the material. Permission is required, regardless of authorship or publisher, except for documents in the public domain.

Legends for illustrations

When symbols, arrows, numbers, or letters are used to identify parts of illustrations, identify and explain each one in the legends. The figure legend must contain a boldface (a) name ("Figure" + arabic figure number) and (b) substantive title.

Abbreviations

Use only standard abbreviations (see list of commonly used abbreviations).

Avoid abbreviations in the title. The full term for which an abbreviation stands must precede its first use in the text unless it is a standard abbreviation for a unit of measurement.

Report measurements in the units in which the measurements were made. In most countries the International System of Units (SI) is standard.

Commonly used abbreviations

Abbreviation or Symbol	Standard Units of Measurement
g	gram
g	gravity
Hz	hertz
h	hour
IU	international unit
K	kelvin
kg	kilogram
L	liter, litre
m	meter, metre
min	min
M	molar
mL	millilitre
mol	mole
N	newton
nm	nanometre
p	probability
rpm	revolutions per min
s	second
wk	week
yr	year

Additional information

The following are useful sources of information. The first two publications are used by the AJMS as standard references.

Style Manual Committee. Council of Biology Editors. *Scientific style and format: the CBE manual for authors, editors, and publishers.* 6th ed. Cambridge University Press, 1994.

Style manual for authors, editors and printers. 6th ed. John Wiley & Sons Australia Ltd, 2002.

O'Connor M, Woodford FP. *Writing scientific papers in English: an ELSE-Ciba Foundation guide for authors.* Amsterdam, Oxford, New York: Elsevier-Excerpta Medica, 1975.

Day RA. *How to write and publish a scientific paper.* Philadelphia, Institute for Scientific Information Press, 1979.

Zeiger M. *Essentials of writing biomedical research papers*. 2nd ed. New York, McGraw-Hill, 2000.

Matthews JR, Matthews RW. *Successful scientific writing: a step-by-step guide for the biological and medical sciences*. 3rd ed. Cambridge, Cambridge University Press, 2007 [Also available in eBook format.]

Medical Training Solutions
Online Training and Competency Assessment for the Clinical Lab and Point of Care

Features/Benefits

- Streamlined tracking and documentation;
- High quality multimedia content developed with the University of Washington;
- Over 50 hours of Continuing Education (CE) credit;
- Create your own tests;
- Upload and track your existing documents;
- Used by over 1,500 laboratories.

As an AIMS member you have exclusive access to the Medical Training Solutions (MTS) online clinical laboratory continuing education programme. Please note only current financial members of AIMS are eligible to access MTS. Free Student members do not have access to this resource.

Overview

AIMS purchases a yearly subscription to the Medical Training Solutions (MTS) online clinical laboratory continuing education programme as one of your member benefits. AIMS members receive unlimited access to the laboratory training and competency assessments from MTS.

MTS is accessible online from work and at home so it is most convenient to you. The laboratory training and competency assessment provides training, continuing education and rotating competency assessment tests on clinical laboratory procedures in an engaging format.

MTS adds new training courses periodically. At the moment there are more than 60 courses in the Training Library and over 90 presentations in the Lecture Library.

https://www.aims.org.au/member-resources/medical-training-solutions-mts/medical-training-solutions

http://www.medtraining.org/

The *Training Library* covers topics in:

- Safety
- Specimen Collection
- Microscopy
- Molecular Techniques
- Chemistry
- Haematology
- Coagulation
- Microbiology
- Body Fluids
- Blood Banking
- Immunology
- and more!!

Australian Institute of Medical and Clinical Scientists

Fellowship of AIMS

The AIMS Fellowship is an attractive and highly competitive option to academic post graduate degrees. The Fellowship is recognised by the Department of Health for meeting the requirements for supervision of category GX and GY laboratories.

TRANSFUSION SCIENCE
CLINICAL BIOCHEMISTRY
CYTOLOGY
HAEMATOLOGY
ANATOMICAL PATHOLOGY
IMMUNOLOGY
MICROBIOLOGY
GENERAL (including Core Laboratory)

Qualification for the Fellowship is by EXAMINATION in one of the eight disciplines.

Candidates for the Fellowship must have been members for a minimum of two years and must meet certain other criteria.

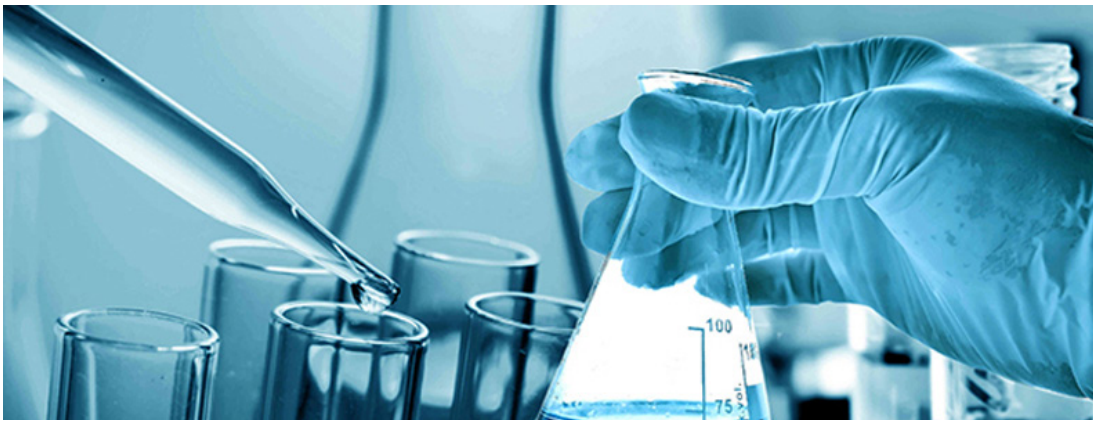
The Fellowship program is modular - candidates must complete:

- two compulsory modules
- two elective modules
- a viva voce examination
- a scientific diseration OR a successful relevant research degree thesis completed within the last two years (eg Honours, Masters, PhD); OR a relevant paper published in a peer reviewed journal.

To enrol in the Fellowship program or for further information please contact the AIMS National Programs Manager:

Ph: +61 7 3876 2988

E mail: programs@aims.org.au



accmlsw

qualified ✓
competent ✓
certified ✓

Australian Council for the Certification of the Medical Laboratory Scientific Workforce

Why become certified?

Your status as a certified medical laboratory professional is a public guarantee that you are qualified, competent and continuing your professional development.

- Recognition of scientific qualifications;
- Certification aligned with competency development and assessment processes;
- Acknowledgement of participation in continuing educational activities;
- Increased professional credibility and prestige in the industry;
- Support of industry standards;
- Demonstrated commitment to superior professionalism;
- Advantage in the recruitment process.

https://accmlsw.wildapricot.org/